International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Comparing Diode Laser and An In-Office Agent in the Treatment of Dentinal Hypersensitivity

Vineet Nair[1], Nairita Saha[2]

[1] Associate Professor, Department of Periodontia, Dr. R. Ahmed Dental College & Hospital, Kolkata
Corresponding Author Email: *drvineet_nair[at]yahoo.co.in*

[2] Assistant Professor, Department of Oral Medicine & Radiology, Burdwan Dental College & Hospital, Burdwan

Abstract: ***Background: 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. Needless to say, it becomes a nightmare to the patient to eat or drink. Aim: This study compared the efficacy of 660nm diode laser, 980nm diode laser and Amorphous Calcium phosphate- Casein Phosphopeptide (ACP-CPP) agent in the treatment of DH. Materials & Methods: A total of 60 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, B and C treated by 660nm diode laser, 980 nm diode laser and ACP-CPP agent respectively. 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 minutes, 1 week, 1 month, 3 months and 6 months. Results: On completion of the study, there was statistically significant reduction in NRS scores within the three groups for the tactile, thermal and air stimuli from baseline to six months. 980nm laser was more effective than 660nm laser at 30 minutes and 1 week but there was no statistically significant difference between the two groups at 1 month, 3 months and 6 months. 660nm and 980nm diode laser were more effective than ACP-CPP agent in reducing DH throughout the study period. Conclusion: 660nm diode laser, 980 nm diode laser and ACP-CPP agent showed definite potential as effective desensitizers when used as an in-office procedure.***

Keywords: Dentin hypersensitivity; Desensitizing agent; Diode Laser; Numeric Rating Scale; Pain

1. 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 (NCCLs) 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 regards 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 hyperaemia, 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 long been an area of research. A commercial preparation comprising Amorphous Calcium Phosphate (ACP) and Casein phosphopeptide (CPP) is used in the management of DH. With the dawn of laser technology and its growing application in dentistry, further therapeutic options such as low-level lasers like diode 660nm and high-power lasers like diode 980nm are accessible for the treatment of dentinal pain. This study was planned to compare the efficacy of 660nm diode laser, 980nm diode laser and Amorphous Calcium phosphate- Casein Phosphopeptide (ACP-CPP) agent in the treatment of DH.

2. Materials and Methods

Sixty patients (males- n=30, females- n=30) aged between 20-50 years and reporting with the chief complaint of DH to the OPD 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. Intensity of pain expressed by each patient was recorded as per Numeric Rating Scale (NRS).[5] 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, 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 last three 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 of the coin into three groups by an investigator-

Group A (n=20)- 660nm diode laser,
Group B (n=20)- 980nm diode laser,
Group C (n=20)- ACP-CPP agent. (GC Tooth Mousse™).

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 one second after the teeth were isolated with cotton rolls.

Application of Laser:
Laser application was performed in accordance with the manufacturer's (Hager & Werken GmbH & Co KG, Germany) instructions. In subjects of group A, 660nm laser was applied on the selected teeth, at a continuous wavelength of 660nm ± 5 nm, 40 mW power, in continuous wave mode and applied in no contact mode, using a fibre of 320-micron diameter. It was applied for 8 seconds at hypersensitive points in three sessions, at intervals of 48 hours. In subjects of group B, 980nm laser (975nm ± 10 nm) was applied at 2W power in continuous wave mode. It was applied in no contact mode, using a fibre of 320-micron diameter. Each site received three applications of one minute each once a week for three weeks.

Application of ACP-CPP Agent (G C Tooth Mousse ™):
Sufficient amount of ACP-CPP agent was applied on to the tooth surfaces of the subjects in group C using an applicator tip and left undisturbed for three minutes. The patients were then instructed to use their tongue to spread the remaining agent throughout the mouth and avoid expectoration and delay swallowing for additional 1 to 2 minutes. They were instructed not to eat or drink for 30 minutes following the application. Patients who initially responded to the tactile, cold, air stimulus with a score of ≥ 5 in the NRS were included in the study. The NRS values were recorded at baseline, 30 minutes, 1 week,1 month, 3 months and at 6 months.

Statistical analysis
Data was entered in Microsoft excel and analysed using SPSS (Version 23.0) package. The results were averaged [mean ± standard deviation] for continuous data and number and percentage for dichotomous data. Normality of data was tested using Shapiro-Wilk test. Proportions were compared using Chi-square (X2) test of significance. Kruskal Wallis test and Mann Whitney U test were used for inter group comparison. "P" value < 0.05 was considered as statistically significant.

3. Results

All the sixty subjects kept their appointments as scheduled and there were no dropouts. The results of the study are presented in Tables 1-2. Comparison of 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 value <0.001) from baseline to six 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 value<0.001) between 660nm diode laser group and 980nm diode laser group after 30 minutes and at 1 week with the 980nm diode laser group showing greater reduction in the mean NRS scores. However, there was no statistically significant difference between these two groups at 1 month, 3 months and at 6 months. When 660nm diode laser group and 980nm diode laser were compared to ACP-CPP agent group, there was a statistically significant difference (P value <0.001) with both the laser groups showing a greater reduction in the mean NRS scores compared to ACP-CPP agent at the various study time intervals.

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

Visit	Group	Median	Min.	Max.	Chi-square	*P* value
COLD TEST						
Pre	A	8.00	6	8	1.045	0.579
	B	8.00	7	9		
	C	8.00	8	9		
After 30 min	A	5.00	4	6	30.117	<0.001
	B	3.00	3	4		
	C	6.00	4	7		
Week 1	A	5.00	5	6	31.666	<0.001
	B	4.00	4	5		
	C	6.00	5	7		
Month 1	A	5.00	5	7	23.323	<0.001
	B	5.00	5	6		
	C	7.00	6	7		
Month 3	A	5.00	5	6	25.659	<0.001
	B	5.00	5	6		
	C	7.00	6	8		
Month 6	A	5.00	5	6	23.333	<0.001
	B	5.00	5	6		
	C	6.00	6	8		
MANUAL SCRATCH TEST						
Pre	A	8.00	6	9	0.699	0.781
	B	8.00	7	9		
	C	8.00	7	8		
After 30 min	A	4.00	3	6	30.271	<0.001
	B	3.00	2	4		
	C	6.00	5	7		
Week 1	A	5.00	4	6	29.541	<0.001
	B	4.00	3	5		
	C	6.00	5	7		
Month 1	A	5.00	4	7	25.095	<0.001
	B	5.00	4	6		
	C	7.00	6	8		
Month 3	A	6.00	5	7	25.075	<0.001
	B	5.00	4	6		
	C	7.00	6	9		
Month 6	A	6.00	5	7	26.237	<0.001
	B	6.00	5	6		
	C	8.00	7	9		
AIR BLAST TEST						
Pre	A	8.00	7	9	0.565	0.833
	B	8.00	7	9		
	C	8.00	7	8		
After 30 min	A	4.00	3	5	25.287	<0.001
	B	3.00	2	4		
	C	5.00	5	6		
Week 1	A	5.00	3	6	26.240	<0.001
	B	4.00	3	5		
	C	6.00	5	7		
Month 1	A	6.00	4	7	20.897	<0.001
	B	5.00	5	6		
	C	7.00	6	7		
Month 3	A	6.00	5	7	25.134	<0.001
	B	6.00	4	7		
	C	7.00	7	8		
Month 6	A	6.00	5	7	27.565	<0.001
	B	6.00	5	7		
	C	8.00	7	9		

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

Cold Test						
Visit	660nm vs 980nm		660nm vs GC Tooth Mousse		980nm vs GC Tooth Mousse	
	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value
Pre	66.500	0.352	76.500	0.813	71.500	0.440
After 30 min	12.000	<0.001	14.500	<0.001	0.000	<0.001
Week 1	14.000	<0.001	9.000	<0.001	0.000	<0.001
Month 1	52.500	0.201	13.500	<0.001	5.000	<0.001
Month 3	66.000	0.600	8.000	<0.001	2.000	<0.001
Month 6	58.500	0.123	13.500	<0.001	7.500	<0.001
Manual Scratch Test						
Visit	660nm vs 980nm		660nm vs GC Tooth Mousse		980nm vs GC Tooth Mousse	
	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value
Pre	72.000	0.717	73.500	0.515	78.000	0.692
After 30 min	16.000	<0.001	11.000	<0.001	0.000	<0.001
Week 1	21.000	<0.001	26.000	<0.001	2.000	<0.001
Month 1	63.000	0.527	15.500	<0.001	6.000	<0.001
Month 3	66.000	0.600	21.000	<0.001	7.500	<0.001
Month 6	52.500	0.166	10.000	<0.001	0.000	<0.001
Air Blast Test						
Visit	660nm vs 980nm		660nm vs GC Tooth Mousse		980nm vs GC Tooth Mousse	
	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value	Mann-Whitney U	*P* value
Pre	161.000	0.805	161.00	0.784	164.500	0.488
After 30 min	35.000	0.008	22.500	<0.001	0.000	<0.001
Week 1	37.500	0.01	37.000	0.009	6.000	<0.001
Month 1	67.500	0.326	27.500	0.002	12.000	<0.001
Month 3	60.500	0.158	16.500	<0.001	5.500	<0.001
Month 6	79.500	0.761	6.000	<0.001	6.000	<0.001

4. Discussion

Dentin hypersensitivity 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.[6] Even though DH is one of the most common problems met by dental professionals, universally acknowledged guidelines for differential diagnosis as well as selection of reliable treatment modalities are deficient.[7]

Our understanding about its aetiology is based principally on data attained from in-vitro and in-situ studies as well as from data obtained from epidemiological surveys.[8] 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.[9] Dentin exposure can be caused by physical, chemical, pathological, 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. [10-12] Periodontal tissue loss or gingival recession is another chief predisposing factor since this leads to exposure of cervical and root dentin.[13] Other factors, such as aging, 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.[14]

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 assessment including immunohistochemical and ultrastructural analyses.[15] 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.[16] There is, however, no evidence confirming that synaptic structures that might link odontoblasts with pulpal nerves actually exist.[17]

The most widely accepted mechanism for DH has been the hydrodynamic theory proposed by Brännström.[18] It states that environmental, mechanical, thermal and chemical changes cause the movement of fluid within dentinal tubules, which stimulate the terminals of pulpal nerve fibres 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 blast as well as thermal (cold) and osmotic (sugar, acid) stimuli are believed to upsurge the outward flow of tubular fluid.[19] 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.[20] The intra-dental myelinated Aβ and some Aσ fibres 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.[19] However, how these non-noxious mechanical stimuli of dentin tubule fluid movements encourage the nociceptive transduction in dental pulpal nerve fibres 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.[21]

The hydrodynamic theory has been challenged by emerging evidence suggesting that odontoblasts might well play an important role in the pathogenesis mechanisms of DH. [22, 23] 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.[24, 25] 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.[26] Recently, the existence of autocrine/paracrine mechanisms for ATP-involved purinergic signalling in cultured odontoblast-like stem cells has been noticed.[27] 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 characterise a novel clarification as to how odontoblasts contribute in a mechanosensory mechanism leading to the pain associated with DH.[8]
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.[28] 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).[29]

Currently no single method of eliciting and assessing cervical dentin sensitivity may be considered ideal.[30] In a systematic review comparing VRS, VAS and NRS it was found that NRS reported better patient compliance than the other scales.[31] Hence an assessment using NRS was used in the present study. In the present study, in the 660nm diode laser group there was a statistically significant reduction in the sensitivity scores from baseline to 6 months. Low level laser therapy such as 660nm diode laser acts at the cellular level increasing the production of tertiary dentin and consequently sealing the dentinal tubules. These findings were similar to a study by R Lizarelli[32] where Low Intensity Laser Therapy (LILT) at irradiation parameters of 660nm, 40mW 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-fibre 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, ie; Gluma Desensitizer, Seal & Protect, Oxa-gel, Fluoride and LILT (660 nm/3.8 J/cm^2/15 mW) 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.

Subjects of the second group were treated using 980nm diode laser. The group showed a statistically significant difference in sensitivity scores from baseline to six months which was in accordance with a study by Mariana-IoanaMiron et al.[33] 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 SEM study[34], 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.

The third treatment group in this study were subjects using ACP-CPP agent (GC Tooth Mousse ™). They showed a statistically significant reduction in sensitivity scores from baseline to six months similar to a study done by Rosaiah et al [35] in which GC Tooth Mousse showed a rapid and sustained desensitizing action and was effective in reducing cervical dentinal sensitivity. This could happen because the ACP-CPP combination localizes in plaque in the form of nano clusters and causes remineralization of enamel at a much faster rate. Studies done by Bhandary et al and Torwane et al have also shown the effectiveness of GC Tooth mousse in reducing hypersensitivity from baseline to 2 weeks and baseline to 21 days respectively as observed in our study. [36, 37]

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.

2) Behavioural 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.

3) Non-invasive treatments for pain relief through occluding dentin tubules and blocking nociceptive transduction/transmission- Application of desensitizing agents is the most frequently used non-invasive treatment for DH. 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 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.[38] It is for this reason that we have included laser and desensitizing agent in our study.

But 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,[39] but clinical trials with sound design have failed to provide evidence that potassium is effective in desensitizing teeth.[40] 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.[41] 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.[42-44] Both low-output and higher output laser application have been reported to be effective for treatment of DH. Some experiments suggest that low output laser might operate by suppressing the excitability of pulpal nerves.[45] Higher output laser is thought to reduce symptoms of DH by inducing the occlusion of dentin tubules.[46]

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.[47] 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.[48] 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.[49]

5. Conclusion

The presence of dentin exposure is usually prerequisite to the development of DH. Accurate diagnosis is the key in selecting the right treatment strategy. 660nm diode laser, 980 nm diode laser and ACP-CPP agent of the three treatment groups showed definite potential as effective desensitizers when used as an in-office procedure though the lasers were found to be more effective. We anticipate that with improved understanding of the underlying nociceptive mechanisms of DH, promising novel therapies will emerge and provide more effective relief for patients with DH.

Conflict of interest- Nil

Financial disclosure- Self funded

References

[1] Mantzourani M, Sharma D. Dentine sensitivity: past, present and future. J Dent. 2013;41(Suppl 4):S3–17.
[2] 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.
[3] 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.
[4] Aranha AC, Pimenta LA, Marchi GM. Clinical evaluation of desensitizing treatments for cervical dentin hypersensitivity. Braz Oral Res. 2009;23:333–9.
[5] Correll DJ. The Measurement of Pain: Objectifying the Subjective. Pain management. 2nd Ed. 2011:191-201.
[6] 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(7):595–602.
[7] Liu, XX., Tenenbaum, H.C., Wilder, R.S. et al. Pathogenesis, diagnosis and management of dentin hypersensitivity: an evidence-based overview for dental practitioners. BMC Oral Health 2020;20:220.
[8] West NX, Lussi A, Seong J, Hellwig E. Dentin hypersensitivity: pain mechanisms and aetiology of exposed cervical dentin. Clin Oral Investig. 2013;17(Suppl 1):S9–19.
[9] 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(5):280–4.
[10] Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited: a new perspective on tooth surface lesions. J Am Dent Assoc. 2004;135(8):1109–18.
[11] 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(2):182–7.
[12] 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(5):274–9.
[13] Smith RG. Gingival recession. Reappraisal of an enigmatic condition and a new index for monitoring. J Clin Periodontol. 1997;24(3):201–5.
[14] Tugnait A, Clerehugh V. Gingival recession-its significance and management. J Dent. 2001;29(6):381–94.
[15] 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(12):1348–55.
[16] Chiego DJ Jr. The early distribution and possible role of nerves during odontogenesis. Int J Dev Biol. 1995;39(1):191–4.
[17] Davari A, Ataei E, Assarzadeh H. Dentin hypersensitivity: etiology, diagnosis and treatment; a literature review. J Dent (Shiraz). 2013;14(3):136–45.

[18] Brannstrom M. Dentin sensitivity. Arsb Goteb Tandlak Sallsk; 1964. p. 15–35.
[19] Matthews B, Vongsavan N. Interactions between neural and hydrodynamic mechanisms in dentine and pulp. Arch Oral Biol. 1994;39(Suppl):87S–95S.
[20] Pashley DH. Mechanisms of dentin sensitivity. Dent Clin N Am. 1990;34(3):449–73.
[21] 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(3):261–72.
[22] 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.
[23] 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(3):261–7.
[24] 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(9):1259–66.
[25] 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(10):2211–23.
[26] 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(5):341–55.
[27] 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(2):242–9.
[28] Miglani S, Aggarwal V, Ahuja B. Dentin hypersensitivity: recent trends in management. J Conservative Dent. 2010;13(4):218–24.
[29] Liu X, Malmstrom HS, Tallents R. Dental restoration induced orofacial pain and its management. Eur J Gen Dent. 2015;4(1):4.
[30] H. Breivik, P. C. Borchgrevink, S. M. Allen, L. A. Rosseland, L. Romundstad, E. K. Breivik Hals, et al. Assessment of pain. Br J Anaesth. 2008 ;101(1):17–24.
[31] M J Hjermstad, Peter M. Fayers, Dagny F. Haugen. Studies Comparing Numerical Rating Scales, Verbal Rating Scales, and Visual Analogue Scales for Assessment of Pain Intensity in Adults: A Systematic Literature Review. Journal of Pain and Symptom Management 2011;41(6)1073-93.
[32] Lizarelli R, F A Miguel, G E Villa, E C Filho et al. Clinical Effects of Low-intensity Laser vs Light-emitting Diode Therapy on Dentin Hypersensitivity. J Oral Laser Applications: 2007;7:129-36.
[33] Mariana-IoanaMiron, DorinDodenciu, Diana Lungeanu, Cosmin Anton Balabuc, Laura Maria Filip, Carmen Todea. An Evaluation of the 980 nm GaAlAs High-level Diode Laser in the Treatment of Dentine Hypersensitivity.TMJ2007;57(4):280-6.
[34] Liu Y, Gao J, Gao Y, XU Sh, Zhan X, Wu B. In Vitro Study of Dentin Hypersensitivity Treated by 980-nm Diode Laser. J Lasers Med Sci 2013;4(3):111-9.
[35] Rosaiah.K, Aruna.K. Clinical Efficacy of Amorphous Calcium Phosphate, G.C. Tooth Mousse and Gluma Desensitizer in Treating Dentin Hypersensitivity. International Journal of Dental Clinics 2011;3(1):1-4.
[36] Bhandary S, Hegde M. N. A clinical comparison of in -office management of dentin hypersensitivity in a short term treatment period. IJBAR 2012;03:3.
[37] Torwane NA, Hongal S, Goel P, Chandrashekhar B.R et al. Effect of two desensitizing agents in Reducing Dentin Hypersensitivity: An in-vivo Comparative Clinical Trial. Journal of Clinical and Diagnostic Research. 2013;7(9):2042-6.
[38] Schmidlin PR, Sahrmann P. Current management of dentin hypersensitivity. Clin Oral Investig. 2013;17(Suppl 1):S55–9.
[39] Orchardson R, Gillam DG. The efficacy of potassium salts as agents for treating dentin hypersensitivity. J Orofac Pain. 2000;14(1):9–19.
[40] Poulsen S, Errboe M, Lescay Mevil Y, Glenny AM. Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev. 2006;2006(3):Cd001476.
[41] 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(2):147–53.
[42] Olley RC, Pilecki P, Hughes N, Jeffery P, Austin RS, Moazzez R, et al. An in situ study investigating dentine tubule occlusion of dentifrices following acid challenge. J Dent. 2012;40(7):585–93.
[43] 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-13A.
[44] 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(1):23–31.
[45] Machado AC, Viana IEL, 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(4):745–53.
[46] McCarthy D, Gillam DG, Parson DJ. In vitro effects of laser radiation on dentine surfaces. J Dent Res. 1997;76(Special Issue):233.
[47] 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(1):463–8.
[48] 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(3):295–306.
[49] 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(4):452–78.