

ASSESSING THE VARIABLES RESPONSIBLE FOR CAUSING DENTINAL HYPERSENSITIVITY – A CROSS SECTIONAL STUDY

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ABSTRACT

Introduction: Dentinal hypersensitivity is one of the commonest complaints encountered in a dental practice. Various causative factors are responsible in exposing the dentinal tubules further manifesting as tooth pain. The most challenging aspect for a dentist is to rule out the possible factors and do responsible intervention at the appropriate time.

Aim and objectives: To investigate the various etiological factors associated in patients who reported with the complaint of dentinal hypersensitivity and to assess whether any correlation exists between various factors and habits of the patients. **Materials and Methods:** A cross sectional study was conducted comprising of 383 subjects (238 males and 145 females). A questionnaire was given to the patients which records the demographic data and the personal habits. A clinical examination was done along the questionnaire. The diagnosis was

confirmed using a blast of air from the three way syringe and the subjective pain was recorded using a Visual Analogue Scale. **Results:** A significantly higher number of male patients reported with hypersensitivity and the mean age was found to be 37.65. A total of 49.6% of the patients reported with gingival recession and 72.8% had non carious tooth structure loss which includes attrition and abrasion. There was statistically significant

relationship with adverse oral habits such as pan chewing and smoking with dentin hypersensitivity. **Conclusion:** Various factors like faulty tooth brushing, type of tooth bristles and habits like night grinding and clenching have contributed to wearing of the tooth surface which can result in dentin hypersensitivity. Habits like smoking, pan chewing, horizontal tooth brushing and unwanted usage of interproximal cleansing aids can result in clinical attachment loss which can elicit a sensitive tooth.

INTRODUCTION

Dentinal hypersensitivity being one of the most prevalent dental problem, affects almost 12.3% of the adults in United States of America according to a study conducted in Northwest, America.^[1] Holland et al described dentinal hypersensitivity as ‘characterized by short, sharp pain arising from exposed dentin in response to a stimuli typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology’.^[2]

Dentin consists of organic component containing collagen fibers in a matrix of collagenous proteins and inorganic components containing hydroxyapatite crystals. The dentinal tubules are found within the dentin and they extend from the pulp to the outer dentinal surface.^[3] Tomes fibers are found within the dentinal tubules along with odontoblast that communicates with the pulp.^[4] There are three types of nerve fibers which are found within the dentinal tubules, namely the A-delta fibers, A-beta and C fibers.^[3] The chief fibers responsible in the perception of pain in dentinal hypersensitivity are the A fibers.^[4]

The main cause of dentinal hypersensitivity is exposed dentinal tubules.^[5] This is in accordance with the most widely accepted theory which is Branstrom’s Hydrodynamic theory.^[4] This theory states that the movement of fluid in the open dentinal tubules results in the stimuli being transmitted to the pulp surface. The pain response results from the wider areas of the dentinal tubules which are closer to the pulp chamber and the fluid movement away from the pulp which activates the nerve associated with the odontoblast at the end of the tubule. This fluid movement stimulates the small, myelinated A-delta fibers resulting in the localized sharp pain which is usually associated with dentinal hypersensitivity.^[3]

The loss of enamel and cementum can cause tooth wear like abfraction, abrasion, attrition and erosion which often results in the exposure of dentin to the oral environment leading to dentinal hypersensitivity.^[6] Abfraction is said to involve eccentric occlusal loading which

leads to deformation and cusp flexion resulting in the damage of enamel rods in the cervical region leading to exposure of coronal root dentin in severe cases.^{[6] [7]} Abrasion is related to a number of factors attributed to tooth brushing and the type of dentrifices used. They are stiffness and configuration of the toothbrush bristles in combination with force, brushing method, duration of brushing, frequency of brushing and abrasiveness of the dentrifice.^[3] Wearing of tooth structure at sites of direct tooth contact is called attrition.^[6] Attrition can be seen in those who have parafunctional habits such as bruxism and clenching and also due to Temporomandibular Joint derangement. A stressful lifestyle is said to contribute to bruxism and clenching.^[8] Erosion can be of extrinsic and intrinsic origin. Medical conditions such as acid reflux disease and disorders such as bulimia which causes gastric acid regurgitation results in intrinsic erosion. Frequent consumption of carbonated drinks, fruits and fruit drinks are by far the most common causes of extrinsic erosion resulting in a more acidic oral environment. Highly acidic foods and drinks have the ability to erode enamel and smear layer. This exposes the dentin which eventually leads to sensitivity and pain.^[3]

Gingival recession is another common cause for dentinal hypersensitivity. As the cementum layer that is covering the root surface is softer than enamel and easily removed by faulty tooth brushing or repeated root planning, it can result in exposure of dentinal tubules.^[6] Some of the causes for gingival recession include age, periodontal disease and surgery, poor oral hygiene, excessive tooth brushing, high frenal attachment, occlusal trauma and thin labial cortical plate of bone.^[3]

The aim of this study is to assess the proportions of individuals with dentinal hypersensitivity according to their personal oral hygiene and demographic data. The study was done to correlate the clinical parameters examined with that of their habits like smoking and pan chewing. It also assessed the correlation between subjective pain scale score with that of amount of root exposure.

MATERIALS AND METHODS

This cross sectional study was done on subjects reporting to the Oral Medicine clinic, with the chief complaint of dentin hypersensitivity. A total of 383 subjects participated in this study. The subjects who were allocated for the study were given a written informed consent before hand.

Out of the 383 subjects examined, 238 were males and 145 were females. The age group of the subjects examined was at the range of 18 to 80. Patients included in the study had at least 28 teeth in the oral cavity and complained of dentinal hypersensitivity in more than 6 teeth. The exclusion criteria were based on the Canadian Advisory Board of Dentin Hypersensitivity.^[9] They are:

- Patients who have a history of systemic diseases like diabetes, hypertension and gastro esophageal reflux disease.
- Patients having dental disease pathology, patients who have previously or currently undergoing treatment for sensitivity.
- Patients who are currently using desensitizing toothpaste, patients under any medication (anti-inflammatory drugs, analgesics, psychiatric drugs).
- Pregnant and breastfeeding women.
- Patients who have undergone periodontal and orthodontic treatment.

Teeth which were excluded in the study included root filled teeth, tooth which was used for abutment and teeth with marginal restoration.^[9]

A personal questionnaire was read out to the patients and the answers were recorded by an assistant. The questionnaire which was given to the patients enrolled for the study included the personal data like age, gender, dietary pattern, brushing habits (type of toothbrush, duration and frequency of brushing), frequency of intake of meals and other habits, if present, like smoking, pan chewing, clenching and night grinding.

An intra oral clinical examination included Simplified Oral Hygiene Index (OHI-S), gingival recession, non-carious tooth structure loss (NCTSL) and restorative status. The OHI-S index is composed of two components, the Debris Index and the Calculus Index. Each of these indexes are based on numerical determinations representing the amount of debris or calculus found on the selected tooth surfaces, example 16, 11, 26, 36, 41, 46. The scores for each index are then added up to obtain the simplified oral hygiene index.^[10]

All the subjects were further diagnosed by a blast of air from a three way syringe connected to an air compressor at a pressure of 60psi under room temperature at about 20°C to 25°C which was held at a distance of 1cm from the tooth surface lasting for 1 second.^[11] The presence of uncomfortable feeling caused by the air stimuli was recorded as subjective pain scales as Visual Analogue Scale (VAS). The VAS scores ranges from 0 to 5 for rating the

severity of tooth pain (Figure 1). This scale is based on the subject's perception of pain experienced during everyday routine intake of hot or cold food or drink, cold air, tooth brushing or sweet or sour foods. The clinical examination was done by vision or tactile examination with the help of a No.23 Shepherds Hook explorer. All the restorations present were also recorded.^[12]

(Annexure 1 Figure 1: VAS scale used to record the subjective pain)

Presence or absence of gingival recession was noted and was graded according to the classification proposed by P.D. Miller in 1985.^[13]

Grade I: Marginal tissue recession not extending to the mucogingival junction. No loss of interdental bone or soft tissue.

Grade II: Marginal tissue recession extends to or beyond the mucogingival junction. No loss of interdental bone or soft tissue.

Grade III: Marginal tissue recession extends to or beyond the mucogingival junction. Loss of interdental bone or soft tissue is apical to the cemento-enamel junction but coronal to the apical extent of marginal tissue recession.

Grade IV: Marginal tissue recession extends to or beyond the mucogingival junction. Loss of interdental bone extends to a level apical to the extent of marginal tissue recession.

The periodontal status of all the teeth were assessed using William's periodontal probe graduated in 1mm. The tip of the instrument was placed parallel to the long axis of the tooth at 20 grams. Force was applied into the gingival sulcus and the probing depth and clinical attachment loss was recorded for all the six sites of the sensitive tooth. This assessment was done to confirm the absence periodontal pathology.

RESULTS

All the data collected from the patient was statistically analyzed using SPSS version. Chi-square test was done to analyze the proportions between the interactive variables of dentinal hypersensitivity. The analysis of variation or ANOVA was done to compare the subjective pain scale with that of the possible risk factors.

Table 1 shows the demographic data showing different proportions of distribution of patients examined for dentinal hypersensitivity. Out of the 383 subjects who came with the complaint of dentin hypersensitivity, 238 (62.1%) were males and 145 (37.9%) were females. The age

of the subjects of the study ranged from 18 years old to 80 years old and the mean age is 37.65 years. 99% of the subjects used toothbrush and toothpaste and 0.3% used Neem stick and finger and tooth powder to clean their teeth. 71.2% of the study subjects brushed once a day compared to 24.8% who brushed twice a day. Regarding the data collected on the type of bristles used by the subjects, 14.8% used soft bristles, 56.3% used medium bristles and 31.7% used hard bristles. Among the individuals examined, 27.4% had smoking habit and 18% had pan chewing habit. 9.7% had night grinding habit and 6.5% had clenching habit.

(Annexure 1 Table 1-Demographic data of the subjects who participated in the study.)

Table 2 shows the clinical parameter examined in the subjects with dentinal hypersensitivity. About 33.7% of the total subjects had poor oral hygiene status, 51.2% had fair oral hygiene status and 15.2% had good oral hygiene status. About 49.6% demonstrated gingival recession, out of which 26.6% had Grade 1 gingival recession, 14.6% had Grade 2, 6.3% had Grade 3 and 1% had Grade 4 gingival recession. On examination for non carious loss of tooth structure, 55.6% have attrition and 17.2% have abrasion. None of the patients examined exhibited erosion and abfraction. One way test was done to compare the VAS scores with gingival recession grades and it was concluded from the result that the higher the grade of gingival recession, the higher the subjective pain of the patient. (Table 3).

(Annexure 1 Table 2- Clinical parameters of the subjects who took part in the study.)

(Annexure 1 Table 3- Correlation between different grades of gingival recession with VAS scores.)

Chi-square test was done to compare the proportions of subjects with night grinding habit and non-carious tooth structure loss like attrition. Subjects who had both night grinding and clenching manifested with attrition and the values were found to be statistically significant, which is <0.001 and <0.003 respectively. (Table 4).

(Annexure 1 Table 4- Correlation between night grinding and clenching habit with attrition.)

Correlation between the proportions of subjects with smoking and pan chewing habits with grades of recession was done by Chi-square test and was found to be statistically significant. (Table 5) Subjects who used toothbrushes with medium and hard bristles showed greater degree gingival recession than soft bristles. (Table 6).

(Annexure 1 Table 5- Correlation between smoking and pan chewing habit with grades of gingival recession.).

(Annexure 1 Table 6- Correlation between types of bristles used and grades of gingival recession.).

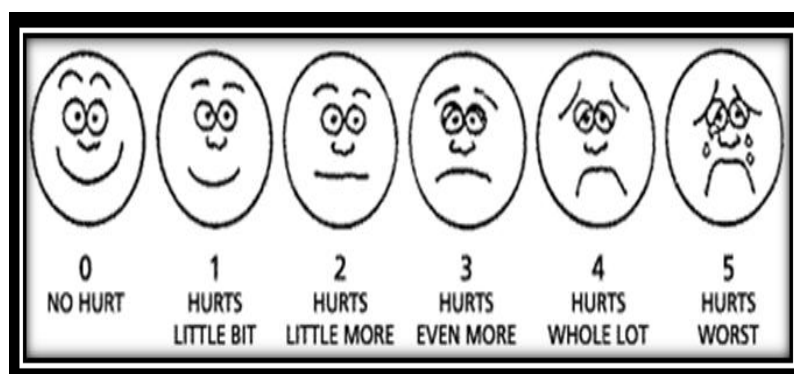


Fig 1: VAS scale used to record the subjective pain

Table 1-Demographic data of the subjects who participated in the study

Demographic Data		Number of patients in percentage (%)
Gender	Male	62.1
	Female	37.9
Use of mechanical plaque control aids	Brush and paste	99.5
	Neem stick	0.3
	Finger and tooth powder	0.3
Brushing frequency	Once	75.2
	Twice	24.8
Types of bristles used	Soft	14.8
	Medium	53.6
	Hard	31.7
Habits	Smoking	27.4
	Pan chewing	18.0
	Night Grinding	9.7
	Clenching	6.5

Table 2- Clinical parameters of the subjects who took part in the study.

Clinical Examination		Number of patients in percentage (%)
Oral Hygiene Index	Poor	33.7
	Fair	51.2
	Good	15.1
Non-carious tooth structure loss	Attrition	55.6
	Abrasion	17.2
Grade of Gingival recession	Nil	51.4
	Grade 1	26.6

	Grade 2	14.6
	Grade 3	6.3
	Grade 4	1.0

Table 3- Correlation between different grades of gingival recession with VAS score.

Gingival recession grade	VAS Score		P-Value
	Minimum	Maximum	
Nil	1	4	<0.001
Grade-1	1	5	
Grade-2	1	5	
Grade-3	2	4	
Grade-4	4	4	

Table 4- Correlation between night grinding and clenching habit with attrition.

Habits		Attrition	P-Value
		% of patients	
Night Grinding	Absent	98.8	<0.001
	Present	83.6	
Clenching	Absent	97.6	<0.003
	Present	90.1	

Table 5-Correlation between smoking and pan chewing habit with grades of gingival recession.

Gingival Recession Grade							P-Value
Habits		NIL	Grade 1	Grade 2	Grade 3	Grade 4	
		% of patients	% of patients	% of patients	% of patients	% of patients	
Smoking	Absent	76.6	74.5	58.9	70.8	25.0	0.010
	Present	23.4	25.5	41.1	29.2	75.0	
Pan Chewing	Absent	86.8	81.4	67.9	87.5	25.0	0.004
	Present	13.2	18.6	32.1	12.5	75.0	

Table 6- Correlation between types of bristles used and grades of gingival recession.

Type of Bristles	Gingival Recession Grade					P-Value
	NIL	Grade 1	Grade 2	Grade 3	Grade 4	
	% of Subject	% of Subjects	% of Subjects	% of Subjects	% of Subjects	
Soft	16.2	14.0	12.7	13.0	0.00	<0.001
Medium	61.9	52.0	40.0	21.7	50.0	
Hard	21.8	34.0	47.3	65.2	50.0	

DISCUSSION

The cause of dentinal hypersensitivity was found to be multifactorial.^[5] This cross sectional study which comprised of 383 patients, was done to assess whether the variables described

were involved in causing dentinal hypersensitivity. The participants, 238 males and 145 females were those with a complaint of dentinal hypersensitivity of ages ranging between 18 years till 80 years. There were a higher percentage of male participants with a complaint of dentinal hypersensitivity than that of female participants. This was in accordance with a study conducted by Que et al in 2012 where they demonstrated the association between gender and dentinal hypersensitivity. The age groups were assessed and it was found that there was a greater prevalence of hypersensitivity as age increases.^[14]

The clinical evaluation of the subjects included assessment of oral hygiene status. Patients with poor and fair oral hygiene had more sensitivity than those with good oral hygiene. A study done by Al-Wahadni et al also concluded that poor oral hygiene due to infrequent tooth brushing were factors associated with dentinal hypersensitivity.^[15]

Subjects who used toothbrush and toothpaste with medium and hard bristles had a greater response to air stimulus from the three way syringe than those who used Neem stick and powder. A study done by Hunter ML in 2002 has demonstrated that faulty tooth brushing can result in abrasion of tooth which can lead to dentin hypersensitivity.^[16]

Tooth wear patterns like attrition and abrasion were found to be contributing factors to dentinal hypersensitivity (p-value = <0.001). Bartlett et al has mentioned that the presence of dentin hypersensitivity is often the only clinical manifestation of active tooth wear.^[17] Habits like night grinding and clenching were also found to be contributing factors to tooth wear. About 49.6% subjects in this study were shown to have gingival recession. All the subjects did not have any active periodontal lesion. On assessment of the subjective pain scales in response to cold air stimulus, it was found that patients who had a higher grade of recession demonstrated with a higher pain score (p-value = <0.001). Strassler et al (2008) has suggested that the causative link to dentin hypersensitivity is gingival recession.^[18] Apical migration of the marginal gingival results in the exposure of cementum. Wearing away of cementum happens due to faulty toothbrushing which causes exposure of the dentinal tubules eventually leading to hypersensitivity.

The results of this study also showed that patients who had the habit of smoking and pan chewing were more prone to dentin hypersensitivity. However this differs from a study done by Dhaliwal et al in 2012 which reported that there no correlation exists between hypersensitivity and smokers.^[9] Smoking and pan chewing can result in clinical loss of

attachment as demonstrated earlier in a study done by Banihashemrad et al in 2007.^[19] It was found that patients who had a habit of pan chewing had higher grades of gingival recession that contributed to dentinal hypersensitivity. This is in accordance with the study conducted by Kumar et al (2004) which concluded that the possible reason for this may be due to the insoluble particulate matter of tobacco and hardness of the areca nut.^[20]

The results of this study demonstrated that patient's perception of pain increases with higher grades of recession. Addy et al in 1987 investigated the distribution between recession, sensitivity and plaque in a group of patients suffering from dentinal hypersensitivity and concluded that there existed a positive correlation between recession and sensitivity.^[21]

CONCLUSION

Dentinal hypersensitivity is a problem that is faced by numerous patients. This study provides evidence that gingival recession and non carious tooth structure loss is the most commonly seen in association with dentinal hypersensitivity. Statistical analysis shows a significant relationship between dentinal hypersensitivity, gingival recession and non carious tooth structure loss. Although it is a relatively a common problem experienced in clinical dental practice, much remains unknown about it. Treatment plans includes in office or at home desensitizing therapy such as fluoride, fluoride varnish, tissue fixatives, oxalates, resin based dental adhesive materials and more. The more recent advancement used in the treatment of dentin hypersensitivity is laser therapy. Some of the examples of laser that has been used successfully in the treatment of dentin hypersensitivity includes CO₂,Nd:YAG and Er-YAG. Treatment plans should not be restricted to one option only as it is not a one-size-fits-all solution. However, in the end meticulous diagnosis, patient counseling and management strategies are crucial to the success of any treatment.

REFERENCE

1. Cruz JC, Wataha JC, Heaton LJ, Rothen M, Sobieraj M, Scott J et al. The prevalence of dentin hypersensitivity in general dental practices in the northwest United States. J Am Dent Assoc, 2013; 144: 288-296.
2. Holland GR, Narhi MN, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. J Clin Periodontol, 1997; 24: 808-13.
3. Saylor, C. D. and Overman, P. R. Dentinal Hypersensitivity: A Review. Academy of Dental Therapeutics and Stomatology, February 2011.

4. Strassler HE, Serio FG. Dentinal Hypersensitivity: Etiology, Diagnosis and Management. In Academy of Dental Therapeutics and Stomatology (pp 2-7). Aegis 2009.
5. Jacobsen PL, Bruce G. Clinical dentin hypersensitivity: Understanding the causes and prescribing a treatment. *J Contemp Dent Pract*, 2001; 1.
6. Bamise CT, Olusile AO, Oginni AO. An Analysis of the Etiological and Predisposing Factors Related to Dentin Hypersensitivity. *J Contemp Dent Pract*, 2001; 9: 052-9.
7. Porto ICCM, Andrade AKM, Montes MAJR. Diagnosis and Treatment of dentin hypersensitivity, *Journal of Oral Science*, 2009; 51: 323-32.
8. Shetty S, Pitti V, Sathish Babu CL, Surendra Kuamr GP, Deepthi BC. Bruxism: A Literature review, *J Indian Prosthodont Soc*, 2010; 10: 141-8.
9. Dhaliwal JS, Palwankar P, Khinda PK, Sodhi PK. Prevalence of Dentin Hypersensitivity: A cross sectional study in rural Punjabi Indians. *J Indian Soc Periodontol*. 2012; 16: 426-9.
10. Moslehzadeh K. Available from: <http://www.mah.se/CAPP/Methods-and-Indices/Oral-Hygiene-Indices/Simplified-Oral-Hygiene-Index/OHI-S>.
11. Que K, Ruan J, Fan X, Liang X, Hu D. A multi-center and cross sectional study of dentin hypersensitivity in China. *J Clin Periodontol*. 2010; 37: 631-7.
12. Caranza FA, Nweman MG. *Clinical Periodontology*, ed. 11, Philadelphia, 1996, W.B. Saunders Company.
13. Que K, Guo B, Jia Z, Chen Z, Yang J, Gau P. A cross-sectional study: Non carious cervical lesion, cervical dentin hypersensitivity and related risk factors. *J Oral Rehabil*, 2013; 40: 24-32.
14. Al- Wahadni A, Lyndon JG. Dentin hypersensitivity in Jordanian Dental Attenders. *J Clin Periodontol*, 2002; 29: 688-93.
15. Hunter ML, Addy M, Pickles MJ, Joiner A. The role of toothpastes and toothbrushes in the aetiology of tooth wear. *Int Dent J*, 2012; 52: 399-405.
16. Bartlett DW. The role of erosion in tooth wear. Aetiology, prevention and management. *Int Dent J*, 2005; 55(4).
17. Strassler HE, Drisko CL, Alexander DC. Dentin Hypersensitivity-Its inter-relationship to gingival recession and acid erosion. *Compend Contin Educ Dent*. 2008; 29(5 Special Issue): 1-9.
18. Banihashemrad SA, Fatemi K, Najafi MH. Effect of smoking on gingival recession. *Dent Res J*, 2008; 5: 1-4.
19. Kumar S, Parmar G, Saiyed HN. Nut and Tobacco chewing. *Br Dent J*, 2004; 197: 292.

20. Addy M, Mostafa P, Newcombe RG. Dentine hypersensitivity: The distribution of recession, sensitivity and plaque. J Dent, 1987; 15: 242-8.