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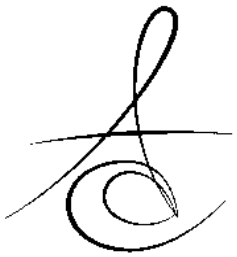

AN ANALYSIS OF AETIOLOGICAL FACTORS RELATED TO DENTIN HYPERSENSITIVITY AND SEVERITY

DENTİN HİPERSENSİTİVİTESİ VE ŞİDDETİYLE İLİŞKİLİ ETİYOLOJİK FAKTÖRLERİN İNCELENMESİ

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SUMMARY

Purpose: In management strategies of dentinal hypersensitivity, aetiological and predisposing factors should be taken into account rather than the treatment alone. The aim of this study was to determine the prevelance and aetiological factors of dentinal hypersensitivity and evaluate relationship to severity.

Material and Methods: The subject population was consisted of 173 patients. All patients were evaluated in respect to aetiological factors related with or without hypersensitivity. Also severity of dentinal hypersensitivity was recorded by using Visual Analoque Scale.

Result: In the survey population, dentinal hypersensitivity was observed as 40,4 % of which was 13,3 % gingival recession, 9,2 % attrision, 5,8 % periodontal disease, 5,2 % abrasion 4,6 % erosion and 2,3 % abfraction. Mean values of Visual Analoque Scale of the factors leading to the dentinal hypersensitivity were observed 4,28 in erosion, 4,25 in abrasion, 3,70 in gingival recession, 3,45 in abfraction, 2,94 in periodontal disease and finally 2,65 in attrision.

Conclusion: This study showed that although attrision followed by gingival recession was the most common aetiologic factor of dentinal hypersensitivity, patients were more affected by non-carious cervical lesions in respect of severity of dentin hypersensitivity.

Key Words: Dentinal hypersensitivity, tooth wear, gingival recession, attrision, erosion, abfraction.

ÖZET

Amaç: Dentin hipersensitivitesinin tedavi stratejisinde tek başına tedaviden ziyade predispozan ve etiyolojik faktörler göz önünde bulundurulmalıdır. Bu çalışmanın amacı dentin hipersensitivitesinin prevelansı ve etiyolojik faktörlerini belirlemek, bu faktörlerle dentin hipersensitivitesinin şiddeti arasındaki ilişkileri değerlendirmektir.

Gereç ve yöntem: Çalışma populasyonu 173 hastadan oluştu. Tüm hastalar dentin hipersensitivitesi ile ilişkili olan ya da olmayan etiyolojik faktörler açısından değerlendirildi. Dentin hipersensitivitesinin şiddeti ise Visual Anolog Skalası kullanılarak kaydedildi.

Bulgular: Populasyonda, % 13,3 dişeti çekilmesi, % 9,2 atrizyon, % 5,8 periodontal hastalık, % 5,2 abrazyon, % 4,6 erozyon ve % 2,3 abfraksiyondan kaynaklanan toplam % 40,4 oranında dentin hipersensitivitesi gözlendi. Dentin hipersensitivitesine neden olan VAS değerleri ortalaması erozyonda 4,8, abrazyonda 4,25, dişeti çekilmesinde 3,70, abfraksiyonda 3,45, periodontal hastalıkta 2,94 ve son olarak atrizyonda 2,65 olarak saptandı.

Sonuç: Bu çalışma, dentin hipersensitivitesinin en yaygın nedenini dişeti çekilmesinden sonra atrizyonun oluşturduğunu, buna rağmen hastaların çürük olmayan servikal lezyonlardan daha çok etkilendiklerini göstermiştir.

Anahtar Kelimeler : Dentin Hipersensitivitesi, diş aşınmaları, dişeti çekilmesi, atrizyon, abrazyon, abfraksiyon**.**

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INTRODUCTION

Dentinal hypersensitivity (DH) has been defined as a short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile osmotic or chemical which can not be described to any other form of dental pathology.[1-6] For dentin to be sensitive, ıt must first be exposed to the oral environment. This exposure may occur by either removal of enamel or denudation of the root surface by loss of the overlying cementum and periodontal tissues. Removal of enamel may occur as a result of non-carious cervical lesions ((NCCLs) (erosion, abrasion, abfraction)) and attrition while exposure of the root may be due to chronic trauma from faulty tooth brushing and habits, acute and chronic inflammatory gingival and periodontal diseases or surgical periodontal treatment.[7,8]

Erosion is the chemical dissolution of tooth structure by acids, which can be intrinsic or extrinsic origin.[9] Abrasion is the mechanic wear of tooth structure by repeated pysical contact principally by tooth brushes and/or abrasive dentin paste.[8,10,11] For abfraction, it has been postulated that the cervical fulcrum area of a tooth is subject to unique stress, torque and moments resulting from occlusal function, bruxing and parafunctional activity. These flexural forces can act to disrupt the normal ordered crystalline structure of the thin enamel and underlying dentin by cyclic fatique leading to cracks, chips and ruptures. Attrition is the loss of tooth tissue due to tooth to tooth contact with no foreign substance intervening and is usually due to parafunctional habits such as bruxism or "grinding" of the teeth.[2,3,12,13]

Gingival recession resulting in exposure of dentinal tubules is the most common cause of dentin hypersensitivity. Chronic exposure to bacterial plaque, tooth brush abrasion, gingival laceration from oral habits such as tootpick use, excessive flossing, crown preperation, inadequate attached gingiva, and gingival loss secondary to disease or surgery are some of the causes of gingival recession. Recessed areas may become sensitive due to the loss of cementum, ultimately exposing dentin.[2,4,14,15]

There are many studies related with prevalance and aetiological factors of DH.[16-19] However, there are limited number of studies on both aetiological factors of DH and associated with DH of these aetiological factors.[20-22] In our studies, we aimed to investigate the prevalance and aetiologic factors of DH, to evaluate relationships between these factors and severity of DH. And also we aimed to investigate relationships between age and these aetiological factors of DH, age and severity of DH.

MATERIAL AND METHODS

The subject population was consisted of 173 patients (108 male, 65 female) with different dental problems who attended to Mareşal Çakmak Military Hospital. The study protocol was approved by the Ethics Committe of the same Hospital and informed consent from all patients was obtained prior to data collection.

All the patients were examinated. Tooth wear (NCCLs and attrision) which had exposed dentin and any evidence of gingival recession were recorded. Restored or carious tooth surfaces and rotationed tooth were excluded from the study. All patients were also examined in the point of view periodontally with CPITN sond. Also severity of DH was recorded by using visual analoque scale (VAS). VAS criteria;

- For each patient, the highest VAS score of each aetiological factor was recorded.
- For each aetiological factor (tooth wear, gingival recession and periodontal disease), we calculated a mean value of VAS by using these highest scores.
- We recorded VAS scores of periodontal disease with CPITN score 3 and 4 which did not include gingival recession.
- If tooth wear, gingival recession and periodontal disease were not associated with DH, they were not taken into consideration calculated for mean values of VAS.
- In determining correlation between age and severity of DH, if the patient has many aetiological factors causing DH, we took into consideration the highest value of VAS.

The investigators evaluated severity of sensitivity by applying a blast of air from an air-water syringe (60 pounds per square inch, 22C°) at a distance of approximately 1mm away by isolating the adjacent teeth by finger. All patients were asked to define their level of DH by using a VAS consisting of

equal units from 0 to 100 (a line of 100 mm). Data from the VAS were recorded by measuring in milimeters the distance between zero point and the sign marked by the patient on the 100-mm line.

Frequency and proportion were calculated. Mann Whitnet U test used to for determine gender differences. Regression cur estimate analysis was used to observe graphics of relationship between age and severity of VAS. In all cases a p-value of less than 0.05 (P<.05) was taken as significant. For these procedures, we used SPSS for Windows, Version 11.0 (SPSS, Chicago).

RESULT

One hundred and seventy three subject were examined with ages ranging from 18 to 61 years (mean age 29,77 years). All of the patients were examined in the point of factors leading to DH. Prevalance of tooth wear, gingival recession and periodontal disease, and also prevalence and percentage of these factors associated with DH and mean values of VAS were given in Table-1. Prevalence of DH was calculated as 40,4 % in all patients.

While gingival recession was observed 52,6 %, only 13,3 % was associated DH in all patients. In the same way, while attrition were observed 41,7 %, only 9,2 % was associated DH in all patients. Abrasion and erosion prevalences were 5,2 %, 4,6 % respectively and all of them were associated with DH. Periodontal disease including CPITN scores 3 and 4 were 13,3 %, only 5,8 % was associated with DH in all patients. Also, Table-1 displays severity of DH. mean value of VAS observed in erosion was 4,28 (the highest value of VAS), in abrasion 4,25, in gingival recession 3,70, in abfraction 3,45, in periodontal disease 2,94 and finally in attrition 2,65 (the least value of VAS).

Correlations between age and tooth wear, age and gingival recession, age and periodontal disease are given in Table-2. Althought the correlations between age and abrasion (P<0.05), age and attrition (P<0.01), age and gingival recession (P<0.01), age and periodontal disease (P<0.01) are significant, erosion and abfraction correlation is not significant (P>0.05).

Age is an important factor in severity of DH. Regression Cubic analyse displays between age and levels of VAS severity in Graphic-I. At 30-40 age VAS level peaks then severity of VAS decreases with age. VAS scores between male and female are significantly different (z=-1,987, P<0.05 Mann Witney U test).

DISCUSSION

DH is a common and painful condition, which can occur on different kinds of provocation factors. Clinic studies and questionnares on DH indicate a prevelance of 4 % to 74 %.[18,23-26] In our study, DH was observed as 40,4 % of which 13,3 % gingival recession, 9,2 % attrition, 5,8 % periodontal disease, 5,2 % abrasion, 4,6 % erosion and 2,3 % abfraction (Table-1).

Table 1. Prevalance of tooth wear, gingival recession, periodontal disease and associated with DH and VAS (Visual analoque scale) means

	Male n	Female n	Age means	Prevalance and percent of aetiologica factors	Prevalance and percent of aetiological factors associated with DH	VAS values means ± Standart Deviation
Attrition	47	25	39,65	72(41.7)	16(9.2)	2,65±0,94876
Abrasion	6	3	35,25	9(5.2)	9(5.2)	4,25±1,96823
Erosion	5	3	32,4	8(4.6)	8(4.6)	4,28±1,10162
Abfraction	2	2	32,5	4(2.3)	4(2.3)	3,45±1,62173
Gingival recession	51	40	35,43	91(52.6)	23(13.3)	3,7±1,55006
Periodontal disease/score 3 and 4 with CPITN	13	10	35,63	23(13.3)	10(5.8)	2,94±1,02328

In our study, we observed 59,6 % tooth wear with exposed dentin including erosion, attrition, abrasion and abfrication in patients with a mean age of 27 years. The prevalence of tooth wear has been reported to be from 5 to 85 percent in various study populations.[14,27] For the majority of the population, any wear on teeth is often limited to enamel, and dentin involvement only occurs in a relatively small proportion of the population.[28] A study by Dugmore and Rock [29] reported that 59,7 % of 1,753, 12-year old children had evidence of tooth wear of which 2,7 % had exposed dentine and this rose to 8.7 by the age of 14 years. Rafeek et al [30] reported that 155 subjects were examined (mean age 40.6 years) of whom 72 % had some degree of tooth wear, with the majority (52 %) exhibiting mild. Smith and et al [22]

reported that one hundred and fifty-six patients with a mean age of 40,6 years were examined of whom 62,2 % had one or more NCCLs.

The prevalence of attrition which had exposed dentin was 41,7 % and attrition was the major aetiological factor of DH followed by gingival recession in all subjects. This finding is similar with Milosevic and Lo [31] who found that 95 % of the subjects were found to have wears in shallow dentine and 41 % in deeper dentin. Saerah and et al [32] (14-77 years-old) reported that while most of the raw tooth wear occurs only in enamel (%76,5), % 23,5 had exposed dentine. All the studies show that attrition makes up the wide proportion of aetiological factors of DH in general population. Erosion prevalence data ranged between 4 and 82 % aged between 18 and 88 in general population.[33] In our study, the prevalence of erosion which had exposed dentin was 4,6 % in all patients. Abrasion and abfraction which had exposed dentin prevalence were respectively 5,2 %, 2,3 % in all patients. Abfraction has been a hypothesis as an aetiological factor in tooth wear. The process is tought to involve eccentric oclusal loading leading to cuspal flexure. This in turn leads to compressive and tensile stresses at the cervical fulcrum area of the tooth with the result of weakened of the cervical tooth structure. The process may be co-destructive rather than directly causal whereby abrasion and/or erosive process are potentiated. It is diffucult to diagnose such lesions properly. Therefore it may be the reason of the lower prevalence of abfraction ın our study.

Althought the prevalence of attrition was found 41,7 % in patients, 9,2 % was associated with DH. Cornelius et al [20] reported that significant percentage of attrition lesions with exposed dentin were not sensitive. Absi et al [34] stated not all exposed dentin is sensitive and in areas of sensitive dentin number of tubules open at the surface was approximately eight times that of non sensitive dentin. Further more, they indicated the mean diamater of open tubules in sensitive dentin was twice those as in non sensitive dentin. They concluded the more open dentinal tubules there are on the surface and greater diameters, the greater the propensity for tubuler fluid flow with given stimulus.[5]

Another finding in our study, all teeth had erosion, abrasion and abfraction lesions were associated with DH. Cornelius and et al [20] reported that while teeth had erosiv lesions with all of them having associated with DH, teeth had abrasion and abfraction lesions were associated with DH 59,7 %, 64 % respectively. This difference may be due to we have evaluated the teeth with exposed dentin. However, there were limited number of studies on these lesions associated with dentin hypersensitivity.

To our study, there were significant correlations between age and attrition, age and abrasion (Table-2). Studies reported that the number of subjects with little or no attrition increased with getting elder while varying degrees of severity of attrition increased with increase in age.[19,28,35] Patients are more likely to have fewer teeth to bear occlusal load, with a loss of the protective mechanisms of natural dentition and diminished quantity and quality of saliva. Also compositional and microstructural changes to enamel and dentin associated with the aging proccess may render the tooth structure more susceptible to lesion formation. There was no correlation between age and erosion. In our study, erosions were observed in the younger patients. It might be associated with the consumption of beverages containing acids in younger people.[36]

Table 2. Relationship between age and tooth wear, age and gingival recession, age and periodontal disease (Spearman rho non parametric correlation).

	Abrasion	Erosion	**Attrition**	**Abfraction**	Gingival recession	Periodontal disease
Age	.178*	.112	.695**	.095	.703**	.237**

*P<0,05 , **P<0,01 statistically significant

In our study, 52,6 % of the population has one or more sites with gingival recession of 1mm or more. Kassab et al [37] reported that more than 50 percent of the population has one or more sites with gingival recession of 1mm or more. Marini and et al [15] reported that gingival recession was observed in at least one dental surface in about 89 % of the individuals analyzed. Gingival recession and subsequent root surface exposure allow more rapid and extensive exposure of dentinal tubules because the cementum layer overlying the root surface is thin and can be easily removed. Although the prevalence of gingival

recession was found 52,6 % in patients, 13,3 % (25 % of gingival recession) associated with DH. Cornelius et al [20] reported that 36,8 % of gingival recession were found to be associated with the dentin hypersensitivity. Like this, our study showed that gingival recessions were not always the cause of DH.

Periodontal disease and/or treatments may play a role in the aetiology of dentin hypersensitivity.[5,24,38,39] In our study, we showed that while periodontitis prevalence was % 13,3 in patients with CPITN score 3 and 4, 5,8 % of all subjects were associated with DH. Chabanski et al [25] reported that the prevalence of DH is between 60 and 98 % in patients with periodontitis. If periodontal disease is left untreated, gingival tissues can be seperated from the teeth and form spaces called pockets that provide a home for bacteria. Periodontal disease can progress until the bone and other tooth supporting tissues are destroyed, are leaving to the root surfaces of teeth exposed. Periodontal pocket may cause DH. Also plaque accumulation in periodontal pocket may be the another factor causing DH. However, conflicting result on the degree of plaque accumulation at sites with DH have been reported. Taani et al [40] reported that there was no correlation for DH with plaque. Pretha et al [41] reported that dentin hypersensitivity alleviating with reducing gingival enflamation. DH is a major problem as many patients are unable to perform adequate oral hygiene in hypersensitive areas thus leading to further plaque accumulation and degradation in gingival or periodontal health. Periodontal therapy including scaling and root planning and pocket elimination surgery are another cause of DH. It has been shown that teeth with periodontally involved roots have a high percentage of viable bacteria within the dentinal tubules, which may cause inflamatory response within the pulp that is in turn expressed as root sensitivity. But, in our study, we did not run across any patient at recall period.

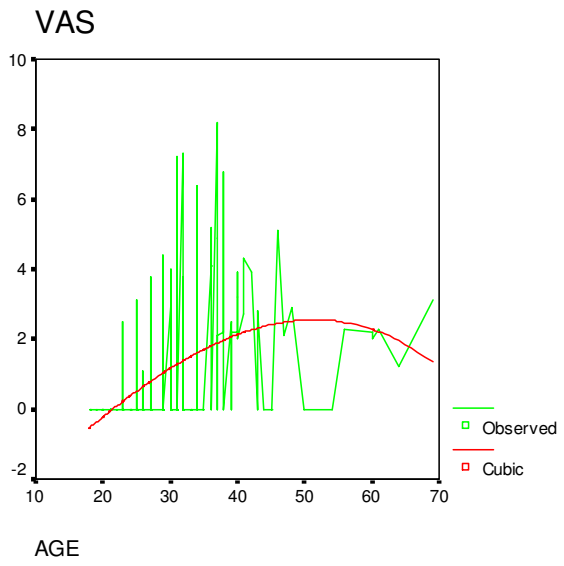
Another finding in our study, gingival recession and periodontal disease were significantly increased with age ($P<0.01$). Similar studies found that frequency of gingival recession and periodontal disease increased with age.[42,43] Some oral changes that are generally thought to be associated with aging including tooth surface loss, periodontal disease and gingival recession. This relationship between the occurance of gingival recession and age may probably be because of the longer period of exposure to the agents that cause gingival recession.[15,44,45]

In our study, we evaluated VAS severity in aetiological factors associated with DH. Mean values of VAS observed in erosion was 4.28 (the most severe of VAS), in abrasion 4.25, in gingival recession 3.70, in abfraction 3.45, in periodontal disease 2.94 and finally in attrition 2.65 (the lower of VAS). Prevalence of gingival recessions associated with DH was higher than another factors. Perhaps, this might be the reason of the lower VAS scores of gingival recession in respect to erosion and abrasion. But generally both gingival recession and NCCL had higher VAS scores. Cao and et al [21] reported that the rate of hypersensitivity rises with the severity of attrition, and the incidence of hypersensitivity in enamel-dentin junction is significantly higher than in dentine concave. Previous study reported that more than 90 percent of hypersensitive surfaces are at the cervical margin on the buccal or labial aspects of the teeth.[5] The cemento-enamel junction is an area of structural weakness where the enamel layer is at its thinnest.[46] However, some patients have a gap revealing a strip of exposed dentine between enamel and cementum.[47] Erosion, abrasion and abfraction are believed to be causative in the formation in this vulnerable area of enamel. Higher VAS scores of erosion and abrasion may be induced by this area as both the most close by the pulp and dentin tubules numbers are fairly too much in this region.[6] In our study, we evaluated of periodontal disease without including gingival recession. We tought this might have caused the lower VAS scores at periodontal diseases. So, ıf periodontal disease which had not gingival recession, severity of DH was observed at lower levels. Attrition had the last VAS severity. Since attrision development tends to be a slow, chronic process that occurs over an extended period, ıt was not suprising to find sclerosis and decrease or lack of sensitivity. Secondary dentin occlusion of open dentinal tubules pulpal retreat and other natural tooth protective measures have slowly adapted to the noxious stimuli thereby minimizing symptoms and maintaining pulpal integrity.[46]

Our results demonstrated that the VAS scores peak at thirties age and then decrease with increasing age for DH (Graphic-I). In previous studies, DH has been shown to peak in 30 to 40 years old.[17,24,25] While

DH mostly occurs in patients who are between 30 and 40 years old, ıt may affect patients at any age.[5,23] A general clinical impression is that elderly people usually are more tolerant to pain. Age is one of the biological factors that has been discussed as important in pain experience. A higher pain threshold in elderly subjects may be a consequence of tissue changes such as reduced vascularity, fatty degeneration and secondary dentin formation.[48]

Our study showed that VAS scores were statistically significantly different between male and female. It affects women more often than man,[17,24] though the sex difference is "rarely statistically significant.[5] However, the pain responce varies substantially from one person to another. It is related to individual tolerance of pain and to physical and emotional factors.[49]



Graphic 1. Age and values of VAS (Visual analoque scale) severity in all patients (Regression Cubic analyse).

CONCLUSION

DH is a problem that plagues many dental patients. This study showed that although attrision followed by gingival recession was the most common aetiological factor of DH, patients were more affected by NCCLs in respect of severity of DH. Also, ıf periodontal disease has not gingival recession, severity of DH associated with periodontal disease was observed at lower levels.

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