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Dentine hypersensitivity in Omani dental patients: a cross-sectional study

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This cross-sectional study was designed to investigate dentine hypersensitivity (DH) and the associated risk factors in patients attending primary oral healthcare clinics in the Muscat Governorate of the Sultanate of Oman. Nine dental practitioners were calibrated against the principal investigator. Patients attending public primary oral healthcare in the Muscat Governorate and who reported DH over a period of one month were evaluated using a questionnaire and an intraoral examination. The duration, initiating factors and two visual analogue scale (VAS) records were used to evaluate patient perception of pain severity associated with dentine hypersensitivity. In addition, the pain perception following an air blast stimulus from a 3-in-1 triple syringe of a dental unit was recorded. Of the 3002 patients recruited for this study, 318 were diagnosed with dentine hypersensitivity, a prevalence of 10.6%. Cold drinks were the main initiating factor with gingival recession present in 56.7%, and cervical abrasion lesions in 12.3% of hypersensitive teeth. There was a statistically significant difference between the VAS of the pain perception of DH and that of an air blast stimulus.

■ Introduction

Dentin hypersensitivity (DH) is a short, sharp pain that arises from an exposed dentine surface in response to thermal, evaporative, tactile, osmotic or chemical stimuli, and it can not be ascribed to any other form of dental defect or pathology[1,2]. The Canadian Advisory Board on Dentine Hypersensitivity (2002) replaced the word 'pathology' in the definition with 'disease'. It can be predisposed by one or a combination of several factors, including loss of enamel, non-carious tooth surface loss, root surface denudation, gingival recession, as well as a sequelae of periodontal therapy[3,4].

The accepted theory regarding DH is the 'hydrodynamic theory' of sensitivity. This theory suggests that a rapid shift of fluids occurs in either direction within the dentinal tubules following stimulus application[5]. This activates sensory nerves in the inner dentine and pulp and hence pain is elicited[6]. It is also believed that there are wider open dentinal tubules and a thinner smear layer in hypersensitive dentine than in non-sensitive dentine[7,8].

Cold is the main initiating factor reported by patients, and it is the stimulus that causes the most trouble to patients[4,9].

DH is relatively common, with variations in the prevalence reported in the literature. The differences

in geographical populations, the clinical setting of the studies, as well as the methodology used for assessment and diagnosis may be the cause for these variations[10-12].

For example, the prevalence reported from a university hospital or periodontal specialist clinic was between 17% and 86%[11-13], whereas those investigated in primary care clinics reported a lower percentage, ranging from 2.8% to 57%[10,14,15].

In a literature search, limited studies that evaluated DH in Middle Eastern and Arabian Gulf regions were found[11,16]. The one study on DH in the Gulf region was conducted in Saudi Arabia in a university dental clinic setting[11].

The objective of the present study was to investigate DH in a cross-sectional population study of patients attending primary oral healthcare clinics in the Muscat Governorate of the Sultanate of Oman. In addition it was intended to investigate any possible predisposing factors (patient oral hygiene practice, smoking habits, gingival recession and cervical abrasion lesion on DH) for patients in this region.

■ Study design

Ethical approval was granted by the Ministry of Health ethical and research committee.

A calibration for buccal gingival recession was initiated, which involved examination of patients by the principal investigator (AA). Reproducibility was checked through re-examination by nine participating examiners; hence a total of 10 examiners participated in data collection. A kappa value of 0.7 was obtained.

Patients attending public services of the primary oral health clinics in the Muscat Governorate of the Sultanate of Oman were then invited to participate in this study. Over the 1-month duration of the study, 3002 patients agreed to participate.

■ Inclusion and exclusion criteria

All permanent teeth were included, except those meeting the following exclusion criteria:

- clinically detectable carious lesion (primary or secondary);
- fractured or cracked tooth syndrome (enamel cracks);
- fracture or restoration with marginal leakage;
- post-restorative sensitivity in a recently restored or crowned tooth;
- palatal-gingival groove;
- patient wearing fixed or removable orthodontic appliance.

Patients (n = 3002) were initially screened by being asked if DH was one of their symptoms[17]. The intention was that a patient's admission or complaint was the main inclusion criterion. Hence the main exclusion criterion was patients who did not report symptoms of dentine hypersensitivity when asked, and therefore the potential examiner error or bias was reduced[12,15].

Two visual analogue scales (VAS) were completed by each patient. One VAS recorded the pain perception of DH, and a second evaluated pain perception immediately following an air blast for 1 sec, from a 3-in-1 triple syringe of a dental unit. During the air blast, the neighbouring tooth was isolated using the examiner's finger[18,19]. Further data were collected in the form of a questionnaire and an intraoral examination.

The questionnaire included information about patient's age, gender, duration of sensitivity, initiating factors, teeth affected, oral hygiene practice (miswak or toothbrushing), cigarette and shisha smoking habits.

Miswak (*Salvadora persica*) is a plant, essentially a chewing stick, used for centuries and considered by Muslims an oral hygiene and spiritual habit[20,21]. Shisha is the word commonly used to describe the tobacco blend smoked through a water pipe. It is relatively common and a recent revival has been observed in the Arabian Peninsula and other countries[22].

The clinical examination assessed and recorded gingival recession, cervical abrasion lesions associated with sensitive teeth, as well as the method employed by general dental practitioners for the management of DH.

■ Statistical analysis

The data were read into an SAS® (version 9.1) database. All summary statistics, frequency distributions, cross-tabulations and formal analyses were performed in SAS®. The formal analysis of the VAS pain

perception of DH was an analysis of variance. Only the factors/covariates that were significant at the 5% level were retained in the model. Interactions between factors and between factors and covariates and quadratic terms of covariates were also considered for inclusion in the model. A square-root transformation was necessary to normalise the residuals.

■ Results

Of 3002 patients (mean age 29.6 years, SD = 9.99, range 11–60 years), 318 were diagnosed with DH, a prevalence of 10.6%. There were 218 females and 100 males, a female to male ratio of 2.2:1.

The highest number of patients with DH was in the 20–29 years age group. The 30–39 years age group had the highest mean number of sensitive teeth (9.65) (Table 1).

Within the 318 patients, 2654 teeth had dentine hypersensitivity. The mandibular incisors (n = 662, 24.9%) were the most commonly affected, followed by the maxillary incisors (n = 464, 17.5%), and the mandibular premolars (n = 396, 14.9%) (Fig 1).

Cold drinks was the main initiating factor in 93.1% (n = 296), followed by cold food in 50.3% (n = 160), and sour food in 27.67% (n = 88) (Fig 2).

On the VAS, the mean patients' DH pain perception score was 5 (SD = 2.24), with the majority (n = 103) recording a score between 4 and 6. The mean patients' pain perception following an air blast stimuli (VAS) was 4.46 (SD = 3.03), with the majority (n = 82) recording a score of 0 to 2 (Table 2). Eight per cent (n = 25) showed no response to the air blast stimulus. There was a statistically significant difference between the VAS of the pain perception of DH and the VAS of the air blast (p = 0.0014).

The mean duration of sensitivity was 2.7 years (SD = 2.98), and 34.3% (n = 109) reported to have had the sensitivity for between 1 and 3 years (Fig 3).

Of the 2654 sensitive teeth, 1505 (56.7%) had gingival recession, with a mean gingival recession of 2.1 mm (SD = 1.16), 26.5% (n = 399) on mandibular incisors and 17.9% on canines (n = 270) (maxillary = 7.2%, mandibular = 10.7%), and 14.8% on mandibular premolars (n = 222) (Fig 4). Most of the gingival recession measurements (89.9%) were in the range of 1–3 mm.

Table 1 Age distribution of patients with dentine hypersensitivity.

Age group	No. of patients with sensitive teeth	Mean no. of sensitive teeth per patient	SD
11–19	43	6.21	5.65
20–29	130	7.96	5.26
30–39	96	9.65	6.04
40–49	30	9.27	5.26
50–59	17	8.29	7.23
60–69	2	3.50	3.54

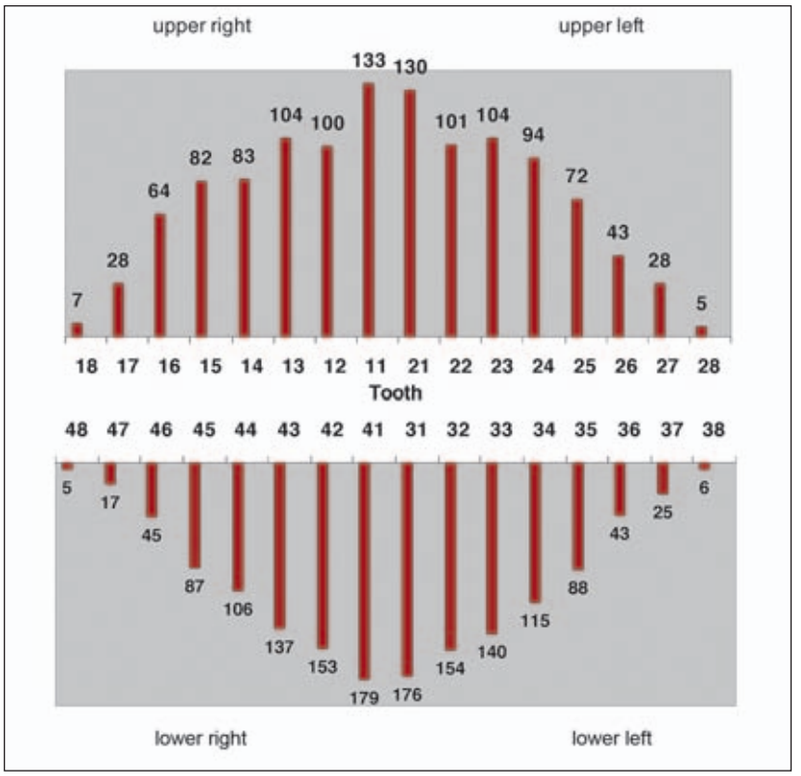

Fig 1 Numbers of teeth with dentine hypersensitivity by tooth number.

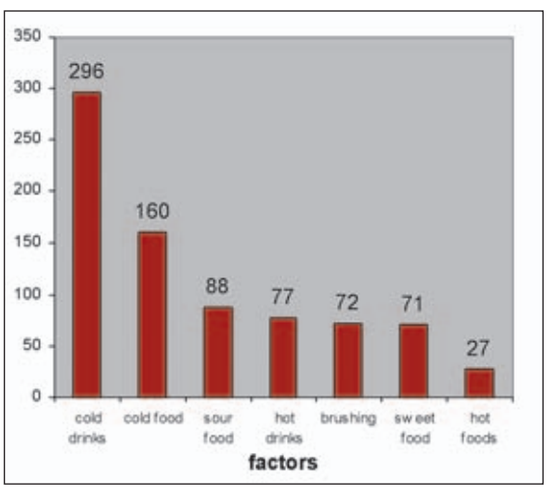

Fig 2 Frequency distribution of the initiating factors of dentine hypersensitivity.

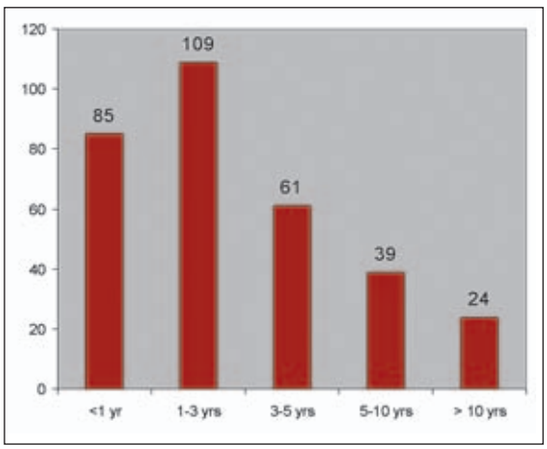

Fig 3 Frequency distribution of the duration of symptoms.

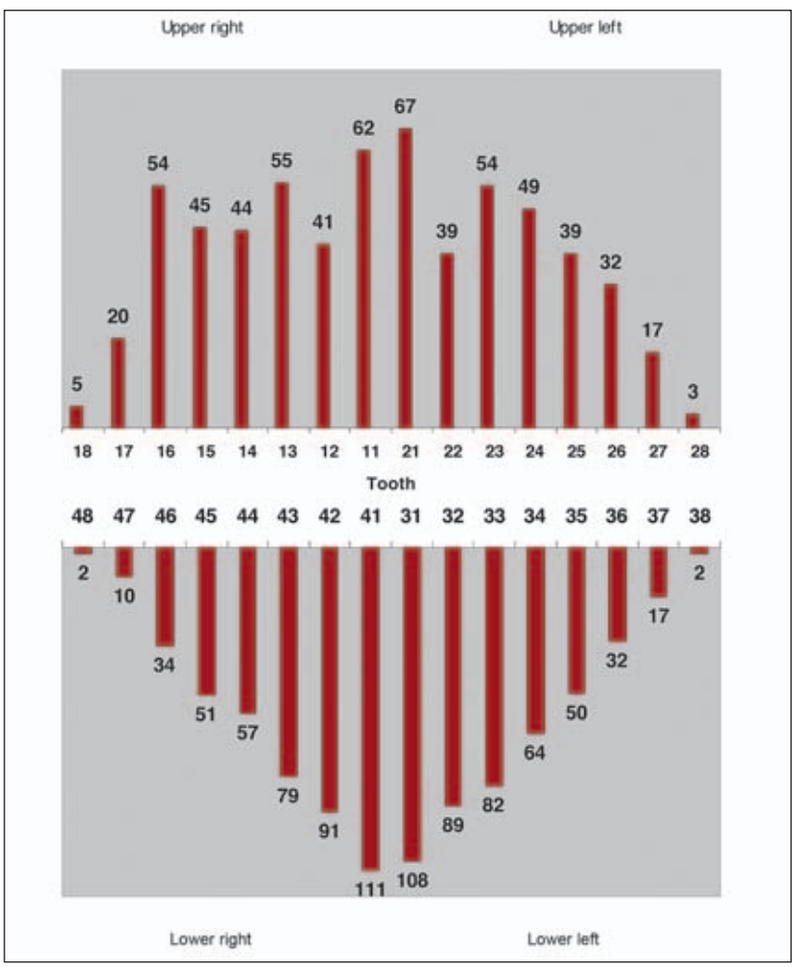

Fig 4 Number of sensitive teeth with gingival recession by tooth number.

Table 2 Visual analogue scale (VAS) of pain perception and after air blast stimulus.

Variable	N	Mean	SD	Minimum	Maximum
VAS pain perception	318	5.00	2.23	0.100	10.00
VAS air blast	318	4.46	3.03	0	10.00

Patients who brushed twice daily (n = 167, 52.52%) recorded a mean number of sensitive teeth of 7.9 (SD = 5.49). Miswak users (n = 29, 9.12%) had a mean average of 8.79 sensitive teeth (SD = 6.17), whereas non-miswak users had a mean of 8.30 (SD = 5.72) (Table 3).

There were 89 (27.99%) patients who used mouthwash, with a mean of 7.85 (SD = 5.28) sensitive teeth. The most frequently used mouthwash (n = 27) was 0.2% chlorhexidine gluconate (corsodyl).

Fourteen patients (4.4%) were cigarette smokers and 11 (3.5%) were shisha smokers (Table 3). Three patients presented with both cigarette and shisha smoking and were mostly males. The mean number of sensitive teeth in patients who smoked cigarettes was higher (10.79, SD = 6.22) than those who were non-cigarette smokers (8.23, SD = 5.71).

Shisha smokers had a higher mean number of sensitive teeth (12.27, SD = 4.94) than those who were non-shisha smokers (8.21, SD = 5.73). The observed correlation between the number of sensitive teeth and the number of cigarettes smoked per day was not statistically significant (r = 0.489, p = 0.075).

Of the sensitive teeth, 326 (12.3%) had cervical abrasion lesions, with premolars being the most commonly affected followed by canines. The mean number of sensitive teeth with abrasion lesions was higher in those who brushed three times or more (1.37) compared with those who brushed once (0.79) daily (Table 3).The most common method used for the management of DH by the general dental practitioners was the prescription of desensitising toothpastes (62.9%), followed by diet analysis and advice (59.4%) and application of fluoride varnish (29.6%).

An analysis of variance of the VAS pain perception of DH was preformed. Since there were 8% who had no response to air blast stimuli despite recording a response on the VAS of pain perception of DH, it was deemed by the authors that the VAS of pain perception of DH response was more representative of patient's pain perception.

Female patients reported higher VAS pain perception than males (p = 0.0382). Patients who had a longer duration of symptoms (p = 0.0356) and patients who reported hot drinks as an initiating factor (p = 0.0475) had higher VAS pain perception. Patients who reported cold drinks as an initiating factor had lower VAS pain perception (p = 0.0130) (Table 4).

■ Discussion

The prevalence of DH (10.6%) in this group was higher than previously reported UK populations[12,14,15]. It was significantly lower than studies from periodontal clinics (98%)[12,23,24]. It was also less than that reported in the only other Arabian Gulf population (42.4% general dental practice and 60.3% periodontal clinic)[11].

There was also a significantly higher prevalence reported in studies in similar primary oral healthcare setting. These studies, however, evaluated DH by self-reporting questionnaire only[10,25-27]. Self-reporting may have overestimated the prevalence in these studies, since a tooth exclusion criterion was not employed.

Variations in reported prevalence could also be due to differences in the populations studied, geographical and cultural backgrounds and the clinical setting of the studies.

There would be less potential for bias if in future studies patients were not asked about dentine hypersensitivity as an inclusion criterion. In addition, a randomly selected sample would be more representative of the population.

Females were more commonly affected by DH, as in other similar studies[10,11,15]. However, gender does not usually have an effect on the reporting of DH[23,24].

Furthermore, the peak age was the 20–29 years age group (n = 130), which was comparable with a study by Orchardson and Collins[9].

Maxillary premolars are usually the most commonly affected, followed by first molars, with incisors being the least sensitive[7,14,15,28]. Incisors were the most commonly affected in the present study, followed by premolars and canines. A similar distribution was reported by Fischer et al[3]. Mandibular incisors in particular were more affected by DH in the present study, concurring with Rees et al[13] and Taani and Awartani[11]. However, patients in the present study were from primary oral healthcare clinics compared with periodontal and oral hygiene clinic patients in the study by Rees et al[13], while in the Taani and Awartani[11] study, the patients were from two separate groups, general dental practice and a periodontal clinic group.

Ture[29] reported a high prevalence of periodontal diseases using a survey that was designed to assess the attitude of Omani people to the state of their teeth, their personal oral hygiene and their acceptance of dental care. Although periodontal disease was not evaluated in the present study, this might have contributed to the similarity with results from studies of cohorts from periodontal clinics.

On the other hand, patients' incorrect and/or vigorous toothbrushing would tend to concentrate on the labial surface of the anterior teeth if enamel discolouration and pitting are present. This is likely in a population with a higher incidence of fluorosis. Studies have shown that Arabian Gulf populations are at risk of fluorosis[30,31]. In addition, a national oral health survey of 15-year-old children indicated that

Table 3 Oral hygiene and smoking habits of patients with dentine hypersensitivity.

Habits			
Oral hygiene	**No. of patients with sensitive teeth**	**Mean no. of sensitive teeth with abrasion lesion**	**SD**
Brushing 0–1/day	43	0.79	1.41
Brushing 2/day	167	0.86	2.17
Brushing ≥ 3/day	108	1.37	2.64
Non-miswak user	289	1.03	2.24
Miswak user	29	0.97	2.57
Non-mouthwash user	229	1.02	2.23
Mouthwash user	89	1.03	2.35
Smoking		**Mean no. of sensitive teeth**	
Non-cigarette smoker	304	8.23	5.71
Cigarette smoker	14	10.79	6.22
Non-shisha smoker	307	8.21	5.73
Shisha smoker	11	12.27	4.94

Table 4 Analysis of variance of VAS pain perception of dentine hypersensitivity.

Effect	**Num df**	**Den df**	**F value**	**P > F**
11–19	1	313	4.33	0.0382
20–29	1	313	4.45	0.0356
30–39	1	313	3.96	0.0475
40–49	1	313	6.25	0.0130

Den, denominator; df, degrees of freedom; Num, numerator

a wide variation in the fluorosis level was present in different regions in the Sultanate of Oman, ranging between 0.6% and 44%, and it was of a moderate to severe degree[32]. This is further evidence that toothbrushing is an important factor for gingival recession and plays some role in influencing the distribution of DH[33,34].

The increase in gingival recession around maxillary incisors in the present study is also thought to be caused by vigorous toothbrushing as an outcome of the high incidence of fluorosis in this region.

Similar to other studies, cold drinks were cited as the main stimulus of DH[3,9,10,13,24,26,27].

Sweet foods were the weakest stimuli, in agreement with other studies[10,14,23,25,26].

As many as 34.3% of patients reported that sensitivity had been present for 1 to 3 years in the present study. Taani and Awartani[11] reported that 23.7% of patients studied had endured the symptom for less than a year. In the present study, 26.7% reported the duration of their sensitivity to be less than one year.

Several studies on DH have evaluated the response on a VAS[16,24,26]. Of the patients in the present study, 8% showed no VAS response to air blast stimulus, although they reported a response from the VAS pain perception of DH. No response to the air blast was similarly reported by Chabanski et al[24] in 6% of their patients.

The Sultanate of Oman average temperature is around 30°C. A cold drink would therefore be colder than the 32–34°C of the air blast. A cold drink could possibly reduce the tooth temperature more in a hot climate than the reduction caused by an air blast. This is a possible reason for the statistical difference observed between the VAS of the pain perception of DH and that of the air blast stimuli ($p = 0.0014$). The air blast stimuli from a 3-in-1 triple syringe for 1 sec at about 32–34°C, although controlled and reproducible, may not be globally representative of the daily DH stimuli. Hence, a global method to measure DH should be investigated, along with a universal index that combines an analogue of pain and the patient's own rating of the effect of pain on their quality of life[17]. Such an index should also take into consideration environmental and climatic effects.

A high percentage (89.8%, n = 1352) of sensitive teeth in this study had 1–3 mm of labial/buccal gingival recession, similar to that reported previously[14,15].

A significant number (52.5%, n = 167) of patients with DH brushed twice daily, while those who brushed nil to once daily had the highest mean number of sensitive teeth. Further investigation is required to evaluate the effect of toothbrushing techniques on this population.

Overall, there were 29 miswak users, and their use of the miswak was combined with toothbrushing. In another study, miswak users were found to have significantly more sites with gingival recession than toothbrush users[21]. In addition, miswak extract in an *in vitro* study resulted in more removal of the smear layer of dentine than chlorhexidine gluconate[35]. Accordingly, increased DH would be anticipated among miswak users. However, no difference in DH was found between the present miswak and non-miswak users. Further studies with a larger sample of miswak users would be required to evaluate the effect of miswak use on DH.

Mouthwashes may contain substances that can erode dentine[36]. The most frequently used mouthwash was 0.2% chlorhexidine gluconate (Corsodyl). The effect of 0.2% chlorhexidine gluconate on the dentine smear layer was investigated by Addy et al[36], and no effect was found in terms of DH. However, 0.2% chlorhexidine gluconate mouthwash may be prescribed to patients as an adjunct in the management of periodontal disease, which was not investigated in the present study; further studies should investigate the relationship between periodontal disease and therapy and DH among Omani patients.

The present study investigated two different smoking habits (cigarette and shisha) in relation to DH. There was no significant correlation between the number of sensitive teeth and the total number of cigarettes smoked per day ($r = 0.489$, $p = 0.075$), in agreement with Rees et al[13]. However, more DH in smokers has been reported[15,16].

In the present study, 11 patients smoked shisha, mostly males between 26 and 42 years old. There was a slightly higher mean number of sensitive teeth (12.3, SD = 4.94) in patients who were shisha smokers compared with those who were non-shisha smokers (8.21, SD = 5.73). There were 14 cigarette smokers and 11 shisha smokers, and 3 of these presented with both cigarette and shisha smoking. Therefore the results regarding smoking habits and

DH must be interpreted with caution. Furthermore the relationship between shisha smoking and DH has not been previously investigated.

Of teeth with DH, 12.3% presented with cervical abrasion lesions, which is less than that reported by Rees et al[13] (23.4%), and Orchardson and Collins[9] (21%). In the first study, the patients were attending a periodontal specialist rather than a primary care clinic. The peak age was between 40 and 50 years, which is higher than in the present study. Cervical abrasion lesions would be more prevalent in older than younger individuals because of the longer exposure to aetiological factors[37]. Premolars, followed by canines, were most commonly affected by cervical abrasion lesions, in agreement with Rees et al[13]. Patients who brushed at least three times daily had the highest number of sensitive teeth with abrasion lesions.

Orchardson and Collins[9] reported a peak age of sensitivity between 20 and 25 years. Their study was in a university dental hygiene clinic, and patients were examined after completion of hygiene phase therapy.

Desensitising toothpastes were the most common method used for the management of DH. This was followed by diet analysis and advice; similar results were reported by Rees[14].

Further studies should investigate the effect of the technique of brushing on DH, the prevalence of periodontal diseases, smoking (cigarette and shisha), and their relationship to DH as well as the relationship between fluorosis and DH in the Omani population.

■ Conclusions

The prevalence of DH in a group of patients attending public services of the primary oral healthcare in the Muscat Governorate of the Sultanate of Oman was 10.6%, and the main predisposing factor was gingival recession.

■ References

1. 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;24:808–813.
2. Addy M. Dentine hypersensitivity: new perspectives on an old problem. Int Dent J 2002;52:367–375.
3. Fischer C, Fischer RG, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro. Brazil J Dent 1992;20:272–276.
4. Dababneh RH, Khouri AT, Addy M. Dentine hypersensitivity: an enigma? A review of terminology, epidemiology, mechanisms, aetiology and management. Br Dent J 1999; 187:606–610.
5. Dowell P, Addy M. Dentine hypersensitivity. A review: aetiology, symptoms and theories of pain production. J Clin Periodontol 1983;10:341–350.
6. Brannstrom M, Astrom A. The hydrodynamics of the dentine: its possible relationship to dentinal pain. Int Dent J 1972;22:219–227.
7. 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;14:280–284.
8. Rimondini L, Baron C, Carrassi A. Ultrastructure of hypersensitive and non-sensitive dentine. J Clin Periodontol 1995; 22:899–902.
9. Orchardson R, Collins WJN. Clinical features of hypersensitive teeth. Br Dent J 1987;162:253–256.
10. Gillam DG, Seo HS, Newman HN, Bulman JS. Comparison of dentine hypersensitivity in selected occidental and oriental populations. J Oral Rehabil 2001;28:20–25.
11. Taani Q, Awartani F. Clinical evaluation of cervical dentine hypersensitivity (CDS) in a patient attending general dental clinics (GDC) and periodontal speciality clinic (PSC). J Clin Periodontol 2002;29:118–122.
12. Rees JS, Addy M. A cross-sectional study of buccal cervical sensitivity in UK general dental practice and a summary review of prevalence studies. Int J Dent Hyg 2004;2:64–69.
13. Rees JS, Jin LJ, Lam S, Kudanowska I, Vowles R. The prevalence of dentine hypersensitivity in a hospital clinic population in Hong Kong. J Dent 2003;31:453–461.
14. Rees JS. The prevalence of dentine hypersensitivity in general dental practice in the UK. J Clin Periodontol 2000;27: 860–865.
15. Rees JS, Addy M. A cross-sectional study of dentine hypersensitivity. J Clin Periodontol 2002;29:997–1003.
16. Al-Wahadni A, Linden GJ. Dentine hypersensitivity in Jordanian dental attenders. A case control study. J Clin Periodontol 2002;29:688–693.
17. Canadian advisory board on Dentine hypersensitivity. Consensus-based recommendations for the diagnosis and management of dentine hypersensitivity. J Can Dent Assoc 2003;69: 221–226.
18. Kleinberg I, Kaufman HW, Wolf M. Measurement of tooth hypersensitivity and oral factors involved in its development. Arch Oral Biol 1994;39:63–71.
19. Ide M, Wilson RF, Ashley FP. The reproducibility of methods of assessment for cervical dentine hypersensitivity. J Clin Periodontol 2001;28:16–22.
20. Khoory T. The use of chewing sticks in preventive oral hygiene. Clin Prev Dent 1983;5:11–14.
21. Eid MA, Selim HA, AL-Shammery AR. The relationship between chewing sticks (MISWAK) and periodontal health. Part III. Relationship to gingival recession. Quintessence Int 1991;22:61–64.
22. Knishkowy B, Amitai Y. Water-Pipe (Narghile) smoking: an emerging health risk behaviour. Paediatrics 2005;116: 113–119.
23. Chabanski MB, Gillam DG, Bulman JS, Newman HN. Prevalence of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department. J Clin Periodontol 1996;23:989–992.
24. Chabanski MB, Gillam DG, Bulman JS Newman HN. Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department. A pilot study. J Oral Rehabil 1997;24:666–672.

25. Gillam DG, Seo HS, Bulman JS, Newman HN. Preception of dentine hypersensitivity in a general practice population. J Oral Rehabil 1999;26:710–714.
26. Irwin CR, McCusker P. Prevalence of dentine hypersensitivity in a general dental population. J Irish Dent Assoc 1997; 43:7–9.
27. Clayton DR, McCarthy D, Gillam DG. A study of the prevalence and distribution of dentine sensitivity in a population of 17–58-year-old serving personnel on an RAF base in the Midlands. J Oral Rehabil 2002;29:14–23.
28. Flynn J, Galloway R, Orchardson R. The incidence of 'hypersensitive' teeth in the west of Scotland. J Dent 1985;13: 230–236.
29. Ture TE. Dental survey: patient attitudes in Oman. Int Dent J 1993;43:585-590.
30. Akpata ES, Fakih Z, Khan N. Dental fluorosis in 12–15-year-old rural children exposed to fluorides from well drinking water in the Hail region of Saudi Arabia. Community Dent Oral Epidemiol 1997;25:324–327.
31. Khalid A. The presence of dental fluorosis in the permanent dentition in Doha. East Mediterr Health J 2004;10:425–428.
32. Ministry of Health. National oral health survey in 15-years old children. Directorate General of Planning. Sultanate of Oman: Ministry of Health, 1996.
33. Sangnes G. Traumatization of teeth and gingival related to habitual tooth cleaning procedures. J Clin Periodontol 1976;3: 94–103.
34. Addy M, Mostafa P, Newcombe RG. Dentine hypersensitivity, the distribution of recession, sensitivity and plaque. J Dent 1987;15:242–248.
35. Almas K. The effect of Salvadora persica extract (miswak) and chlorhexidine gluconate on human dentin: a SEM study. J Contemp Dent Pract 2002;15:27–35.
36. Addy M, Ioyn T, Adams D. Dentine hypersensitivity: effects of some proprietary mouth washes on the dentine smear layer: a SEM study. J Dent 1991;19:148–152.
37. Aw TC, Lepe X, Johnson GH, Mancl L. Characteristics of non-carious cervical lesion: a clinical investigation. J Am Dent Assoc 2002;133:725–733.