## A study of 342 oral keratotic white lesions induced by qat chewing among 2500 Yemeni

Aiman A. Ali<sup>1</sup>, Ali K. Al-Sharabi<sup>2</sup>, José M. Aguirre<sup>3</sup>, R. Nahas<sup>4</sup>

<sup>1</sup>Department of Oral Pathology and Medicine, Faculty of Dentistry, University of Sana'a, Yemen, <sup>2</sup>Department of Periodontology, Faculty of Dentistry, University of Sana'a, Yemen, <sup>3</sup>Department of Stomatology, University of the Basque Country/EHU, Spain, and <sup>4</sup>Consultant Oral Surgery-Implantology, Bremen, Germany

BACKGROUND: Qat chewing is a common habit in Yemen. Various studies demonstrated clear effects of this habit on the systemic organs of the human body. The lack of studies, however, on the effects of this habit on oral mucosa was the major motive of this study.

METHODS: This cross-sectional study was made on 2500 Yemeni citizens (mean age 27 years, 1818 males and 682 females). Clinical protocol was made for all cases with a full intraoral examination. A new grading system was applied for each case with oral white lesion.

RESULTS: Of our sample, 1528 cases (61.12%) were qat chewers; of them, 342 cases (22.4%) had oral keratotic white lesions at the site of qat chewing, while only 6 (0.6%) non-chewer cases had white lesions in their oral cavity (P < 0.000000, Odds ratio = 46.43, RR = 36.26). According to our grading system; 14, 5.9 and 2.4% were grade I, II and III.

CONCLUSIONS: We conclude that qat chewing can provoke the development of oral keratotic white lesions at the site of chewing. The prevalence of these lesions and its severity increase as duration and frequency increase. | Oral Pathol Med (2004) 33: 368-72

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The chewing of qat leaves (*Catha edulis*) is common in certain countries of East Africa such as Ethiopia, Kenya, Somalia and Djibouti, and it is a very common habit in Yemen. The effects of qat were reported in the literature as early as 1237 by the Arabian physician Najuib Al-Deen who proposed the use of qat for the treatment of depressive states (1).

Qat chewing is a habit extensively practised in Yemen by men and women. This habit involves inserting tender leaves of gat into one side of mouth and start chewing it for a while and then storing them in the same vestibular side. The process is repeated until a noticeable pouch is observed (Fig. 1). Qat chewing is usually practised in session that starts after lunch for 2–10 h/day, and during this session, qat chewers always consume water and/or soft drinks. As this habit is more than chewing (chewing and storing), an Arabic word (Takhzeen) is used in Yemen to describe this process.

The growing use of qat has motivated an interest in further knowledge of its active ingredients and their medical and pharmaceutical effects. The most active chemical ingredients of gat are alkaloids such as cathinone and cathine, and tannins such as tannic acid (2). Cathinone is the main psychoactive constituent of qat, and has a similar action to amphetamine. Cathinone produces its central nervous system (CNS) effects by inducing the release of neurotransmitters such as dopamine from the pre-synaptic storage through the same mechanism as amphetamine (3). The effects of qat chewing on cardiovascular system appear rapidly including congestion, tachycardia, palpitation (4) and hypertension (5), while its effects on gastrointestinal tract (GIT) are mainly gastritis and constipation (6). In the mouth, frequent and prolonged time period of qat chewing may cause stomatitis, pain at the site of chewing, stress and staining on teeth (4, 7). Hill and Gibson reported in their study on 127 Yemeni in 1987 that some degree of oral keratosis, but not dysplasia or malignancy, was observed among qat chewers (8); however, they did not make any histopathological study to confirm the absence of dysplasia or malignancy. A recent study concluded that gat chewing increases the risk for several oral lesions including gingivitis, periodontitis, temporomandibular junction (TMJ) click and mouth dryness (9).

The lack of studies on the effects of qat chewing on oral mucosa was the major motive to make this study, and the objectives were to study the prevalence of oral keratotic white lesions in our sample, and to prove the relationship between these lesions and qat chewing as an aetiological factor.



Figure 1 Yemeni chewing qat at the left side.



This cross-sectional study was made on 2500 Yemenis randomly reported to the Faculty of Dentistry, Sana'a University during the period 2/2001–8/2002, mean age 27.01 years varying from 5 to 85 years, SD = 13.4; of them, 1818 (72.7%) were males and 682 (27.3%) females. Individuals included in this study came to the dental school in order to get routine dental procedures such as conservative, prosthetic and surgical extractions. In this country, people rarely come for mucosal lesion diagnosis. Patients with systemic diseases were excluded as some systemic diseases such as renal failure are documented to cause oral white lesions.

A special questionnaire was designed for this study and revised to include maximum information of the exposure under the study 'qat chewing habit' and its effects manifested by various grades of whitening. The questionnaire was filled for each case with a full intraoral clinical examination using the normal examination tools and dental unit light. The questionnaire and clinical examination included the following:

- 1. Personal data: case number, age, sex and occupation.
- 2. Habit's information: a chewer (1) or a non-chewer (0), how many years he has been a chewer (1–40), and frequency (number of qat-chewing sessions per week) (1–7).
- Any other habit that may affect the oral mucosa such as:
  - 3.1. Smoking: a smoker (1–4) or a non-smoker (0), if he is a smoker it has been recorded as: (i) less than 10 cigarettes daily, (ii) more than 10 and less than 20 cigarettes daily, (iii) more than 20 cigarettes daily and (iv) x-smoker (a person who left smoking for more than 1 year).
  - 3.2. Mada'a (water pipe): a smoker (1) or a non-smoker(0).
  - 3.3. Shamma (smokeless tobacco) or what is so-called in some regions as tombak: a user (1) or a non-user (0).
  - 3.4. Any other habits such as tumbol or betel nut chewing: a user (1) or a non-user (0).



Figure 2 Oral keratotic white lesion (grade I) induced by qat chewing.



Figure 3 Oral keratotic white lesion (grade III) induced by qat chewing.

- 4. Clinical examination: includes evaluation of all parts of the oral mucosa, particularly the site of chewing if he is a chewer; according to this evaluation, we coded the normal mucosa as (0); however, in case of a presence of oral keratotic white lesions, it was found that these lesions in qat chewers vary in their clinical features; therefore, we graded it as follows:
  - 4.1. Grade I: mild whitening at the site of qat chewing that is similar to leukoedema as defined in Burket's Oral Medicine (10); see Fig. 2.
  - 4.2. Grade III: a very clear oral keratotic white lesion at the site of chewing that is similar to homogenous leukoplakia as defined in Malmö International Seminar for oral white lesions (11); see Fig. 3.
  - 4.3. Grade II: oral white lesions at the site of chewing which are defined more than grade I and less than grade III.

This study has been conducted by two examiners of good experience (AAA, AKA) according to previous agreement on examination and scoring.

Table 1	The presen	Table 1 The presence of chewers by decades						
Decade	Decade 0-10 11-20	11–20	21–30	31–40	41–50	51–60	61–70	71 and more
Males	%0	331 (61.8%, $n = 536$ )	683 (79.7%, $n =$	108 (90%, $n = 120$ )	857) $108 (90\%, n = 120)$ $96 (84.2\%, n = 114)$ $75 (93.8\%, 0\%, 0\%, 0\%)$	75 (93.8%, 0%	35 (77.8%, $n = 45$ ) 2 (28.6%, $n = 7$ )	2 (28.6%, n = 7)
Females	%0	13 $(8.2\%, n = 159)$	60 (26.5%, $n = 226$ )	80 (63.5%, $n = 126$ ) 25 (43.9%, $n = 57$ )	25 $(43.9\%, n = 57)$	n = 80 20 (48.8%, $n = 41$ ) 0%	%0	%0

All collected data has been fed into a specialised computer program (SPSS) for data analyses, and by using  $\chi^2$ -test reliable results were only documented when  $P \le 0.05$ .

## **Results**

This study was made on 2500 Yemeni citizens (mean age 27.01 years, SD = 13.4); of them, 1818 (72.7%) were males and 682 (27.3%) females.

Of our sample, 1528 cases (61.12%) were qat chewers, most of them were male (1330, 87.04%), while only 198 (12.9%) of chewers were female. This habit started in the 2nd decade, as there is no qat chewer for less than 10 years; however, 61.8 and 8.2% of males and females, respectively, were chewers in the 2nd decade. This percentage increased to become 93.8 and 48.8% of males and females, respectively, in the 6th decade (Table 1).

Of 2500 cases, only 659 (26.36%) were smokers and they were distributed as follows: 295 (11.8%), 174 (7%) and 152 (6.1%) were mild, moderate and severe smokers, respectively. This habit was more common in males than in females (32.8 and 9.4%, respectively). However, the percentage of Mada'a smokers was higher in females than in males (48 (7%) and 67 (3.7%), respectively). In relation to smokeless tobacco, 40 (1.6%) cases of the entire sample were user of Shamma (smokeless tobacco); of them 33 (1.8%, n = 1818) were males and 7 (1%, n = 682) females.

White lesions appeared in 342 (22.4%, n = 1528) qat chewers; these lesions were of various degrees; 215 (14%), 90 (5.9%) and 37 (2.4%) were grades I, II and III, respectively. While only 6 (0.6%, n = 972) white lesions occurred in non-chewers, these lesions were homogenous leucoplakias (GIII in our grading) induced by smoking or smokeless tobacco. These differences statistically were highly significant (P < 0.00000). As we found that white lesions among qat chewers occur more frequently in females than in males, 65 (32.8%, n = 198) and 277 (20.8%, n = 1330), respectively.

According to other aetiological factors related to oral white lesions such as smoking Mada'a and Shamma, we found that oral white lesions appeared in 210 (31.9%, n = 659), 44 (38.3%, n = 115) and 29 (72.5%, n = 40) of smokers, Mada'a users and Shamma users, respectively, while similar lesions occurred in 138 (7.5%, n = 1841), 304 (12.7%, n = 2385) and 319 (13%, n = 2460) of non-smokers, Mada'a non-users and Shamma non-users, respectively. These results statistically were significant (P < 0.0000). However, of all individuals with oral white lesions, 90 (25.9%) cases were only gat chewers but neither smokers nor Mada'a and Shamma users, while the others were gat chewers and users of one or more of the mentioned habits. In relation to the period of chewing and frequency per week and the incidence of oral white lesions, we found that these lesions did not appear in persons who are chewers for less than 2 years; however, 75.7% of these lesions occurred in persons who are chewers for more than 5 years (Table 2), and while only 2.7% of chewers

Table 2 White lesions versus time of chewing: cross-tabulation

	Time of c	hewing				
	Normal	Chewer for 1 year	2–5 years chewer	6–10 years chewer	More than 10 years chewers	Total
White lesions						
Normal mucosa	966	53	611	330	192	2152
	99.4%	100.0%	88.0%	80.5%	51.8%	86.1%
Grade I			65	53	97	215
			9.4%	12.9%	26.1%	8.6%
Grade II			14	22	54	90
			2.0%	5.4%	14.6%	3.6%
Grade III	6		4	5	28	43
	0.6%		0.6%	1.2%	7.5%	1.7%
Total	972	53	694	410	371	2500
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Table 3 White lesions versus frequency of chewing: cross-tabulation

	Frequency of chewing								
	0	One per week	Two per week	Three per week	Four per week	Five per week	Seven per week	Total	
White lesions									
Normal mucosa	966	522	147	32	5	34	448	2152	
	99.4%	97.4%	81.7%	68.1%	50%	75.6%	62.8%	86.1%	
Grade I		12	17	6	5	8	167	215	
		2.2%	9.4%	12.8%	50.0%	17.8%	23.5%	8.6%	
Grade II		2	14	9		3	62	90	
		0.4%	7.8%	19.1%		6.7%	8.7%	3.6%	
Grade III	6		2				35	43	
	0.6%		1.1%				4.9%	1.7%	
Total	972	536	180	47	10	45	710	2500	
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	

who chew once per week presented oral white lesions; however, 37.2% of the daily qat chewers showed same lesions at the site of chewing (Table 3).

## Discussion

Many aetiological factors may provoke the development of oral white lesions; smoking is the most important one (11). The prevalence of these lesions vary from 0.4 to 11.6% all over the world (12); however, through our observations, gat chewers had oral keratotic white lesions higher than normal. This encouraged us to perform this study to prove that gat chewing is another important factor for developing oral white lesions, but the potential malignant transformation of these lesions is questioned and not studied yet. As these lesions are not uniform and vary in their clinical features, none of the international classifications for oral white lesions can be applied on the lesions of our study as they are a special entity. Therefore, we intended to propose a simple classification or grades that are based on international descriptions (10, 11). While our cross-sectional study is not representative for the prevalence of qat chewing in Yemen society, it can demonstrate the morbidity of this habit through the high incidence of oral white lesions among qat chewers, as we found that 22.4% of qat chewers in our sample had oral white lesions while only 0.06% of non-chewers had similar lesions (P < 0.00000, Odds ratio = 46.43, RR = 36.26). Despite that some persons in our sample were smokers and Shamma or Mada'a users, and that the relationship between these habits and the presence of oral white lesions was statistically significant, the site and appearance of these lesions are strongly related to gat habit as all of them occurred at the site of qat chewing and did not extend to any other part of the oral cavity, and the appearance of these lesions vary from mild whitening to homogenous leucoplakia (none of them was non-homogenous), while we know that leucoplakias induced by smoking may also appear as non-homogenous (11, 13) and anywhere in the oral cavity (12). Moreover, when we remove these factors (smoking and smokeless tobacco), the risk remains as 90 of the entire cases with oral white lesions occurred in individuals who were gat chewers but neither smokers nor Shamma or Mada'a users, which supports that gat chewing is the main cause of developing these lesions. On the other hand, the incidence of these lesions increases as time and frequency of gat chewing increase, so that we found 2.6% of chewers who chew one day per week compared to 37% of daily chewers had oral white lesions and 11% of chewers for less than 5 years compared to 48% of persons who are chewers for more than 10 years had oral white lesions, as well as most of the white lesions grade III occurred in persons who are daily qat chewers for at least 10 years, see Tables 2 & 3. These results are in agreement with the general observations of Lugman and

Danowski (1976) (7), and Hill and Gibson (1987) (8) who reported that qat chewing could cause stomatitis and some degree of oral keratosis.

The severity of white lesions in this study was related to frequency and duration of chewing as we found that 95% (n=37) of white lesions GIII occurred in persons who chew qat daily, and 76% of these lesions reported in persons who have been chewing for more than 10 years. However, the presence of severe lesions (GIII) in the oral cavity of qat chewers less than 10 years or the absence of white lesions, in general, in persons who chew qat daily and who have been chewing for more than 10 years may be related to the quantity of qat chewed because the amount of qat consumed during qat sessions varies dramatically among chewers. Qat is marked in Yemen as bundles or bags and not by weight; thus, it was difficult to score the quantity in our study.

The exact aetiology of these keratotic white lesions among qat chewers is not exactly known, but what are the understandable mechanisms so far: the mechanical friction during chewing, the chemical constituents or additives to qat or both of these mechanisms. Finally, we have to mention that the consumption of alcohol had not documented in our study as we are working in an Islamic country where this habit is forbidden and is difficult to ask about it.

According to the above-mentioned observations, we conclude that qat chewing can provoke the developing of various degrees of oral keratotic white lesions. The prevalence of these lesions and its severity increases as duration and frequency increase.

## References

 Lebras M, Fretillere Y. Les aspects medicaux de la consummation habitualle du cath. *Med Trop* 1965; 25: 720–32.

- 2. Afendi AH. Comparative Study of Nutritional Status Assessed by Anthropometrics and Biochemical Measurements as Well as Food Habits of Chosen Population in Yemen and Poland. Poland: A. M. Poznan, 1992.
- Kalix P. Cathinone: a natural amphetamine. J Pharmacol Toxicol 1992; 70: 77–86.
- 4. Halbach H. Medical aspects of the chewing of khat leaves. *Bull WHO* 1972; 47: 21–9.
- Hassan NM, Gunaid AA, Abdulla AA, Abdulkader ZY, Almansoob MK, Awad AY. The effect of qat chewing on blood pressure and heart rate in healthy volunteers. *J Trop Doc* 2000; 30: 107–8.
- 6. Kennedy J. A medical evaluation of the use of qat in the north of Yemen. *Soc Sci Med* 1983; **17**: 783–93.
- Luqman W, Danowski TS. The use of qat in Yemen: social and medical observations. Ann Int Med 1976; 85: 246–9.
- 8. Hill CM, Gibson A. The oral and dental effects of qat chewing. *J Oral Surg* 1987; **63**: 433–6.
- Alsharabi A. Oral and Para-Oral Lesions Caused by Takhzeen (Chewing) Al-Qat. Doctoral Thesis. Al-Khartoum, Sudan: University of Khartoum, 2002.
- Lynch MA, Brightman VJ, Greenberg MS. Burkets Oral Medicine, 9th edn. New York: Lippincott-Raven, 1997.
- Axell T, Holmstrup P, Kramer IR, Pindborg JJ, Shear M. International seminar on oral leukoplakia and associated lesions to tobacco habits 1983; Malmo. Comm Dent Oral Epidemiol 1984; 12: 145–54.
- Aiman A. Evaluación de parámetros clinico-patológicos de importancia pronóstica en la leucoplasia oral. Tesis Doctoral. Leioa (España): Universidad del País Vasco/ EHU, 1997.
- Axell T, Pindborg JJ, Smith CJ, Van Der Wall I. International collaborative group on oral white lesions with special reference to precancerous and tobacco related lesions: conclusion of an international symposium held in Uppsala, Sweden. J O Pathol Med 1996; 25: 49–54.

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