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Nicotine Stomatitis and Its Relation to Carcinoma of the Hard Palate in Reverse Smokers of Chuttas

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Clinical surveys and biopsy studies demonstrated a close correlation among reverse smoking, nicotine stomatitis, and carcinoma of the palate.

Nicotine stomatitis (stomatitis nicotina) of the palate has been described by Thoma.¹ Saunders² found that nicotine stomatitis of the palate was caused by tobacco smoke striking the palate. Orifices of the mucous glands of the hard palate become red and the surrounding mucosa remains normal or is blanched slightly. The lesions occur in the posterior part of the hard palate mucosa and they can become exaggerated. The lesion becomes a firm, painless papule and the red circular area on it becomes an umbilication. The lesions can be multiple. Quigley et al.,³ Schwartz,⁴ Van Wyk,⁵ Sutherland,⁶ and El-Mostehy, Yamane, and Bisada⁷ have studied this lesion macroscopically and microscopically.

We had a unique opportunity to study this lesion in reverse smokers of "chuttas" (homemade cigars) (Figs 1, 2). The habit of reverse smoking, which is common in females in certain parts of India, is called "adda poga." Girls learn this habit from their mothers at an early age. It is a socially acceptable habit. Reverse smoking also is done by men, but to a lesser extent. In the villages 25% of the men and 50% of the women who are more than 20 years old are reverse smokers. Preliminary hospital-based studies have shown that there is a statistically significant relationship between reverse smoking and the development of nicotine stomatitis,⁸ and also between reverse smoking and carcinoma of the hard palate.⁹

Materials and Methods

Six thousand people (2,600 men and 3,400 women) in the dental out-patient clinic of our hospital and 9,400 people (3,955 men and 5,455 women) in the surrounding villages were surveyed. The survey was conducted to determine smoking habits and the occurrence of nicotine stomatitis in different types of smokers and in each sex. Two of us (C.R.R.M.R. and C.R.) graded the nicotine stomatitis lesions as mild, moderate, or severe in 182 persons in the hospital survey and 538 persons in the field survey. Nicotine stomatitis was graded as mild (Fig 3) when red circular areas occurred over a slightly raised blanched mucosa of the glandular zone of the hard palate. The lesion was graded as moderate (Fig 4) when papules about 2 to 4 mm in size with central umbilication less than 2 mm in diameter were observed in the same area. The lesion was graded as severe (Fig 5) when the papules were more than 4 to 5 mm in size with central umbilication more than 2 to 3 mm in diameter. There may be associated pigmentation of the palate.

Biopsy specimens of the palates were taken from 456 people who had nicotine stomatitis. No one had sought medical attention, probably because nicotine stomatitis is symptomfree. Biopsy specimens could be obtained only from people who came to the clinic for dental problems. But the specimens could not be taken from all those with lesions; they were only obtained from patients who could be convinced that biopsy was harmless (148 men and 308 women). The 148 men consisted of 70 ordinary smokers of chuttas; 48 reverse smokers of chuttas, 12 cigarette smokers, and 18 bidi smokers. The 308 women consisted of 280 reverse

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FIG 1.—Woman smoking cigar with lit end inside mouth.

smokers and 28 ordinary smokers of chuttas. The biopsy specimens were studied serially whenever necessary. The serial section study was done only after the first 150 biopsy specimens were seen. The sections were evaluated according to the following criteria: irregular epithelial stratification, basal cell hyperplasia, increased mitotic figures, abnormal mitosis, increased nuclear-cytoplasmic ratio, loss of polarity of cells, cellular and nuclear pleomorphism, hyperchromatism, keratinization of single or groups of cells in the prickle cell layer, and enlarged nuclei in the epithelial cells.

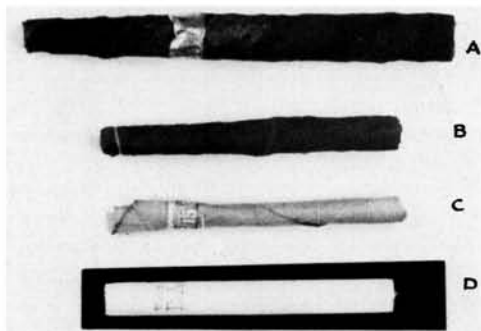


FIG 2.—A, factory-made cigar; B, homemade cigar (chutta); C, bidi; D, cigarette.



FIG 3.—Mild nicotine stomatitis (red circular areas are black in this photograph).

If two to three of these criteria were present, the condition was diagnosed as mild atypical change (Fig 6). If more than three were present, moderate atypical change was diagnosed (Fig 7); and if all were present, severe atypical change (Fig 8) was diagnosed.

Two-hundred fifty consecutive patients with oral carcinomas were studied to determine the prevalence and type of oral cancer. Smoking habits of these people were noted. Of these 250 patients, there were 154 with carcinomas of the hard palate. The presence or absence of nicotine stomatitis was checked, as was the location of the earliest lesions.

Results

Table 1 indicates the type of smoking habit, the number of people, sex, and the number of people with nicotine stomatitis in the hospital and field surveys. Nicotine stomatitis was associated most often with



FIG 4.—Moderate nicotine stomatitis had larger papules with umbilications. Dark area is site of biopsy.

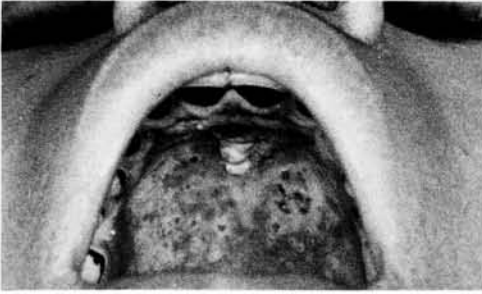


FIG 5.—Severe nicotine stomatitis with larger papules and irregular umbilication.

reverse smoking of chuttas. Next in order of frequency were ordinary smoking of chuttas, bidi smoking, and cigarette smoking. The results of the field and hospital studies were almost the same. A slightly lesser percentage of male reverse smokers had this lesion. This is due to the fact that men do not always smoke in reverse manner. Most of the women are reverse smokers.

Table 2 shows the grading of nicotine stomatitis in association with the various types of smoking habits for both sexes. Severe lesions were associated more often with



FIG 6.—Mild atypical changes of epithelium lining duct of glands (H&E stain; orig mag $\times 60$, reproduced at 87%).

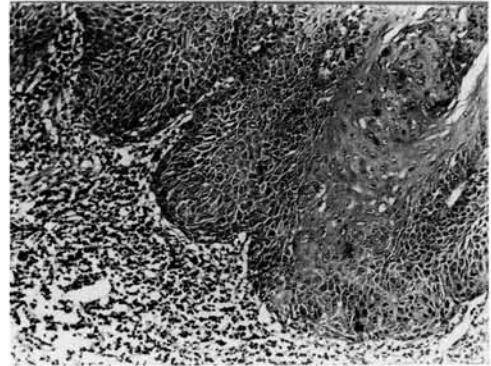


FIG 7.—Moderate atypical changes of epithelium (H&E stain; orig mag $\times 100$, reproduced at 66%).

reverse smoking of chuttas than with ordinary smoking of chuttas. In the cigarette and bidi smokers, no severe lesions were observed. The association between nicotine stomatitis and reverse smoking is apparent.

The histological study was done on 456 biopsy specimens of nicotine stomatitis. The first 150 biopsy specimens were not studied serially; serial section studies were done on the other specimens. Whenever the ducts of the glands were observed in the sections, serial sections were taken. All sections were stained with hematoxylin and eosin (H&E). In the study of the first few biopsy specimens, we thought that the ducts of the glands were obstructed by keratotic plugging.¹⁰ But when the serial sections were studied, we found that the ducts usually were not blocked and the continuity of the lumen of the ducts could be traced. Atypical changes in the surface epithelium usu-



FIG 8.—Severe atypical changes of epithelium (H&E stain; orig mag $\times 60$, reproduced at 66%).

TABLE 1
INCIDENCE OF NICOTINE STOMATITIS

Smoking Habit	Hospital Survey				Field Survey			
	Men		Women		Men		Women	
	N	%	N	%	With Nicotine Stomatitis	%	N	%
Chutta (ordinary smokers)	445	26.29	120	33.33	716	31.43	23	44.92
Chutta (reverse smokers)	258	62.79	638	78.68	413	69.49	1,520	76.00
Cigarette smokers	528	13.07	137	16.82
Bidi smokers	323	21.98	158	20.88

TABLE 2
MACROSCOPIC GRADING OF NICOTINE STOMATITIS

Smoking Habit	Hospital Study						Field Study					
	Men			Women			Men			Women		
	Mild	Moderate	Severe	Mild	Moderate	Severe	Mild	Moderate	Severe	Mild	Moderate	Severe
Chutta (ordinary smokers)	24	7	2	2	60	14	11	3
Chutta (reverse smokers)	8	11	2	27	32	22	25	14	19	109	121	150
Cigarette smokers	18	4	5
Bidi smokers	20	3	6	1

TABLE 3
SMOKING HABITS ASSOCIATED WITH VARIOUS ATYPICAL CHANGES

Smoking Habit	Sex	Mild Atypical Change	Moderate Atypical Change	Severe Atypical Change	Micro-invasive Carcinoma	Total
Chutta (ordinary smokers)	Male	10	10
	Female	4	4
Chutta (reverse smokers)	Male	10	4	14
	Female	84	22	8	11	125
Cigarette smokers	Male	2	2
	Female
Bidi smokers	Male	4	4
	Female
Total		114	26	8	11	159

ally were near the mouths of the ducts of the glands of the palate and extended only a limited way beyond the opening of the ducts.¹¹

Table 3 indicates the association between smoking habits and various atypical changes. Although mild atypical changes were observed in all types of smoking, the moderate and severe types and microinvasive carcinomas (Fig 9) were seen only in reverse smokers. Almost a third of the patients had atypical changes of surface epithelium. Microinvasive carcinoma changes occurred in 11 of the 456 biopsy specimens (2.4%).

The 250 consecutive intraoral carcinomas analyzed consisted of 154 carcinomas of the hard palate, 26 of the tongue, 23 of the cheek, 16 of the retromolar region, 11 of the floor of the mouth, 6 of the soft palate, 5 of the commissural region, 4 of the lip, 3 of the gingiva, and 2 of the oropharynx. Table 4 indicates the type of smoking habits of the 250 patients with intraoral cancer.



FIG 9.—Focus of microinvasive carcinoma (H&E stain; orig mag $\times 30$, reproduced at 66%).

The predominant type of oral cancer in women was carcinoma of the hard palate; carcinomas of other sites were almost insignificant. All but 2 of 110 women with carcinoma of the hard palate were reverse smokers.

Discussion

NICOTINE STOMATITIS.—Thoma¹ described nicotine stomatitis as a lesion that occurs in pipe smokers in regions that are not covered by dentures. Saunders² described nicotine stomatitis as a papular leukoplakia in the palate that is caused by tobacco smoke striking the palate more directly than other regions. He described the red circular areas as the orifices of the mucous glands. Schwartz⁴ also thought that this type of lesion was caused by tobacco. Van Wyk⁵ considered these lesions to be associated with a long history of smoking, especially pipe smoking. Sutherland⁶ reported that these lesions are reversible if smoking is stopped.

Histologically, Thoma¹ described the papule as a retention cyst caused by keratin plugging of the ducts. Saunders² described ulcerative lesions with granulation tissue and chronic inflammation. Van Wyk⁵ observed dysplasia in 2 of 43 people with nicotine stomatitis. He described squamous metaplasia of the ducts and glands, hyperkeratosis and parakeratosis of the mucosa, partial occlusion of the lumen of the ducts, and cyst formation of ducts. Schwartz⁴ and Van Wyk⁵ thought that some of these lesions were precancerous.

Our surveys in the hospital and field have shown that severe nicotine stomatitis occurs most in women who are reverse smokers

TABLE 4
SMOKING HABITS OF PEOPLE WITH INTRAORAL CARCINOMAS

Site of Carcinoma	Total Carcinomas	Men				Women			
		Ordinary Smokers of Chuttas	%	Reverse Smokers of Chuttas	%	Ordinary Smokers of Chuttas	%	Reverse Smokers of Chuttas	%
Palate	154	5/44*	11.36	39/44	88.64	2/110	1.82	108/110	98.18
Tongue	26	9/21	42.85	6/21	28.57	1/5	20.00	0/5	...
Check	23	10/18	55.56	4/18	22.22	0/5	...	4/5	80.00
Retromolar region	16	5/11	45.45	3/11	27.27	1/5	20.00	4/5	80.00
Floor of mouth	11	3/7	42.85	1/7	14.28	0/4	...	3/4	75.00
Soft palate	6	2/5	40.00	2/5	40.00	1/1	100.00	0/1	...
Commissural region	5	1/4	25.00	2/4	50.00	0/1	...	1/1	100.00
Lip	4	0/3	...	2/3	66.67	0/1	...	1/1	100.00
Gingiva	3	0/1	...	0/1	...	2/2	100.00	0/2	...
Oropharynx	2	1/2	50.00	0/2

* Nominator gives the number of persons with the habit and the denominator the total number of carcinomas for that sex at that site.

of chuttas. The lesion occurs in ordinary smokers of chuttas in both sexes and in cigarette and bidi smokers; but in these groups the lesions occur in fewer people and are milder.

Nicotine stomatitis was associated with definite atypical changes in about a third of the patients. The atypical changes ranged from mild to severe. The severe grades of all occurred in the reverse smokers of chuttas, especially in women. Moreover, there were foci of microinvasive carcinomas in 2.4% of the people with nicotine stomatitis in the hospital study. In the field survey, about 2% of the reverse smokers with nicotine stomatitis probably had malignant ulcers.¹²

In our studies, nicotine stomatitis was localized in the glandular zone of the hard palate and did not extend to the exterior half of the palate. Soft palate glands usually do not show this lesion, probably because the number of glands here are less and are not backed by bone, and are embedded in muscle.¹³ In the hard palate region the glands are more numerous and are backed by bone; they may occur as papules because of hyperplasia of the glands. In the anterior half of the palate there are no glands and this lesion does not occur.

The atypical changes of the surface epithelium usually are confined to the mouths of the ducts of the glands and to a little of the surrounding epithelium. In the severe forms, the atypical changes are present

in the lining of the epithelium of the ducts and also to a certain extent in the surrounding epithelium on the surface. The "heaping" of the epithelium because of hyperkeratosis and parakeratosis on the surface around the opening of the ducts contributes to the papule formation. But hyperplasia of the glands must occur to account for the elevation of the papule to even 5 mm over the base of the bone. Moreover, serial sectioning does not indicate dilatation or cystic change of the ducts or dilatation of the glands in the pure nicotine stomatitis lesion. The dilatation of the ducts and atrophy of the glands with interacinar fibrosis was observed only in the older reverse smokers who had been smoking in reverse for more than three to four decades.

Morrow and Suarez¹⁴ have studied the mucosal changes in intraoral smoking in people from Cartagena, Colombia. Biopsy specimens were obtained from 79 persons; 67 of the people were older than 40 years of age and had been reverse smokers for a long time. Interacinar fibrosis was noted; ducts of the glands showed hyperplasia and dilatation, and were filled with fibrin. Thick keratin was found on top of the glands. These findings are similar to our findings in the regressive lesions in long-time reverse smokers. In these people, the papules of nicotine stomatitis regress and become flattened; umbilication becomes shallow and causes a cobblestone appearance and ultimate conversion to leukoplakia. In our

field studies, nicotine stomatitis was found to occur in young women who smoked in reverse for about five to ten years.

CARCINOMAS.—Of the 250 consecutive intraoral carcinomas we observed, 154 were carcinomas of the hard palate. Of these, 110 were in women and 44 were in men. Carcinoma of the hard palate usually starts to the left or right of the midline of the glandular zone of the hard palate as a small ulcer. It involves the whole glandular zone of the hard palate on the left or right side of the midline, and then extends to the opposite side. The extension of the growth on to the anterior half of the hard palate or to the soft palate is not common. Primary carcinoma of the soft palate (6 of 250 intraoral carcinomas) is not common when compared with the hard palate.

In almost all people with carcinomas of the hard palate, nicotine stomatitis also occurred at the site of the growth (Fig 10). The histology of these lesions is similar to the histology of nicotine stomatitis lesions. In some instances there was not enough of the glandular zone of the hard palate left to identify nicotine stomatitis.

Carcinoma of the hard palate occurred more in women than in men. All but 2 of the 110 women and all but 5 of the 44 men with carcinoma of the hard palate were reverse smokers of chuttas. The two women and the five men were ordinary smokers of chuttas.

Nicotine stomatitis and carcinoma of the hard palate occur most in women who are reverse smokers of chuttas. Both occur in the same site in the hard palate. They usually do not extend to the anterior half of the palate or to the soft palate. Nicotine stomatitis does not occur in the cheeks, floor of the mouth, or on the tongue; carcinomas in these regions are not common in reverse

smokers. Histopathological study of nicotine stomatitis lesions shows atypical changes to the extent of microinvasive carcinomas. Carcinoma of the hard palate was associated with nicotine stomatitis in almost all people in whom the glandular zone of the hard palate could be seen.

Thus, there is an association among reverse smoking, nicotine stomatitis, and carcinoma of the hard palate, epidemiologically, statistically, clinically, and pathologically; and it is likely that nicotine stomatitis is a precancerous lesion in reverse smokers.

Conclusions

Six thousand people in our hospital outpatient clinic and 9,400 people in villages were surveyed to determine the prevalence of reverse smoking of chuttas and other types of smoking habits. The occurrence of nicotine stomatitis was noted. In reverse smokers of chuttas, nicotine stomatitis was common. Biopsy specimens (456) of the palate were obtained. A third of the specimens showed atypical changes of the epithelium and 2.4% showed microinvasive carcinomas. Two-hundred fifty patients with intraoral carcinomas were analyzed and carcinoma of the hard palate was found to be the commonest intraoral carcinoma in women. Almost all the carcinomas of the hard palate occurred in reverse smokers of chuttas. Wherever mucosa of the glandular zone of the hard palate could be seen, nicotine stomatitis was present. Thus, there is a close correlation among reverse smoking, nicotine stomatitis, and carcinoma of the hard palate. Nicotine stomatitis could be considered precancerous.

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FIG 10.—Hard palate of reverse smoker shows carcinoma and nicotine stomatitis.

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