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Cross-Sectional Study

Prevalence of Smokeless Tobacco and Areca Nut Use and Its Association with Oral Mucosal Lesions: A Cross-Sectional Study of 1,209 Patients in Northwest India

Hyun-Woo Park[1]*, Oleksandr V. Petrenko[2], Dina M. Saleh[1]

[1] Department of Oral and Maxillofacial Surgery, College of Dentistry, Seoul National University, Seoul, South Korea.
[2] Department of Oral Surgery, Faculty of Dentistry, Dnipro State Medical University, Dnipro, Ukraine.

***E-mail** ✉ hyunwoo.park@gmail.com

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ABSTRACT

The use of chewing tobacco has emerged as a significant public health concern across India. Rates of oral cancer continue to rise in the country, with a noticeable increase among younger individuals. This investigation aimed to determine how common chewing tobacco use is and to explore its relationship with the presence of lesions affecting the oral mucosa. A cross-sectional assessment was carried out with 1,209 patients visiting the Department of Oral Medicine and Radiology at Vyas Dental College, Jodhpur. Data were collected through a structured form that documented demographic characteristics, tobacco habits, and other harmful practices. A single calibrated examiner evaluated the oral mucosa for lesions, and each diagnosis underwent confirmation. Most respondents were men (81.8%) and fell within the 26–35-year age bracket. The most common habit involved chewing mixtures that combined tobacco with areca nut (48.2%). Over 36% of individuals used processed tobacco (90%) and did so at least four times daily. About 25% of participants exhibited tobacco pouch keratosis. Chewing tobacco showed a strong association with the onset of oral mucosal abnormalities, particularly among male users and those from lower socio-economic backgrounds. In addition to targeted cessation programs, modifying the cultural acceptance of chewing tobacco and enforcing strict control measures in both public and workplace settings is crucial.

Keywords: Smokeless tobacco, Areca nut, Oral mucosal lesions, Northwest India

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Introduction

The practice of consuming quid—which may include betel quid, tobacco, areca nut, and related substances—has been rooted in cultural traditions for centuries. This custom is especially widespread throughout South and Southeast Asia but has extended globally due to migration and cultural exchange. Although deeply ingrained in social and cultural life, quid use is increasingly evaluated for its negative health consequences, particularly its link to oral mucosal lesions (OMLs). Conditions such as leukoplakia (LP), erythroplakia, and oral submucous fibrosis (OSMF) are not harmless; many possess premalignant potential and contribute to oral cancer risk [1–3].

The distribution and frequency of quid intake are shaped by a combination of cultural, socio-economic, and behavioral influences. Understanding these elements is essential for developing effective public health strategies that address the harms of quid consumption. This introductory section outlines the epidemiological profile of quid use, including its prevalence patterns, demographic predictors, and the biological pathways connecting quid exposure to OML development. Recent literature is incorporated to

synthesize current insights and identify persisting research gaps.

Epidemiological landscape of quid use

Betel quid and similar preparations are commonly used across several regions worldwide. Countries such as India, Pakistan, Bangladesh, Sri Lanka, and Taiwan report high levels of consumption, and in many communities, the practice carries cultural significance. According to the Global Adult Tobacco Survey (GATS), more than 600 million people worldwide use betel quid, with Southeast Asia showing the most concentrated usage [4]. In India, estimates indicate that 20%–40% of the population regularly consumes betel quid [5]. Motivations vary and may include cultural customs, stress reduction, improved alertness, and socio-economic pressures.

Socio-demographic determinants

The profile of quid users shows recognizable trends. Gender differences are pronounced—men typically use quid more frequently than women, although this disparity is gradually shrinking in certain areas [6]. Age also contributes to usage patterns, with many beginning in adolescence and continuing into middle adulthood. Socio-economic status (SES) remains a major factor; individuals from lower income brackets often have higher usage rates, partly because quid products are inexpensive and widely available compared with alternatives like cigarettes or alcohol [7, 8].

Biological mechanisms and oral mucosal lesions

The development of oral mucosal abnormalities in habitual quid chewers stems from multiple biological pathways. The major ingredients of quid—tobacco, areca nut, and betel leaf—carry numerous harmful agents. Areca nut contains alkaloids such as arecoline, which promote excessive fibroblast activity and increased collagen buildup, contributing to disorders like OSMF [9]. Tobacco products, whether used in smokeless forms or smoked, provide additional carcinogens—including nitrosamines—that further elevate cancer risk [10].

Several forms of OMLs occur in individuals who routinely chew quid. One of the most frequently observed is leukoplakia, which appears as whitish areas on the oral lining, with reported occurrence ranging from 5% to 30%, depending on consumption patterns [11]. Erythroplakia is comparatively uncommon but carries a far higher likelihood of malignant progression, with transformation rates reaching 50% in some reports [12]. Oral submucous fibrosis, a chronic condition with premalignant potential, is especially widespread in South Asian regions, with estimates of 2%–8% among individuals who use quid [13, 14].

Recent population-based research has strengthened the evidence linking quid chewing to OMLs. A Taiwanese longitudinal study tracking 10,000 individuals over ten years documented markedly elevated oral cancer rates among quid users compared to non-users [15]. Likewise, an Indian investigation involving 5,000 participants found that habitual quid chewers had a tenfold greater likelihood of developing leukoplakia, demonstrating a clear dose–response association [16].

At the molecular level, multiple biomarkers have been found in the oral tissues of long-term quid users. Notably, p53 gene alterations occur more frequently in the mucosa of chronic betel quid chewers, suggesting a possible mechanism for malignant progression [17]. Epigenetic disturbances, including DNA methylation variations, have also been tied to OSMF development, illustrating the combined impact of environmental and genetic contributors [18].

The public health consequences of these patterns are substantial. Given the widespread use of quid and its strong link to both OMLs and oral cancer, targeted prevention strategies are essential. Community-oriented education about quid-related harm, coupled with cessation support, is crucial. Stronger regulations over the sale and promotion of quid—particularly where adolescents are concerned—may also help reduce consumption [19, 20].

The present investigation focused on:

(a) measuring how widespread chewing tobacco use is in western Rajasthan,

(b) determining how frequently oral mucosal lesions occur in the same population, and

(c) evaluating the relationship between these lesions and tobacco-chewing habits in this region.

Materials and Methods

Study design

This work involved a descriptive cross-sectional assessment of 1,209 individuals who presented to the Department of Oral Medicine and Radiology at Vyas Dental College, Jodhpur.

Ethical consent

Ethical clearance was granted by the Institutional Review Board (IRB) at Vyas Dental College, Jodhpur. Before participation, every subject received a clear explanation of the study's intent in accessible language, and written consent was obtained to ensure voluntary participation and awareness of their rights.

Sample size estimation and sampling technique

The sample size was based on an expected 52% prevalence of quid use among North Indian populations, with a permissible error of 0.05, resulting in a required minimum of 1,209 participants. Over a one-year period, 6,400 visitors to the Department were screened. From these, 1,209 individuals who consumed processed, unprocessed, or mixed preparations of tobacco and areca nut were chosen using a convenience sampling method.

Participants were excluded if they had stopped quid use for two years or more, or if they reported habits such as smoking, alcohol consumption, drug abuse, or the use of other addictive substances. Additional exclusions included existing amalgam or composite restorations, metal crowns, prior graft procedures, or ongoing management of any oral mucosal lesions.

Data collection

Information for all 1,209 individuals was gathered through interviews conducted in their regional language to ensure accurate reporting of primary symptoms, previous medical details, and demographic background. Socioeconomic classification was determined using the Kuppuswamy scale. Chewing-related behaviors—including the type and physical form of the quid—were documented on a case sheet adapted from a modified WHO Oral Health Assessment Form. Participants were sorted into three principal categories based on quid composition and again into three subgroups according to the form used **(Tables 1 and 2)**. Additional records included the number of times quid was consumed daily, the total years of the habit, and the daily retention time of the quid in minutes.

Classification of oral mucosal lesions

Quid-associated mucosal changes received numerical designations: 0 – Homogeneous leukoplakia, 1 – Non-homogeneous leukoplakia, 2 – Erythroplakia, 3 – Betel chewer's mucosa, 4 – Quid-related lichenoid reaction, 5 – Oral submucous fibrosis, 6 – Tobacco pouch keratosis, 7 – Carcinoma, and 8 – No lesion.

Clinical examination

A standardized protocol was followed using sterilized mouth mirrors and gauze under artificial lighting. Each segment of the oral lining was reviewed in sequence for changes related to quid consumption. Findings were interpreted with updated WHO criteria and verified by an Oral Medicine and Radiology specialist. When lesions suggestive of quid use were identified, incisional biopsy was advised, and consent was obtained from those willing to proceed. Lesions were photographed, counseling regarding habit cessation was provided, and follow-up visits were scheduled.

Calibration of the examiner

A single examiner, trained and calibrated beforehand, conducted all evaluations. Calibration consisted of diagnosing 20 different quid-related lesions assigned by an oral diagnostician, followed by a kappa reliability calculation. After two weeks, 10 of these cases were randomly reassessed to determine intra-examiner consistency. The kappa score was 0.9, reflecting a high level of agreement.

Statistical analysis

Data were entered into Microsoft Excel and analyzed with IBM SPSS Statistics for Windows, Version 22.0 (Armonk, NY, USA). Tests used included the chi-square test, bivariate procedures, and multiple regression. A multinomial regression model examined the association between lesion type (dependent variable) and chewing-related variables such as quid type, quid form, habit duration, and frequency. Age, sex, and socioeconomic class were incorporated as covariates. The threshold for statistical significance was $p < 0.05$.

Results and Discussion

Table 1 presents the demographic distribution of the sample. Most participants were from western Rajasthan, with the largest age band being 26–35 years (28.8%), followed by 16–25 years (26.4%) and 36–45 years (22.1%). Men accounted for 81.8% of the total group and showed higher tobacco usage than women. Socioeconomic categorization revealed that upper-lower (36.1%) and lower-middle (28.9%) groups were predominant. The most frequently reported quid mixture was tobacco combined with areca nut (48.2%), a pairing well known for its link to various OMLs. The processed form of tobacco was overwhelmingly common (90.8%), and such products typically include additives that enhance oral risk. A considerable portion (36.9%) used tobacco 4–35 times per day, a pattern strongly associated with lesion prevalence ($p < 0.01$). Nearly half (45.7%) held the quid in their mouths for 16–70 minutes, and longer retention—up to 1,440 minutes—aligned with more advanced lesions. Regarding the duration of the habit, 49.3% reported chewing for 24–108 months.

Table 1. Distribution of oral mucosal lesions according to socio-demographic details, quid type, quid form, and habit history.

Variable	Category / Response	N (%)
Oral Mucosal Lesions	No lesion	260 (21.5)
	Leukoplakia	128 (10.5)
	Oral submucous fibrosis	254 (21.0)
	Betel chewer's mucosa	18 (1.4)
	Quid-induced lichenoid reaction	62 (5.1)
	Tobacco pouch keratosis	302 (24.9)
	Other lesions	185 (15.3)
Age group (years)	5–15	12 (1.0)
	16–25	328 (26.4)
	26–35	358 (28.8)
	36–45	274 (22.1)
	46–55	103 (8.3)
	56–65	67 (5.4)
	66–75	46 (3.7)
	>75	21 (1.7)
Gender	Female	220 (18.2)
	Male	989 (81.8)
Socio-economic class	Upper class	19 (1.5)
	Upper-middle class	46 (3.7)
	Lower-middle class	359 (28.9)
	Upper-lower class	448 (36.1)
	Lower class	337 (27.1)
Type of quid used	Tobacco only	351 (29.0)
	Areca nut only	275 (22.8)
	Both tobacco + areca nut	583 (48.2)
Form of tobacco consumed	Processed only	1,097 (90.8)
	Unprocessed only	48 (3.9)
	Both processed and unprocessed	64 (5.3)
Daily frequency of chewing	Low (1 pouch/day)	364 (30.1)
(number of pouches per day)	Medium (2–3 pouches/day)	399 (33.0)
	High (≥4 pouches/day)	446 (36.9)
Duration per chew (minutes)	Low (1–15 min)	404 (33.4)
	Moderate (16–70 min)	553 (45.7)
	High (≥75 min)	252 (20.9)
Monthly frequency of chewing	Low (1–18 pouches/month)	191 (15.9)
(total pouches per month)	Moderate (24–108 pouches/month)	597 (49.3)
	High (120–600 pouches/month)	421 (34.8)

Table 2 outlines how different oral lesions relate to demographic attributes, types of quid, their preparation, and patterns of use. The age brackets of 16–25, 26–35, and 36–45 years showed a noticeably greater occurrence of LP, OSMF, QILR, and TPK compared with the remaining groups, with this variation reaching statistical significance ($p < 0.01$). Across every lesion category, men displayed higher rates than women, and this contrast was statistically meaningful ($p < 0.01$). Individuals in the UL and LM economic segments showed elevated levels of these lesions, especially LP, OSMF, QILR, and TPK.

Table 2. Bivariate comparison of oral mucosal lesions across socio-demographic factors.

Variable	Category	No lesion n (%)	Leukoplakia (LP) n (%)	Oral Submucous Fibrosis (OSMF) n (%)	Betel Chewer's Mucosa (BCM) n (%)	Quid-Induced Lichenoid Reaction (QILR) n (%)	Tobacco Pouch Keratosis (TPK) n (%)	Other lesions n (%)	p-value
Age group (years)	5–15	10 (0.8)	1 (0.1)	1 (0.1)	0	0	0	0	
	16–25	102 (8.4)	35 (2.9)	66 (5.5)	5 (0.4)	26 (2.2)	88 (7.4)	26 (2.2)	
	26–35	57 (4.7)	28 (2.3)	79 (6.5)	9 (0.7)	21 (1.7)	89 (7.4)	75 (6.2)	
	36–45	47 (3.9)	28 (2.3)	51 (4.2)	3 (0.2)	9 (0.7)	95 (7.9)	41 (3.4)	
	46–55	28 (2.3)	14 (1.2)	16 (1.3)	0	4 (0.3)	25 (2.1)	16 (1.3)	**0.000***
	56–65	12 (1.0)	10 (0.8)	16 (1.3)	0	0	12 (1.0)	17 (1.4)	
	66–75	3 (0.2)	4 (0.3)	19 (1.6)	1 (0.1)	2 (0.2)	10 (0.8)	7 (0.6)	
	>75	1 (0.1)	8 (0.7)	6 (0.5)	0	0	3 (0.2)	3 (0.2)	
Gender	Female	48 (4.0)	15 (1.2)	72 (6.0)	3 (0.2)	12 (1.0)	22 (1.8)	48 (4.0)	**0.000***
	Male	212 (17.5)	113 (9.3)	182 (15.1)	15 (1.2)	50 (4.1)	280 (23.2)	137 (11.3)	
Socio-economic status	Upper	2 (0.2)	5 (0.4)	6 (0.5)	0	0	3 (0.2)	3 (0.2)	
	Upper-middle	5 (0.4)	4 (0.3)	18 (1.5)	0	2 (0.2)	10 (0.8)	7 (0.6)	
	Lower-middle	71 (5.9)	38 (3.1)	68 (5.6)	4 (0.3)	11 (0.9)	108 (8.9)	59 (4.9)	**0.000***
	Upper-lower	82 (6.8)	42 (3.5)	95 (7.9)	8 (0.7)	22 (1.8)	113 (9.3)	86 (7.1)	
	Lower	100 (8.3)	39 (3.2)	67 (5.5)	6 (0.5)	27 (2.2)	68 (5.6)	30 (2.5)	

BCM: Buccal chewers mucosa
Significant at $p < 0.05$
*Highly significant at $p < 0.001$

Table 3 presents the relationship between lesion profiles and quid characteristics, including type, form, and habit-related variables. Chewing both tobacco and areca nut had a substantially stronger association with OMLs, LP, OSMF, and TPK than chewing tobacco alone ($p < 0.001$). Processed tobacco users appeared frequently among those exhibiting OMLs, with this pattern being statistically robust ($p < 0.001$). Individuals chewing 4–35 times daily showed a marked increase in OML frequency ($p < 0.001$). Prolonged retention of quid (75–1,440 minutes) also aligned with higher lesion prevalence ($p < 0.01$). Participants with chewing histories lasting 24–108 months demonstrated a statistically significant connection with OMLs ($p < 0.001$).

Table 3. Bivariate comparison of lesion types in relation to tobacco form, quid type, and habit duration.

Variable	Category	No lesion n (%)	Leukoplakia (LP) n (%)	Oral Submucous Fibrosis (OSMF) n (%)	Betel Chewer's Mucosa (BCM) n (%)	Quid-Induced Lichenoid Reaction (QILR) n (%)	Tobacco Pouch Keratosis (TPK) n (%)	Other lesions n (%)	p-value
Type of quid used	Tobacco only	69 (5.7)	43 (3.6)	22 (1.8)	0	5 (0.4)	188 (15.6)	24 (2.0)	
	Areca nut only	123 (10.2)	21 (1.7)	80 (6.6)	6 (0.5)	12 (1.0)	16 (1.3)	17 (1.4)	**<0.001***
	Both tobacco + areca nut	68 (5.6)	64 (5.3)	152 (12.6)	12 (1.0)	45 (3.7)	98 (8.1)	144 (11.9)	

Form of tobacco	Processed only	238 (19.7)	113 (9.3)	235 (19.4)	8 (0.7)	54 (4.5)	291 (24.1)	158 (13.1)					
	Unprocessed only	9 (0.7)	4 (0.3)	11 (0.9)	6 (0.5)	3 (0.2)	7 (0.6)	8 (0.7)	**<0.001***				
	Both processed & unprocessed	13 (1.1)	11 (0.9)	8 (0.7)	4 (0.3)	5 (0.4)	4 (0.3)	19 (1.6)					
Daily frequency of chewing (pouches per day)	Low (1 pouch/day)	153 (12.7)	24 (2.0)	44 (3.6)	2 (0.2)	15 (1.2)	95 (7.9)	32 (2.6)					
	Medium (2–3 pouches/day)	79 (6.5)	37 (3.1)	94 (7.8)	6 (0.5)	28 (2.3)	92 (7.6)	63 (5.2)	**<0.001***				
	High (≥4 pouches/day)	30 (2.4)	65 (5.4)	116 (9.6)	10 (0.8)	19 (1.6)	115 (9.5)	90 (7.4)					
Duration each quid kept in mouth (minutes)	Low (1–15 min)	137 (11.3)	33 (2.7)	64 (5.3)	2 (0.2)	20 (1.7)	101 (8.4)	48 (4.0)					
	Moderate (16–70 min)	99 (8.2)	56 (4.6)	126 (10.4)	6 (0.5)	32 (2.6)	147 (12.2)	87 (7.2)	**<0.001***				
	High (≥75 min)	24 (2.0)	39 (3.2)	64 (5.3)	10 (0.8)	10 (0.8)	54 (4.5)	50 (4.1)					
Overall duration of habit (months)	Low (1–18 months)	73 (6.0)	14 (1.2)	33 (2.7)	4 (0.3)	14 (1.2)	39 (3.2)	14 (1.2)					
	Moderate (24–108 months)	133 (11.0)	66 (5.5)	120 (9.9)	9 (0.7)	36 (3.0)	153 (12.7)	80 (6.6)	**<0.001***				
	High (120–600 months)	54 (4.5)	48 (4.0)	101 (8.4)	5 (0.4)	12 (1.0)	110 (9.1)	91 (7.5)					

BCM: Buccal chewers mucosa
Significant at $p < 0.05$
*Highly significant at $p < 0.001$

Table 4 summarizes the multinomial logistic regression exploring how lesions (dependent variable) relate to multiple predictors.

Table 4. Multinomial logistic regression assessing the association between lesions and independent variables.

Oral Lesion	Predictor Variable	Adjusted Odds Ratio (95% CI)	p-value
Leukoplakia	Age (per year increase)	1.716 (1.336–2.204)**	<0.001
	Male gender (vs female)	1.732 (0.893–3.358)	0.104
	Socio-economic status (lower vs higher)	1.492 (1.014–2.195)*	0.042
	Type of quid (mixed/both vs single)	1.061 (0.801–1.405)	0.679
	Form (processed vs unprocessed)	0.913 (0.580–1.437)	0.694
	Daily frequency of consumption (higher)	4.061 (2.784–5.923)**	<0.001
	Duration per quid in mouth (longer)	0.948 (0.637–1.409)	0.790
	Overall duration of habit (longer)	1.377 (0.964–1.965)	0.078
Oral Submucous Fibrosis (OSMF)	Age (per year increase)	1.416 (1.129–1.776)**	0.003
	Male gender (vs female)	0.546 (0.344–0.868)**	0.010
	Socio-economic status (lower vs higher)	1.098 (0.785–1.537)	0.585
	Type of quid (mixed/both vs single)	1.954 (1.520–2.511)**	<0.001
	Form (processed vs unprocessed)	0.601 (0.389–0.929)**	0.022
	Daily frequency of consumption (higher)	2.977 (2.163–4.098)**	<0.001
	Duration per quid in mouth (longer)	1.032 (0.739–1.442)	0.854
	Overall duration of habit (longer)	1.546 (1.152–2.076)**	0.004
Quid-Induced Lichenoid Reaction	Age (per year increase)	1.012 (0.527–1.943)	0.972
	Male gender (vs female)	0.645 (0.165–2.516)	0.527
	Socio-economic status (lower vs higher)	1.180 (0.453–3.077)	0.735
	Type of quid (mixed/both vs single)	1.989 (0.855–4.628)	0.111
	Form (processed vs unprocessed)	3.173 (1.693–5.945)**	<0.001
	Daily frequency of consumption (higher)	3.173 (1.374–7.324)**	0.007
	Duration per quid in mouth (longer)	1.789 (1.033–3.099)**	0.038
	Overall duration of habit (longer)	0.769 (0.336–1.761)	0.534
Tobacco Pouch Keratosis	Age (per year increase)	1.124 (0.762–1.659)	0.556
	Male gender (vs female)	0.776 (0.371–1.622)	0.500
	Socio-economic status (lower vs higher)	1.280 (0.741–2.212)	0.376
	Type of quid (mixed/both vs single)	2.672 (1.697–4.208)**	<0.001
	Form (processed vs unprocessed)	1.111 (0.648–1.907)	0.701
	Daily frequency of consumption (higher)	2.182 (1.342–3.550)**	0.002
	Duration per quid in mouth (longer)	0.890 (0.528–1.498)	0.660
	Overall duration of habit (longer)	0.960 (0.613–1.502)	0.857

Significant at $p < 0.05$

*Highly significant at $p < 0.001$

Leukoplakia

People who chewed both unprocessed and processed tobacco with areca nut showed a 1.06-fold greater probability of leukoplakia compared with those using processed tobacco alone (OR = 1.08, 95% CI: 0.8–1.4). High-frequency chewers exhibited roughly a fourfold increase in risk (OR = 3.92, 95% CI: 2.78–5.92). Advancing age was linked to a 1.7-times higher likelihood of LP (OR = 1.71, CI: 1.3–2.2). Men faced a 1.7-fold higher risk relative to women (OR = 1.73, CI:

0.89–3.35). Subjects from UL and L socio-economic groups showed about a 1.5-times higher chance of LP than those from upper classes (OR = 1.01–2.19). Long-term chewers had a 1.37-fold greater probability of leukoplakia compared with short-term chewers (OR = 1.37, 95% CI: 0.96–1.96).

OSMF

Individuals using both tobacco and areca nut were almost twice as likely (1.9-fold) to develop OSMF as those relying solely on tobacco (OR = 1.92, 95% CI: 1.52–2.51). Chewing at higher frequencies increased the probability nearly threefold (OR = 2.9, 95% CI: 2.16–4.09). Those with moderate-length chewing histories were 1.5 times more susceptible (OR = 1.54, 95% CI: 1.15–2.07). Older participants were 1.4 times more likely to develop OSMF (OR = 1.4, CI: 1.12–1.77). UL and L economic groups showed a 1.1-times increased risk compared with upper classes (OR = 1.09, CI: 0.78–1.5).

Quid-induced lichenoid reaction (QILR)

Tobacco combined with areca nut increased the odds of QILR by 2.7 times compared with tobacco-only users (OR = 2.67, 95% CI: 1.69–4.2). Use of both unprocessed and processed tobacco with areca nut showed a 1.1-fold rise relative to processed-tobacco-only users (OR = 1.11, 95% CI: 0.64–1.9). A chewing frequency on the higher end raised the likelihood by 2.2 times (OR = 2.18, 95% CI: 1.34–3.55). Age contributed to a modest 1.12-fold increase (OR = 1.12, CI: 0.76–1.65). Men displayed roughly 1.3-times higher risk than women (OR = 1.28, CI: 0.74–2.21).

Tobacco pouch keratosis group

Individuals with high-frequency chewing showed a markedly greater likelihood (2.9-fold) of developing TPK when compared with low-frequency users (OR = 2.9, 95% CI: 2.13–3.95). Advancing age slightly increased susceptibility, with older adults having about 1.1 times the risk relative to younger individuals (OR = 1.1, CI: 0.9–1.36). Male participants demonstrated substantially higher vulnerability, being 2.9 times more prone to TPK than females (OR = 2.13, CI: 1.53–4.76). Those with a moderate duration of chewing habits exhibited a 1.5-fold increase in TPK likelihood compared with shorter-duration users (OR = 1.55, 95% CI: 1.17–2.06).

This investigation examines quid-use patterns and their connection to OMLs in western Rajasthan. The analysis identified strong links between various quid compositions and the presence of LP, OSMF, QILR, and TPK. These observations are consistent with earlier research from related regions, providing a broader context for risk elements associated with quid-related mucosal changes [21, 22].

The most affected ages were 26–35 years, followed by the 16–25 and 36–45 groups, indicating heightened vulnerability among younger adults. The statistically significant association across these brackets mirrors prior studies from India and Southeast Asia, where similar age distributions were found at increased risk [23, 24]. Gupta *et al*. also noted peak OML prevalence in the 20–40-year age band, supporting comparable demographic tendencies [5].

This concentration of cases among younger adults is often attributed to early initiation of quid use during adolescence, resulting in extended exposure by early adulthood. Thomas *et al.* reported that both early uptake and prolonged usage substantially raise OML risk [25]. Social pressures and stress common in these age clusters may further contribute to higher quid consumption [1].

The present study identified a pronounced gender difference, with men displaying significantly higher OML rates than women—an outcome repeatedly reported in previous literature [24]. Cultural norms in western Rajasthan may facilitate more frequent quid consumption among men. Research by Warnakulasuriya *et al*., Tsai *et al*., and Lee *et al*. similarly documented greater lesion prevalence in male users, likely connected to higher chewing frequency and longer usage periods [1, 10, 15].

Biological responses to quid components may also vary by sex, as men often have higher rates of co-exposures such as smoking and alcohol, which are recognized aggravating factors in South Asian populations [22]. These gender-linked differences point to the need for targeted prevention strategies.

Socio-economic status also emerged as an important variable. Individuals from UL and LM SES groups experienced higher OML burdens, consistent with the findings of Boffetta *et al*., who noted elevated quid use in lower-income segments due to affordability and accessibility [7]. Limited healthcare access and reduced awareness further heighten susceptibility in these groups. Similar SES-related disparities have been documented in Sri Lanka and Pakistan [3, 26]. These trends highlight the necessity of community-focused interventions aimed at high-risk economic strata.

The study also identified the combination of tobacco and areca nut as the most prevalent quid type, strongly associated with LP, OSMF, and TPK. This supports previous observations from South Asia, where this mixture is common and recognized as a major predictor of lesion development. Nair *et al*. noted its particular connection to OSMF in Indian populations [8].

Processed quid forms were the most frequently consumed and showed significant associations with multiple lesions. Javed *et al*. previously described processed products as containing higher carcinogen levels due to additives and flavor enhancers [14]. Chung *et al*. similarly reported increased oral cancer incidence among processed-quid users [9]. These findings suggest that chemical alterations during processing intensify pathogenic potential.

Parallel results from Taiwan and Malaysia also link processed quid products with greater OML and oral cancer prevalence [2, 23]. Such evidence strengthens the argument for stricter oversight on the manufacturing and distribution of processed quid.

High-frequency quid use (4–35 times daily) showed a strong link with the presence of OMLs, a trend that mirrors earlier reports [6]. Petersen *et al*. noted that frequent chewing raises OML risk because the oral tissues are repeatedly exposed to irritants [12].

The study further indicated that moderate or prolonged periods of quid held in the mouth were tied to greater OML occurrence, echoing Tsai *et al*.'s observation that extended quid–mucosa contact heightens lesion development [10].

Long-term chewing habits were also implicated. Lee *et al*. documented that individuals with over 10 years of quid use faced considerably higher odds of oral cancer [2]. These combined insights underline the value of reducing both chewing frequency and retention time to limit OML prevalence among vulnerable groups.

Specific lesions and contributing factors

Leukoplakia

Participants who used tobacco–areca nut mixtures in either raw or processed forms exhibited higher LP rates. This parallels conclusions by Warnakulasuriya *et al*., who described a strong LP association with the combined habit [1].

Older adults were disproportionately affected, supporting Shah *et al*.'s findings that age-related cumulative exposure increases LP susceptibility [3].

Men also showed elevated LP risk, consistent with patterns reported by Lee *et al*. and Chiu *et al*., both noting greater LP frequency in male chewers [2, 6].

Higher LP likelihood among individuals from UL and LM SES groups further matches previous work that highlighted socio-economic influences on oral health disparities [5].

Oral submucous fibrosis

OSMF appeared more frequently in those who chewed both tobacco and areca nut—especially processed variants. This outcome aligns with the conclusions of Nair *et al*. and Murti *et al*., who identified this combination as a dominant OSMF determinant [8, 11]. Heavy daily use and extended chewing periods were also linked with OSMF, supporting Thomas *et al*., who showed that cumulative exposure promotes progressive fibrosis [16].

The increased vulnerability among older individuals echoes reports by Shah *et al*. and Gupta *et al*., attributing this pattern to longer exposure durations [3, 5].

A modest rise in risk among UL and LM SES participants mirrors socio-economic patterns previously described by Boffetta *et al*. and Jacob *et al.* [7, 13].

Quid-induced lichenoid reaction

QILR was strongly associated with combined tobacco–areca nut use, consistent with earlier literature. Winstock *et al.* observed that QILR occurred more often in mixed-substance chewers than in tobacco-only users [22]. The reaction's inflammatory basis suggests that irritants—particularly in processed products—may provoke immune-driven mucosal changes. High-frequency chewing was also tied to QILR, reinforcing Lee *et al*.'s findings of increased QILR in individuals who chewed multiple times per day [2]. This study additionally indicated slightly higher QILR rates in men and older participants, though these trends were weaker than those seen in other lesions. Thomas *et al*. reported similar demographic tendencies among male quid users [25].

Tobacco pouch keratosis

TPK appeared more frequently among people who used tobacco often, particularly when relying on processed varieties. This pattern corresponds with observations by Chung *et al*. and Javed *et al*., who highlighted high chewing frequency and processed tobacco use as major contributors to TPK development [9, 14]. The link between TPK and extended periods of quid held in the oral cavity also reflects findings by Warnakulasuriya *et al*., who demonstrated that longer mucosal exposure to tobacco increases the likelihood of keratotic changes [1].

Men showed a higher probability of developing TPK than women, echoing the trends noted by Shah *et al.* and Petersen *et al*., both reporting greater TPK prevalence in male chewers [12, 19]. The pattern of increased TPK risk among individuals in the UL and LM socio-economic categories reinforces earlier evidence that economic background influences vulnerability and must be considered in preventive planning.

This investigation has certain constraints. Its cross-sectional design offers only a snapshot of current conditions, and because the data were collected from patients at a dental college in western Rajasthan, the findings should be interpreted cautiously.

Conclusion

This study enhances understanding of how different quid-related habits contribute to the onset of oral mucosal conditions. Comparison with previous research shows that combining tobacco with areca nut, particularly in processed preparations, significantly elevates the likelihood of lesions, including leukoplakia, oral submucous fibrosis, quid-induced lichenoid response, and tobacco pouch keratosis. Strong associations with age, sex, SES groups, frequency of chewing, and time spent retaining quid highlight the need for targeted public health strategies addressing these risks. Continued investigation and inter-regional cooperation will be important for designing effective approaches to reduce the impact of quid-related oral disorders.

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