

Impact of Tobacco Use on Oral Health, Pulmonary Disorders, Oral Cancer, and Lung Cancer: An Original Observational Study.

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Received: 08-04-2026

Revised: 20-04-2026

Accepted: 07-05-2026

Published: 30-05-2026

Abstract

Background: Tobacco use is a major risk factor for both oral and respiratory diseases, causing damage to the oral mucosa, periodontal tissues, and respiratory epithelium. However, integrated assessment of dental and pulmonary health among different types of tobacco users remains limited in routine clinical practice. This study aimed to evaluate the association between tobacco use patterns and oral as well as pulmonary health outcomes. **Materials and Methods:** This comparative observational study included 500 adults aged 18–75 years. Participants were categorized into four groups: non-users, smoked-tobacco users, smokeless-tobacco users, and dual users. Oral health assessment included the Oral Hygiene Index-Simplified (OHI-S), Community Periodontal Index (CPI), oral mucosal examination, and biopsy when indicated. Pulmonary evaluation comprised assessment of respiratory symptoms, spirometry, chest radiography, and computed tomography when clinically required. Statistical comparisons were performed among the four groups. **Results:** The mean age of participants was 43.8 ± 12.6 years, and 68.2% were male. Poor oral hygiene was significantly more prevalent among tobacco users, particularly dual users (48.8%), compared with non-users (12.8%) ($p < 0.001$). Moderate-to-severe periodontitis was observed in 58.4% of dual users versus 18.4% of non-users. Oral potentially malignant disorders were identified in 3.2% of non-users, 10.4% of smoked-tobacco users, 22.4% of smokeless-tobacco users, and 28.8% of dual users ($p < 0.001$). Obstructive spirometric abnormalities were most common among smoked-tobacco users (28.0%) and dual users (34.4%). The prevalence of oral cancer increased across groups from 0.8% in non-users to 5.6% in dual users, while lung cancer was predominantly detected among smoked-tobacco and dual users. **Conclusion:** Tobacco use demonstrated a significant dose- and pattern-dependent association with poor oral hygiene, periodontal destruction, oral potentially malignant disorders, obstructive pulmonary dysfunction, oral cancer, and lung cancer. Dual tobacco users exhibited the highest disease burden. These findings highlight the importance of integrating tobacco cessation counseling, oral cancer screening, and respiratory health evaluation into routine dental and medical care to facilitate early detection and prevention of tobacco-related diseases.

Keywords: tobacco; oral health; periodontitis; chronic obstructive pulmonary disease; oral cancer; lung cancer.



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INTRODUCTION

Tobacco use is one of the most preventable causes of morbidity and mortality worldwide and remains strongly linked with malignancy, chronic respiratory disease, cardiovascular disease, and adverse oral health outcomes. Global burden analyses demonstrate that tobacco-attributable disease continues to affect both high-income and low- and middle-income countries, with substantial disability and premature mortality among adults of working age [1]. The carcinogenicity of smoked tobacco and involuntary smoke exposure has been established for multiple organs, particularly the lung, oral cavity, pharynx, larynx, and esophagus [2,3].

The oral cavity is often the first anatomical site exposed to tobacco constituents. Smoked and smokeless products contain nicotine, tobacco-specific nitrosamines, polycyclic aromatic hydrocarbons, aldehydes, and heavy metals that alter epithelial integrity, salivary function, local immunity, and the oral microbiome. Tobacco users are therefore at increased risk of tooth staining, halitosis, delayed wound healing, gingival recession, periodontal attachment loss, leukoplakia, oral submucous fibrosis, erythroplakia, and oral squamous cell carcinoma [4,5]. The relationship between tobacco and periodontal disease is biologically plausible because nicotine-mediated vasoconstriction, impaired neutrophil function, oxidative stress, and altered fibroblast activity reduce host defense and periodontal repair [6].

Smokeless tobacco is particularly relevant in South Asian settings because products such as gutkha, khaini, zarda, and tobacco-containing betel quid are held in direct contact with the buccal mucosa and gingiva for prolonged periods. Meta-analytic evidence has shown a significant association between smokeless tobacco use and oral cancer, with risk varying by region, sex, and product type [7]. Similarly, smoked tobacco produces chronic airway inflammation, mucus hypersecretion, impaired mucociliary clearance, emphysema, chronic obstructive pulmonary disease (COPD), and lung cancer [8,9]. Persistent smoking accelerates lung function decline, whereas cessation improves respiratory outcomes and may reduce mortality in COPD patients [10].

Although the independent effects of tobacco on oral disease, COPD, oral cancer, and lung cancer are well established, clinical practice often separates dental and pulmonary screening. Patients who attend dental clinics may not undergo respiratory risk assessment, and patients attending respiratory clinics may not receive structured oral mucosal screening. This separation can delay recognition of multisystem tobacco damage. The present study was therefore designed to evaluate the combined impact of tobacco-use pattern on oral health status, pulmonary disorders, oral potentially malignant disorders, oral cancer, and lung cancer in an adult outpatient population. The aim was to compare oral and respiratory outcomes among non-users, smoked-tobacco users, smokeless-tobacco users, and dual users, and to identify clinical indicators associated with higher disease burden.

MATERIALS AND METHODS

Study design and setting: This comparative observational study was conducted at Rajurkar Hospital Chest, Sleep Institute and Dental Clinic, Chandrapur, Maharashtra, over 18 months. The study followed institutional ethical principles, and written informed consent was obtained from all participants before examination.

Study population: Adults aged 18–75 years attending outpatient services were screened. Participants were categorized into four equal groups: Group A, non-users of tobacco; Group B, smoked-tobacco users; Group C, smokeless-tobacco users; and Group D, dual users of smoked and smokeless tobacco. A total sample size of 500 was finalized with 125 participants in each group. Tobacco exposure was recorded using a structured questionnaire that included product type, frequency, duration, age at initiation, pack-years for smoked tobacco, and frequency-years for smokeless tobacco.

Inclusion and exclusion criteria: Participants with at least one year of regular tobacco exposure were included in the tobacco-user groups. Non-users had no current or past regular tobacco habit. Patients with previously treated head-and-neck cancer, active pulmonary tuberculosis, known interstitial lung disease unrelated to smoking, edentulous status preventing periodontal scoring, immunosuppressive therapy, or incomplete diagnostic evaluation were excluded.

Oral assessment: A trained dental examiner recorded oral hygiene status using the Oral Hygiene Index-Simplified (OHI-S), gingival bleeding, periodontal pocket depth, and Community Periodontal Index (CPI). Periodontitis severity was categorized as none/mild or moderate/severe according to clinical pocketing and attachment loss. Oral mucosal examination was performed under adequate illumination using mouth mirrors and gauze. Lesions suggestive of leukoplakia, erythroplakia, tobacco pouch keratosis, oral submucous fibrosis, non-healing ulcer, or suspicious induration were photographed and referred for biopsy when indicated.

Pulmonary assessment: Respiratory symptoms including chronic cough, sputum, exertional dyspnea, wheeze, hemoptysis, and recurrent lower respiratory infection were recorded. Spirometry was performed according to standard acceptability criteria, and obstructive abnormality was defined by a reduced post-bronchodilator FEV1/FVC ratio. Chest radiography was performed for symptomatic patients and all high-risk smoked or dual users aged 40 years or above. Computed tomography and bronchoscopy were advised when radiographic or clinical findings suggested malignancy.

Outcome variables: Primary outcomes were poor oral hygiene, moderate-to-severe periodontitis, oral potentially malignant disorder (OPMD), obstructive pulmonary abnormality, clinically diagnosed COPD, confirmed oral cancer, and confirmed lung cancer. Secondary outcomes included mean OHI-S score, mean FEV1% predicted, and association between duration of tobacco use and lesion prevalence.

Statistical analysis: Data were entered in Microsoft Excel and analyzed using SPSS version 26. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were expressed as frequency and percentage. Intergroup comparison was performed using one-way ANOVA for continuous variables and chi-square test for categorical variables. Binary logistic regression was used to estimate adjusted odds ratios for OPMD and obstructive pulmonary abnormality after controlling for age, sex, alcohol use, and socioeconomic status. A p-value <0.05 was considered statistically significant.

RESULTS

The study included 500 participants with a mean age of 43.8 ± 12.6 years. Males constituted 68.2% of the sample. Tobacco users had a longer mean habit duration in the dual-use group (15.8 ± 7.2 years) than in the smoked-only group (13.9 ± 6.8 years) and smokeless-only group (14.4 ± 7.0 years). Alcohol use was more common among smoked and dual users. Baseline demographic characteristics are shown in Table 1.

Oral health deterioration was significantly associated with tobacco use. Mean OHI-S score was lowest among non-users (1.4 ± 0.6) and highest among dual users (3.2 ± 0.9). Moderate-to-severe periodontitis was detected in 18.4% of non-users, 42.4% of smoked users, 46.4% of smokeless users, and 58.4% of dual users ($p < 0.001$). OPMDs were most frequently observed among smokeless and dual users, with leukoplakia, tobacco pouch keratosis, and oral submucous fibrosis being the commonest lesions. Confirmed oral cancer was present in 14 participants overall, with the highest proportion among dual users. Oral findings are summarized in Table 2.

Pulmonary morbidity showed a pattern related to inhalational exposure. Chronic respiratory symptoms were reported by 13.6% of non-users, 39.2% of smoked users, 18.4% of smokeless users, and 46.4% of dual users. Obstructive spirometric abnormality was significantly higher among smoked and dual users than non-users and smokeless-only users. Mean FEV1% predicted was 92.6 ± 10.3 in non-users and 71.5 ± 17.8 in dual users. COPD was diagnosed in 30 participants, and confirmed lung cancer was detected in 11 participants, mainly in smoked and dual users. Pulmonary and cancer-related outcomes are presented in Table 3.

On adjusted logistic regression, dual tobacco use was independently associated with OPMD (adjusted odds ratio [aOR] 9.41; 95% CI 4.10–21.59; $p < 0.001$) and obstructive pulmonary abnormality (aOR 4.72; 95% CI 2.16–10.32; $p < 0.001$) compared with non-use. Habit duration of more than 10 years was associated with higher odds of OPMD (aOR 3.26; 95% CI 1.89–5.63) and COPD (aOR 2.84; 95% CI 1.51–5.32).

Table 1. Demographic and tobacco-use characteristics of study participants

Variable	Non-users (n=125)	Smoked tobacco (n=125)	Smokeless tobacco (n=125)	Dual users (n=125)	p-value
Age (years), mean $\pm$ SD	40.6 $\pm$ 11.8	44.5 $\pm$ 12.4	43.1 $\pm$ 12.1	47.0 $\pm$ 13.0	0.002
Male sex, n (%)	72 (57.6)	94 (75.2)	80 (64.0)	95 (76.0)	0.004
Rural residence, n (%)	48 (38.4)	53 (42.4)	68 (54.4)	71 (56.8)	0.006
Alcohol use, n (%)	12 (9.6)	39 (31.2)	24 (19.2)	45 (36.0)	<0.001
Habit duration (years), mean $\pm$ SD	-	13.9 $\pm$ 6.8	14.4 $\pm$ 7.0	15.8 $\pm$ 7.2	0.081
Daily exposure >5 times/day, n (%)	-	41 (32.8)	58 (46.4)	67 (53.6)	<0.001

Table 2. Oral health and oral mucosal findings by tobacco-use pattern

Outcome	Non-users (n=125)	Smoked tobacco (n=125)	Smokeless tobacco (n=125)	Dual users (n=125)	p-value
OHI-S score, mean $\pm$ SD	1.4 $\pm$ 0.6	2.6 $\pm$ 0.8	2.8 $\pm$ 0.9	3.2 $\pm$ 0.9	<0.001
Poor oral hygiene, n (%)	16 (12.8)	43 (34.4)	49 (39.2)	61 (48.8)	<0.001
Moderate/severe periodontitis, n (%)	23 (18.4)	53 (42.4)	58 (46.4)	73 (58.4)	<0.001
Gingival recession, n (%)	18 (14.4)	49 (39.2)	52 (41.6)	66 (52.8)	<0.001
Oral potentially malignant disorder, n (%)	4 (3.2)	13 (10.4)	28 (22.4)	36 (28.8)	<0.001
Confirmed oral cancer, n (%)	1 (0.8)	2 (1.6)	4 (3.2)	7 (5.6)	0.031

Table 3. Pulmonary disorders and cancer-related outcomes by tobacco-use pattern

Outcome	Non-users (n=125)	Smoked tobacco (n=125)	Smokeless tobacco (n=125)	Dual users (n=125)	p-value
Chronic respiratory symptoms, n (%)	17 (13.6)	49 (39.2)	23 (18.4)	58 (46.4)	<0.001
FEV1% predicted, mean $\pm$ SD	92.6 $\pm$ 10.3	76.8 $\pm$ 16.2	87.4 $\pm$ 12.1	71.5 $\pm$ 17.8	<0.001
Obstructive spirometric abnormality, n (%)	7 (5.6)	35 (28.0)	12 (9.6)	43 (34.4)	<0.001
Clinically diagnosed COPD, n (%)	3 (2.4)	13 (10.4)	4 (3.2)	17 (13.6)	<0.001
Abnormal chest imaging, n (%)	6 (4.8)	25 (20.0)	8 (6.4)	31 (24.8)	<0.001
Confirmed lung cancer, n (%)	0 (0.0)	4 (3.2)	1 (0.8)	6 (4.8)	0.012

DISCUSSION

The present study demonstrates that tobacco exposure is associated with a broad spectrum of oral and pulmonary morbidity, with the highest burden among dual users. Poor oral hygiene, gingival recession, moderate-to-severe periodontitis, OPMDs, obstructive spirometric abnormality, COPD, oral cancer, and lung cancer were all more frequent among tobacco users than among non-users. These findings support the concept that tobacco is not a site-limited risk factor but a multisystem exposure capable of producing cumulative epithelial, periodontal, and pulmonary injury.

The observed periodontal differences are consistent with the biological effects of nicotine and other tobacco constituents on periodontal tissues. Tobacco reduces gingival blood flow, alters neutrophil chemotaxis and phagocytosis, increases oxidative stress, and impairs fibroblast proliferation and collagen turnover. These changes may explain why tobacco users frequently show advanced attachment loss with comparatively muted gingival bleeding. Previous reviews have emphasized that tobacco aggravates periodontal destruction and may reduce response to periodontal therapy [4,6]. In this study, moderate-to-severe periodontitis was more than three times higher among dual users than non-users, suggesting that combined smoked and smokeless exposure may intensify periodontal tissue damage.

The higher prevalence of OPMDs among smokeless and dual users was clinically important. Direct mucosal contact with smokeless products exposes the buccal mucosa, gingiva, and vestibule to carcinogens for prolonged periods. This mechanism supports the common occurrence of tobacco pouch keratosis, leukoplakia, and oral submucous fibrosis in users who keep the quid in a fixed vestibular location. The present findings agree with meta-analytic evidence that smokeless tobacco is significantly associated with oral cancer, particularly in South and South-East Asian populations where high-risk products are culturally embedded [7]. The presence of oral cancer even in a small outpatient sample indicates that opportunistic oral screening remains essential.

Pulmonary abnormalities were concentrated among smoked and dual users, reflecting the impact of inhaled combustion products on the airways and lung parenchyma. Tobacco smoke contains thousands of chemicals, including oxidants and carcinogens that induce chronic airway inflammation, mucus gland hypertrophy, ciliary dysfunction, small airway remodeling, and alveolar destruction. COPD is characterized by persistent airflow limitation and is strongly linked to smoking exposure [8,9]. In the present study, mean FEV1% predicted was lowest among dual users, and obstructive spirometric abnormality was sixfold higher among dual users than non-users. These findings indicate that respiratory screening should be considered in dental patients with substantial smoking history.

The cancer findings should be interpreted with caution because the study was not designed as a population cancer-incidence study. Nevertheless, confirmed oral cancer was more frequent among smokeless and dual users, while confirmed lung cancer was mainly detected among smoked and dual users. This pattern is consistent with carcinogenic mechanisms established by epidemiological and experimental evidence. IARC evaluations and large epidemiological reviews have classified tobacco smoke as carcinogenic to humans and causally associated with lung and upper aerodigestive tract cancers [2,3]. Longitudinal mortality studies further show major survival disadvantages among smokers and substantial benefits

after cessation [11,12]. Secondhand smoke also contributes to lung cancer risk, reinforcing the need to protect household and workplace contacts [13].

The association between tobacco duration and disease burden in this study suggests a cumulative exposure-response relationship. Participants with more than 10 years of tobacco use had higher odds of OPMD and COPD. This is clinically relevant because many tobacco users normalize symptoms such as tooth staining, halitosis, chronic cough, and oral burning until advanced disease develops. Early cessation remains the most effective intervention. Evidence from smoking cessation and COPD studies shows improvement in lung-function indicators, respiratory symptoms, exercise tolerance, and mortality after quitting [10,14]. Similarly, oral health benefits include improved gingival healing, reduced progression of periodontal disease, and lower future cancer risk when cessation occurs before malignant transformation.

The study has limitations. First, it was hospital-based and may over-represent symptomatic individuals. Second, tobacco exposure was self-reported, which may underestimate use due to social desirability bias. Third, the cross-sectional design allows association but not definitive causality. Fourth, detailed quantification of product-specific nitrosamine exposure and passive smoke exposure was not performed. Despite these limitations, the study provides practical clinical evidence that combined dental and pulmonary evaluation can detect a wider spectrum of tobacco-related disease than single-specialty screening.

The findings have direct public health implications. Dental clinics should not restrict tobacco counseling to oral hygiene advice alone; they should document tobacco pattern, examine the oral mucosa systematically, refer suspicious lesions, and encourage respiratory evaluation for smokers with chronic cough or dyspnea. Similarly, respiratory clinics should ask about smokeless tobacco and refer patients for oral screening. Integrating brief cessation counseling, nicotine dependence assessment, behavioral support, and referral to tobacco-cessation services can reduce the burden of both oral and pulmonary disease.

CONCLUSION

Tobacco use was significantly associated with poor oral hygiene, periodontal destruction, oral potentially malignant disorders, obstructive pulmonary abnormality, COPD, oral cancer, and lung cancer. Smokeless tobacco showed a stronger relationship with oral mucosal lesions and oral cancer, whereas smoked tobacco was more strongly associated with pulmonary dysfunction and lung cancer; dual users experienced the highest overall disease burden. Routine integration of oral examination, spirometry-based respiratory screening, and structured tobacco-cessation counseling can improve early detection and prevention of tobacco-related morbidity.

REFERENCES

1. GBD 2019 Tobacco Collaborators. Spatial, temporal, and demographic patterns in prevalence of smoking tobacco use and attributable disease burden in 204 countries and territories, 1990-2019: a systematic analysis from the Global Burden of Disease Study 2019. *Lancet*. 2021;397(10292):2337-2360. doi:10.1016/S0140-6736(21)01169-7. PMID:34051883.
2. Sasco AJ, Secretan MB, Straif K. Tobacco smoking and cancer: a brief review of recent epidemiological evidence. *Lung Cancer*. 2004;45 Suppl 2:S3-S9. doi:10.1016/j.lungcan.2004.07.998. PMID:15552776.
3. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Tobacco smoke and involuntary smoking. *IARC Monogr Eval Carcinog Risks Hum*. 2004;83:1-1438. PMID:15285078.
4. Reibel J. Tobacco and oral diseases. Update on the evidence, with recommendations. *Med Princ Pract*. 2003;12 Suppl 1:22-32. doi:10.1159/000069845. PMID:12707498.
5. Gajendra S, McIntosh S, Ghosh S. Effects of tobacco product use on oral health and the role of oral healthcare providers in cessation: a narrative review. *Tob Induc Dis*. 2023;21:12. doi:10.18332/tid/157203. PMID:36741542.
6. Zhang Y, He J, He B, Huang R, Li M. Effect of tobacco on periodontal disease and oral cancer. *Tob Induc Dis*. 2019;17:40. doi:10.18332/tid/106187. PMID:31516483.
7. Asthana S, Labani S, Kailash U, Sinha DN, Mehrotra R. Association of smokeless tobacco use and oral cancer: a systematic global review and meta-analysis. *Nicotine Tob Res*. 2019;21(9):1162-1171. doi:10.1093/ntr/nty074. PMID:29790998.
8. Laniado-Laborin R. Smoking and chronic obstructive pulmonary disease. *Int J Environ Res Public Health*. 2009;6(1):209-224. doi:10.3390/ijerph6010209. PMID:19440278.
9. Decramer M, Janssens W, Miravittles M. Chronic obstructive pulmonary disease. *Lancet*. 2012;379(9823):1341-1351. doi:10.1016/S0140-6736(11)60968-9. PMID:22314182.
10. Wang Z, Qiu Y, Ji X, Dong L. Effects of smoking cessation on individuals with COPD: a systematic review and meta-analysis. *Front Public Health*. 2024;12:1433269. doi:10.3389/fpubh.2024.1433269.
11. Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ*. 2004;328(7455):1519. doi:10.1136/bmj.38142.554479.AE. PMID:15213107.

12. Jha P, Ramasundarahettige C, Landsman V, Rostron B, Thun M, Anderson RN, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128. PMID:23343063.
13. Kim CH, Lee YC, Hung RJ, McNallan SR, Cote ML, Lim WY, et al. Exposure to secondhand tobacco smoke and lung cancer by histological type: a pooled analysis of the International Lung Cancer Consortium. *Int J Cancer.* 2014;135(8):1918-1930. doi:10.1002/ijc.28835. PMID:24615328.
14. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1: the Lung Health Study. *JAMA.* 1994;272(19):1497-1505. doi:10.1001/jama.1994.03520190043033. PMID:7966841.
15. Forey BA, Thornton AJ, Lee PN. Systematic review with meta-analysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. *BMC Pulm Med.* 2011;11:36. doi:10.1186/1471-2466-11-36. PMID:21672193.
16. Warnakulasuriya S. Causes of oral cancer-an appraisal of controversies. *Br Dent J.* 2009;207(10):471-475. doi:10.1038/sj.bdj.2009.1009. PMID:19946320.
17. Bagnardi V, Rota M, Botteri E, Tramacere I, Islami F, Fedirko V, et al. Light alcohol drinking and cancer: a meta-analysis. *Ann Oncol.* 2013;24(2):301-308. doi:10.1093/annonc/mds337. PMID:22910838.
18. Drope J, Schluger N, Cahn Z, Drope J, Hamill S, Islami F, et al. The Tobacco Atlas. 6th ed. Atlanta: American Cancer Society and Vital Strategies; 2018. PMID:30605123.