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Original Article

Association between dental scaling and the lower risk of oral cancer: A population-based nested case-control study

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Abstract *Background/purpose:* Oral cancer is a common malignancy that associates with oral inflammatory reactions. Dental scaling can effectively reduce periodontal inflammation to facilitate oral health. This may contribute to a less conducive environment for oral cancer initiation and progression. The aim of this study was to investigate the association between dental scaling and oral cancer.

Materials and methods: A retrospective nested case-control study was conducted by using Taiwanese Longitudinal Health Insurance Database. This study captured patients with oral cancer identified from the cancer registry. Each oral cancer patient was matched with eight non-oral cancer controls based on age and sex. Conditional logistic regression was used to assess the association between the last dental scaling prior to the index date and oral cancer occurrence.

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Results: Dental scaling significantly lowered the risk of oral cancer occurrence (aOR: 0.75, 95 % CI: 0.67–0.85). Individuals with different intervals of dental scaling prior to the index date showed a lower rate of oral cancer than those who did not received dental scaling ($P < 0.001$). In addition, the utility of dental scaling was found significantly associated with oral cancer stages ($P < 0.001$). Oral cancer patients with dental scaling prior to the index date presented a lower chance toward advanced oral cancer stage than those oral cancer patients without any dental scaling.

Conclusion: Taken together, this nationwide population-based study indicated that dental scaling exhibited a significantly lower risk of oral cancer in Taiwanese population.

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Introduction

Oral cancer is a wide-spread malignant neoplasia with lesions on the lip or oral cavity.¹ In the field of malignancy, oral cancer is the sixth most common cancer worldwide which influences the Southern Asian residents and prominent in male population.² There are several risk factors that attribute to the development of oral cancer. Cigarette smoking is one the well-known risk factors for oral cancer development which was more than 80 percent higher in those who never smoked.³ In addition, betel quid chewing is another significant risk factor of oral cancer as well as oral potentially malignant disorders in Southern Asia.⁴ Some systemic diseases including coronary heart disease and diabetes mellitus (DM) are also associated with the development of oral cancer.⁵ Within oral cavity itself, the presence of periodontitis is significantly related to the concurrence of oral cancer and the management of periodontitis cannot be overlooked.⁶

Dental scaling is a common method for the treatment of periodontal diseases.⁷ Regular dental scaling can reduce the severity and inflammatory response of periodontitis.⁸ In addition, dental scaling was found to associate with the decreased risk of Parkinson's disease,⁹ pyogenic liver abscess,¹⁰ and pneumonia.¹¹ Nevertheless, there was rare research to investigate the correlation between dental scaling and the risk of oral cancer. Evidenced based findings support the hypothesis that periodontitis is an independent risk factor for oral cancer.¹² Therefore, we proposed that dental scaling can reduce the periodontal inflammation which may also serve as a protective factor for oral cancer.

However, the association between dental scaling and the risk of oral cancer still needs further investigation. Taiwanese National Health Insurance Research Database (NHIRD) has facilitated many population-based longitudinal studies in Taiwan.^{13–15} The purpose of the current study was to evaluate the association of dental scaling with oral cancer occurrence in a large, nationally representative, population-based design in Taiwan. In addition, the relation of dental scaling to oral cancer stages was also analyzed.

Materials and methods

Ethnic approval

The study obeys the declaration of Helsinki in 1964 and its later revisions. The current study was approved by the Institutional Review Board of Chung Shan Medical University Hospital (CS2-23031). No informed consent from participants was required.

Data source

Taiwanese NHIRD has the claimed data which indicate codes for participants' characters, image exams, laboratory exams, diagnoses, procedures, surgeries, and medications. The population of NHIRD was nearly 23 million Taiwanese with a follow-up period from January 1, 2000 to December 31, 2020. As described previously,¹⁶ the available data in NHIRD include the International Classification of Diseases Ninth Revision (ICD-9) diagnostic code, ICD-10 diagnostic code, age, sex, image exam codes, laboratory exam codes, medical department codes, procedure codes, and international ATC codes for prescriptions. The current study utilized the Longitudinal Health Insurance Database (LHID) 2000, a sub-database of NHIRD, which contains approximately 2 million participants randomly selected from NHIRD in 2000. The available data and the follow-up period of LHID 2000 is identical to NHIRD. Another sub-database within NHIRD is the Taiwan National Cancer Registry. This database includes information on tumor stage, surgical procedures, chemotherapy, radiation therapy, and targeted therapy. The primary site of tumor is classified based on ICD for Oncology, third version (ICD-O-3) coding.

Subject selection

A retrospective nested case-control study was conducted. The participants were considered to have oral cancer if they were confirmed by the cancer registry (ICD-O-3 = C00–C10) from 2007 to 2017 with the age older than 18 years. The index date was defined as the initial date of oral cancer diagnosis. In addition, the participants that diagnosed with other types

of cancers were excluded from the current study based on the ICD-9 and ICD-10 codes. Sequentially, one oral cancer participant was matched to eight non-oral cancer participants by age and sex. The non-oral cancer participants constituted as the control group. Finally, a total of 4882 and 39056 participants were enrolled into the oral cancer group and control group, respectively. The flowchart of subject selection is illustrated in Fig. 1.

Dental scaling and covariates

The exposure in this study was dental scaling which includes both localized and full-mouth scaling according to the treatment codes 91003C and 91004C (Supplementary Table 1). Only dental scaling performed before the index date was considered. The study categorized patients based on the timing of their dental scaling into three intervals: those who received scaling within two years before the index date, those who received scaling between two and four years before the index date, and those who received scaling more than four years before the index date. In addition, some local and systemic diseases were also included in the analysis model to evaluate their association to oral cancer development and further adjust their effects. The comorbidities included hypertension, DM, hyperlipidemia, chronic liver disease, chronic kidney disease, chronic obstructive pulmonary disease (COPD), rheumatoid arthritis, ankylosing spondylitis, ischemic heart disease, stroke, hepatitis B, hepatitis C, and periodontitis. The definitions of these confounders were according to the ICD-9 or ICD-10 diagnostic codes (Supplementary Table 1). To ensure the accuracy of comorbidities in this study, those defined as at least three outpatients visit or one admission were accounted.

Statistical analysis

Statistical analyses for the current study were performed using SAS version 9.4 (SAS Institute Inc., Cary, NC, USA).

Descriptive analysis was used to illustrate demographic and comorbid characteristics between two groups. The independent t-test and chi-square test were employed to compare these characteristics. Subsequently, conditional logistic regression was used to calculate crude odds ratios (cOR) and adjusted odds ratios (aOR) with 95 % confidence intervals (CI) for dental scaling between oral cancer group and control group.

Additionally, this study examined the association between the timing of dental scaling and oral cancer. Conditional logistic regression was used to determine aOR and 95 % CI for oral cancer among subgroups based on the last dental scaling before the index date. We also defined the stage III or IV oral cancer as the advanced oral cancer to calculate the aOR for advanced oral cancer among dental scaling subgroups and the intervals of last dental scaling prior to the index date within oral cancer patients. Statistical significance was set at $P < 0.05$.

Results

The demographic characteristics of oral cancer and control groups are illustrated in Table 1. The study included a total of 43,938 participants, consisting of 4,882 patients diagnosed with oral cancer (case group) and 39,056 non-oral cancer patients (control group). The participants in both groups were matched by age and sex to minimize the confounding variables. The mean age of studied population was 55 years. The age, sex, and follow-up duration were identical due to the matching process ($P > 0.05$). The average follow-up time for both groups was 14.15 years. The ratio of dental scaling was significantly higher in control group (76.04 %) than oral cancer group (64.60 %) ($P < 0.001$). In addition, the ratio of hypertension, hyperlipidemia, chronic liver disease, DM, COPD, stroke, hepatitis B, hepatitis C, and periodontitis were also significantly higher in oral cancer group ($P < 0.05$). Moreover, the interval of last dental scaling prior to the index date and the

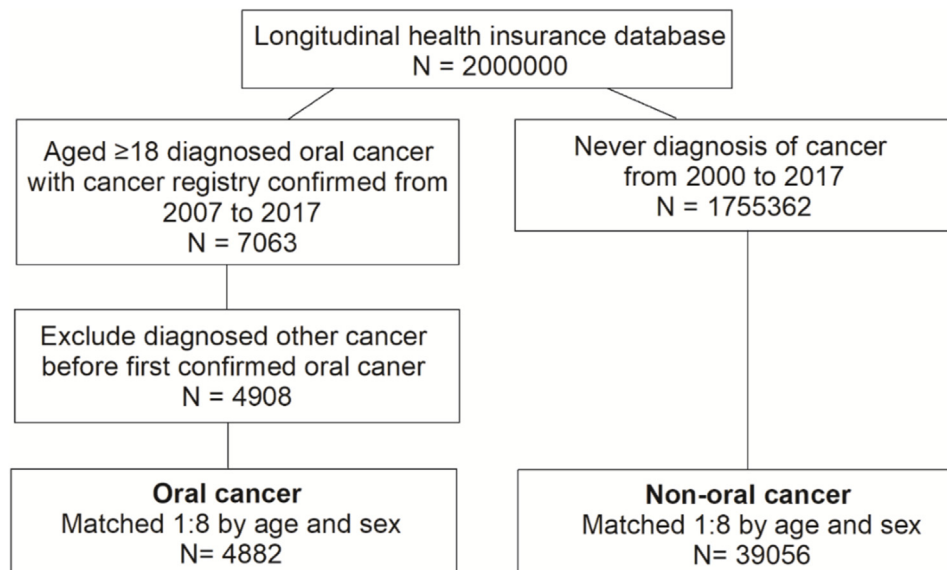


Figure 1 The flowchart of this nationwide population-based nested case-control study.

Table 1 Demographic characteristics of oral cancer and non-oral cancer groups.

| | Non-oral cancer N = 39056 | Oral cancer N = 4882 | P value |
|--|---------------------------|----------------------|---------|
| Follow-up period (year) | 14.15 ± 3.67 | 14.15 ± 3.67 | n.s |
| Age | | | n.s |
| 18-39 | 3472 (8.89 %) | 434 (8.89 %) | |
| 40-54 | 16304 (41.75 %) | 2038 (41.75 %) | |
| 55-64 | 11784 (30.17 %) | 1473 (30.17 %) | |
| ≥65 | 7496 (19.19 %) | 937 (19.19 %) | |
| Mean ± SD | 55.24 ± 11.69 | 55.24 ± 11.69 | n.s |
| Sex | | | n.s |
| Female | 4160 (10.65 %) | 520 (10.65 %) | |
| Male | 34896 (89.35 %) | 4362 (89.35 %) | |
| Dental scaling | | | <0.001* |
| No | 9359 (23.96 %) | 1728 (35.40 %) | |
| Yes | 29697 (76.04 %) | 3154 (64.60 %) | |
| Hypertension | 12789 (32.75 %) | 1790 (36.67 %) | <0.001* |
| Hyperlipidemia | 10243 (26.23 %) | 1376 (28.19 %) | 0.003* |
| Chronic liver disease | 6997 (17.92 %) | 1131 (23.17 %) | <0.001* |
| Chronic kidney disease | 936 (2.40 %) | 134 (2.74 %) | 0.137 |
| Diabetes mellitus | 6316 (16.17 %) | 1068 (21.88 %) | <0.001* |
| COPD | 3506 (8.98 %) | 524 (10.73 %) | <0.001* |
| Rheumatoid arthritis | 365 (0.93 %) | 50 (1.02 %) | 0.542 |
| Ankylosing spondylitis | 254 (0.65 %) | 23 (0.47 %) | 0.136 |
| Ischemic heart disease | 5265 (13.48 %) | 650 (13.31 %) | 0.748 |
| Stroke | 2895 (7.41 %) | 426 (8.73 %) | 0.001* |
| Hepatitis B | 1874 (4.80 %) | 187 (3.83 %) | 0.003* |
| Hepatitis C | 584 (1.50 %) | 113 (2.31 %) | <0.001* |
| Periodontitis | 18521 (47.42 %) | 1869 (38.28 %) | <0.001* |
| The last dental scaling prior to the index date (year) | | | <0.001* |
| None | 9359 (23.96 %) | 1728 (35.40 %) | |
| ≤2 | 15851 (40.59 %) | 1526 (31.26 %) | |
| 2–4 | 5224 (13.38 %) | 508 (10.41 %) | |
| >4 | 8622 (22.08 %) | 1120 (22.94 %) | |
| Oral cancer stage | | | |
| 0 | | 28 (0.57 %) | |
| I | | 1179 (24.15 %) | |
| II | | 941 (19.27 %) | |
| III | | 621 (12.72 %) | |
| IV | | 2113 (43.28 %) | |

The mean and standard deviation present continuous variables, while the sample count and percentage represent categorical variables. COPD: Chronic obstructive pulmonary disease, N: number, SD: standard deviation. n.s: non-significance, * Statistical significance: $P < 0.05$.

oral cancer stage are presented in [Table 1](#). The TNM classification and treatment status of oral cancer are shown in [Supplementary Tables 2 and 3](#). T2 represented the largest proportion of T-stage cases (1,507 cases, 30.87 %), followed by T1 (1,344 cases, 27.53 %). For N-stage, N0 was the most frequent (2,575 cases, 52.74 %), followed by N2B as the second most common (748 cases, 15.32 %). The majority of M-stage cases were classified as M0 (4,572 cases, 93.65 %). A total of 3,430 patients (70.26 %) underwent surgery, while radiotherapy was administered to 2,115 patients (43.32 %) with the highest number in Stage IV (1,396 cases). Systemic therapy was given to 2,018 patients (41.34 %) with predominantly in Stage IV (1,407 cases). Chemotherapy was received by 1,929 patients (39.51 %) with the most cases in Stage IV (1,345 cases).

As shown in [Table 2](#), dental scaling was found to significantly lower the risk of oral cancer development (aOR: 0.75, 95 % CI: 0.67–0.85, $P < 0.001$) after the adjustment of multiple covariates. In addition, some co-morbidities such as chronic liver disease, DM, and hepatitis B were found to correlate to the existence of oral cancer ($P < 0.05$).

Concerning the risk of oral cancer in patients with the last dental scaling prior to the index date was shown in [Table 3](#). Patients with different dental scaling intervals prior to the index date showed a lower oral cancer rate as compared to those who did not received dental scaling after the adjustment of all co-morbidities ($P < 0.05$).

As shown in [Table 4](#), the ratio of non-dental scaling in oral cancer group was from 10.71 % in stage 0 and up to 44.25 % in stage IV. On the contrary, the ratio of dental

Table 2 Conditional logistic regression of the risk of oral cancer.

| | cOR (95 % CI) | P value | aOR ^a (95 % CI) | P value |
|------------------------|------------------|---------|----------------------------|---------|
| Dental scaling | 0.56 (0.51–0.61) | <0.001 | 0.75 (0.67–0.85) | <0.001* |
| Hypertension | 1.18 (1.07–1.29) | 0.0005 | 1.10 (0.98–1.22) | 0.110 |
| Hyperlipidemia | 1.12 (1.02–1.23) | 0.0148 | 1.00 (0.89–1.12) | 0.999 |
| Chronic liver disease | 1.33 (1.20–1.46) | <0.001 | 1.33 (1.19–1.49) | <0.001* |
| Chronic kidney disease | 1.08 (0.85–1.39) | 0.5272 | 0.97 (0.74–1.27) | 0.828 |
| Diabetes mellitus | 1.50 (1.35–1.67) | <0.001 | 1.48 (1.31–1.68) | <0.001* |
| COPD | 1.12 (0.98–1.29) | 0.0943 | 1.11 (0.96–1.29) | 0.149 |
| Rheumatoid arthritis | 1.04 (0.70–1.56) | 0.8383 | 1.02 (0.67–1.55) | 0.934 |
| Ankylosing spondylitis | 0.70 (0.41–1.19) | 0.1838 | 0.82 (0.47–1.43) | 0.482 |
| Ischemic heart disease | 0.96 (0.85–1.09) | 0.5312 | 0.91 (0.79–1.05) | 0.184 |
| Stroke | 1.11 (0.96–1.29) | 0.1707 | 0.99 (0.84–1.16) | 0.888 |
| Hepatitis B | 0.79 (0.65–0.96) | 0.0154 | 0.72 (0.58–0.89) | 0.003* |
| Hepatitis C | 1.47 (1.10–1.97) | 0.0091 | 1.22 (0.89–1.67) | 0.217 |
| Periodontitis | 0.70 (0.64–0.76) | <0.001 | 1.09 (0.98–1.21) | 0.124 |

cOR: crude odds ratio.

aOR: adjusted odds ratio.

CI: confidence interval.

COPD: Chronic obstructive pulmonary disease.

* Statistical significance: $P < 0.05$.^a Adjusted for dental scaling and all co-morbidities.**Table 3** The risk of oral cancer at the last dental scaling prior to the index date.

| | Oral cancer/N | aOR (95 % CI) |
|--------------------|---------------|------------------|
| Time (year) | | |
| None | 1728/11087 | Reference |
| ≤2 | 1526/17377 | 0.70 (0.61–0.80) |
| 2–4 | 508/5732 | 0.66 (0.56–0.78) |
| >4 | 1120/9742 | 0.83 (0.73–0.94) |

N = number of oral cancer and non-oral cancer groups.

aOR: adjusted for all co-morbidities.

CI: confidence interval.

Time: the last dental scaling prior to the index date.

Table 4 The distribution of patients within different oral cancer stages with and without dental scaling.

| | Dental scaling | | P value |
|--------------------------|----------------|----------------|---------|
| | None | Yes | |
| Oral cancer stage | | | <0.001 |
| 0 | 3 (10.71 %) | 25 (89.29 %) | |
| I | 260 (22.05 %) | 919 (77.95 %) | |
| II | 299 (31.77 %) | 642 (68.23 %) | |
| III | 231 (37.20 %) | 390 (62.80 %) | |
| IV | 935 (44.25 %) | 1178 (55.75 %) | |

scaling in oral cancer group was from 89.29 % in stage 0 and down to 55.75 % in stage IV. Taken together, dental scaling was highly associated with oral cancer stages ($P < 0.001$).

To further investigate the association of dental scaling and different stage oral cancers, the grade of oral cancer was divided into non-advanced oral cancer (stage I/II) and advanced oral cancer (stage III/IV). As shown in Table 5,

Table 5 The association of dental scaling and the risk of advanced oral cancer.

| | Advanced oral cancer/N | aOR (95 % CI) |
|-----------------------|------------------------|------------------|
| Dental scaling | | |
| No | 1166/1728 | Reference |
| Yes | 1568/3154 | 0.69 (0.60–0.81) |
| Time (year) | | |
| None | 1166/1728 | Reference |
| ≤2 | 650/1526 | 0.52 (0.43–0.62) |
| 2–4 | 267/508 | 0.73 (0.58–0.91) |
| >4 | 651/1120 | 0.82 (0.69–0.97) |

Advanced oral cancer: stage III/IV oral cancer.

N = total number of oral cancer.

aOR: adjusted for all co-morbidities.

Time: the last dental scaling prior to the index date.

oral cancer patients with dental scaling significantly reduced the advanced oral cancer development (aOR: 0.69, 95 % CI: 0.60–0.81, $P < 0.001$). Moreover, the individuals received any previous dental scaling demonstrated the lower risk of developing advanced oral cancer in oral cancer population ($P < 0.001$).

Discussion

Dental scaling can reduce the periodontal immune–inflammatory reaction to enhance oral health. Poor oral hygiene is a common risk factor of oral cancer and allows excessive microflora growth resulted in species balance. To the best of our knowledge, this is the first findings to demonstrate the relationship between dental scaling and oral cancer. The results showed that dental scaling was found to significantly lower the risk of oral cancer

occurrence. In agreement with our findings, Cobb & Sot-santi¹⁷ who reported that dental scaling could reduce the ratio of oral cancer via the enhancement of oral hygiene. Consistently, Chang et al.¹⁸ reported that poor oral hygiene could worsen the survival outcomes of head and neck cancer patients. Deng et al.¹⁹ demonstrated that the inadequate oral hygiene significantly elevated the risk of oral cancer and its subtypes by a composite oral hygiene score. Recently, a meta-analysis study also indicated that poor oral hygiene could increase the risk of oral cancer.²⁰ The above evidences may also partly explain that oral cancer patients demonstrated a lower rate of dental scaling compared to non-oral cancer population in this study. Taken together, it is speculated that the eradication of oral inflammation through dental scaling can retard the risk of oral cancer.

Oral microorganisms play an important role in the etiology and pathogenesis of oral cancer.^{6,10} Periodontal pathogens as well as inflammatory mediators can invade into saliva and blood that might lead to oral carcinogenesis. A narrative review showed that periodontitis is related to the occurrence of oral cancer and the recurrent periodontitis can elevate the incidence of oral cancer.²¹ In addition, a registry-based cohort study indicated that patients with periodontitis exhibited a higher risk of developing oral cancer.²² However, our findings demonstrated that periodontitis patients exhibited a borderline significant of oral cancer concurrence (aOR = 1.09, 95 % CI = 0.98–1.21). The reason is not quite clear. It might be due to the different diagnostic code, study design, and databank used. Therefore, the relationship between periodontitis and dental scaling in patients with oral cancer is worth to further evaluation.

Our findings also revealed that patients with different intervals of dental scaling prior to the index date showed a lower rate of oral cancer than those who did not receive dental scaling. Oral cancer patients with previous dental scaling revealed a lower percentage of stage III/IV oral cancer progression. Moreover, the periodically dental scaling with an interval not longer than four years demonstrated the protect effect of oral cancer. Similar results were shown that oral cancer individuals with severe systemic inflammation was associated with worse overall survival.²³ In addition, most oral cancer patients are usually tentative diagnosis in an advanced stage (stages III/IV) on their first medical visit.²⁴ Taken together, in addition to early diagnosis and screening of oral cancer, dental scaling might play a pivotal role to prevent or reduce the possible oral carcinogenesis.

The strength of this study is the use of nationwide population-based database which can provide both sufficient sample size and statistical power. These factor can increase the generalizability to assess the association of dental scaling and oral cancer. However, some limitations in the current study still need to be addressed. First, claimed data were used instead of actual medical documents that may limit the knowledge to the codes for each disease and management. For example, many crucial information including smoking, alcohol consumption, betel quid chewing, and the socioeconomic status cannot be obtained from NHIRD. Second, personal details for dental scaling such as the severities of periodontal diseases, oral

hygiene from caregivers, and improved education regarding good oral hygiene practices were not also included in the NHIRD. Finally, the retrospective design of this study will reduce the homogeneity of study population compared to a prospective one despite the age- and sex-matching process. Cohort study design may be necessary to assess the causal relationship between dental scaling and oral cancer.

In conclusion, dental scaling is associated with lower risk of oral cancer after adjusting multiple covariates. Furthermore, oral cancer patients with dental scaling showed a lower chance toward advanced oral cancer progression. Currently, Taiwan national health insurance offers dental prophylaxis twice a year to the public. This policy is not only good for periodontal health but also a potential modifiable factor in the primary prevention of oral cancer. However, further prospective studies to investigate the causal relationship between dental scaling and oral cancer are mandatory.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jds.2025.03.037>.

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