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Association of Cigarette Consumption and Mucosal Thickening in the Paranasal Sinuses on MRI

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ABSTRACT

Objective: To investigate the impact of cigarette consumption on mucosal thickening in paranasal sinuses and the relationships of smoking-related factors and dental status with mucosal thickening at different maxillary sinus locations using MRI.

Materials and methods: This retrospective study investigated 1094 paranasal sinuses on MRIs by correlating mucosal thickening with smoking-related factors. Presence/absence of maxillary posterior teeth was correlated with mucosal thickening on the maxillary sinus floor and other sinus locations.

Results: Compared with nonsmokers, current and former smokers exhibited similarly more mucosal thickening in the maxillary (58.9%/62.8% vs 38.3%, $P < .01$) and ethmoid (22.7%/17.1% vs 6.6%, $P < .01$) sinuses, but not in the sphenoid sinus ($P = .08$) and similarly more mucosal thickening on the medial/superior walls of the maxillary sinus (40.4%/41.5% vs 18.4%, $P < .01$). Patients with mucosal thickening in the maxillary and ethmoid sinuses had greater daily cigarette consumption and years of smoking than those without mucosal thickening in these sinuses ($P < .01$). Edentulous individuals had more mucosal thickening on the maxillary sinus floor compared to dentate individuals (43.7% vs 32.2%, $P < .01$).

Conclusion: Cigarette consumption is associated with increased mucosal thickening in paranasal sinuses, with a dose-response association. This thickening may be persistent after quitting smoking. Cigarette smoking may have a more pronounced impact on the sinus mucosa in areas near the primary maxillary ostium, while dental status may be more likely to affect that on the maxillary sinus floor.

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Introduction

The paranasal sinuses are lined with mucous membranes and connect to the nasal cavity through small openings termed ostia, which facilitate the drainage of mucus and the exchange of air across these structures.[1] Benign mucosal thickening in

the paranasal sinuses is commonly observed in adults, with an incidence ranging from 32% to 49%.[2-5] These mucosal thickening can be asymptomatic and are often detected incidentally on cross-sectional imaging conducted for various indications, including ear-nose-throat (ENT) and neurological symptoms. The majority of these thickening do not require clinical intervention and can disappear over time. However, for patients who need head and neck surgeries that commonly involve the paranasal sinuses, such as orbital decompression and skull base surgery, mucosal thickening in the paranasal sinuses may potentially increase the risk of postoperative complications. For dental practitioners, a comprehensive understanding of the health and potential pathologies of the oral-maxillofacial region, including the paranasal sinuses, is essential. The maxillary sinus, in particular, is often involved in surgical procedures in the posterior maxilla, such as dental implant placement, sinus augmentation, apical surgery of maxillary posterior teeth, and orthognathic surgery.[6-8] The maxillary and ethmoid sinuses together form the osteomeatal complex, that is the common pathway for drainage and ventilation of the maxillary sinus. Therefore, knowledge of potential factors that associate with mucosal thickening in the paranasal sinuses, particularly the maxillary sinus, may assist clinical practitioners in assessing the risk of postoperative complications prior to surgical procedures involving the maxillary sinus.

Many studies have investigated the possible biomechanisms that cause mucosal thickening in the paranasal sinuses. Cigarette consumption is one of the most common factors that adversely affect sinonasal epithelial mucociliary clearance, innate immune function, and olfactory mucosal metaplasia.[9] However, the existing evidence remains controversial regarding the association between cigarette consumption and mucosal morphological thickening in the paranasal sinuses. Some studies have reported a positive association between cigarette consumption and mucosal thickening in the maxillary sinus,[10-12] while others have found a negative association[13,14] or no association.[15-17] Yet, none of these studies specifically investigated the locations of the mucosal thickening within the maxillary sinus. Cigarette smoke may have a greater impact on the mucosa of the superior and medial walls of the maxillary sinus, which form the primary ostium that allows inhaled smoke to access the sinus via this area. In contrast, the mucosa on the sinus floor may be more likely affected by dental conditions of the teeth in the posterior maxilla due to their close proximity. Moreover, details of cigarette consumption, such as history, intensity, and duration of smoking, which may contribute differently to the impact of sinus mucosal thickening, were not investigated, particularly regarding the mucosal thickening in the ethmoid or sphenoid sinus.

Therefore, the objectives of this study were to (1) investigate the association between cigarette consumption, including smoking status (current, former, and nonsmokers), intensity (ie, daily cigarette consumption), duration (ie, years of smoking), and pack-years of smoking, and mucosal thickening in the paranasal sinuses using MRI and (2) evaluate the associations of the smoking-related factors and dental status with mucosal thickening in different locations of the maxillary sinus.

Materials and methods

Study design

This retrospective study was designed following the STROBE Statement and Checklist[18] and approved by local institutional review board. The study was conducted in full accordance with the Declaration of Helsinki 2013 and its later amendments.

Study subject

This study investigated head and neck MRI scans from 547 adult patients who met the following inclusion criteria:

1. MRI performed between 2013 and 2021 for suspected head and neck cancer due to elevated plasma Epstein-Barr virus (EBV) DNA levels and the coverage of MRI included at least maxillary, ethmoid, and sphenoid sinuses;
2. Patients diagnosed without any head and neck cancer and with a minimum follow-up of 2 years; and
3. Cigarette consumption information was retrievable.

Patients were excluded if they met any of the following exclusion criteria:

1. Patients with history of surgical procedures in the regions of paranasal sinuses or
2. Presence of motion or metal artefacts in the regions of the maxillary, ethmoid, and sphenoid sinuses on MRI scans.

Image acquisition

The head and neck MRI scans were performed using a 3 T whole-body MRI system (Philips Healthcare) and included at least the following sequences: (1) axial spin-echo–based T2-weighted images (repetition time/echo time of 2500-4000/80-100 ms; field of view of 22 cm; section thickness of 4 mm without a slice gap; number of slices of 30; echo train length of 15-17; sensitivity encoding factor of 1; number of signals acquired of 2), (2) axial spin-echo–based T1-weighted images (repetition time/echo time of 500/10-20 ms; field of view of 22 cm; section thickness of 4 mm without a slice gap; number of slices of 30; echo train length of 4; sensitivity encoding factor of 1; number of signals acquired of 2), and (3) coronal T1- or T2-weighted images.

Image assessment

The bilateral paranasal sinuses were assessed separately.

Mucosal morphological thickening in the maxillary sinus

The status of the mucosa within the sinus was evaluated according to the classification adapted from Soikkonen and Ainamo[19] and previous studies[20,21] (Figure 1):

1. Healthy/shallow mucosal thickening of less than 2 mm
2. Flat/irregular mucosal thickening of more than 2 mm
3. Semispherical/cystic thickening
4. Complete/partial opacification exceeding 50% of the sinus
5. Mixed flat and semispherical thickening

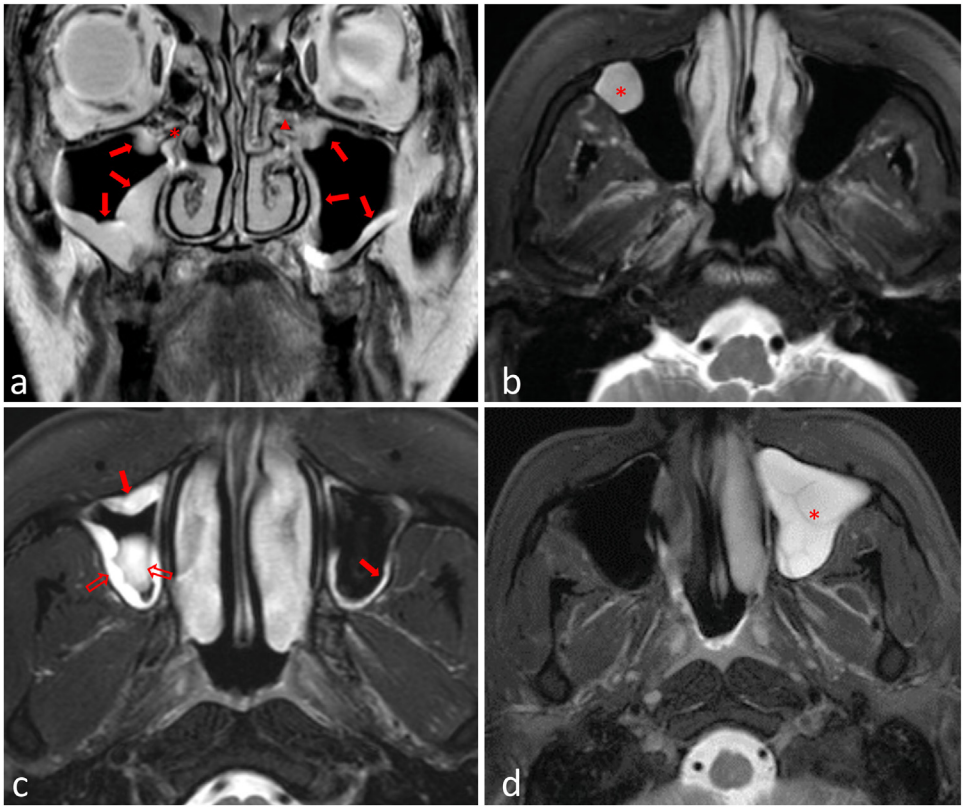

Fig. 1 – Representative T2-weighted MR images on the coronal (a) and axial (b-d) planes demonstrating various mucosal morphological thickening in the maxillary sinus: flat or irregular thickening >2 mm on the lateral, inferior, medial, superior, and/or posterior walls (solid arrows in a and c), semispherical thickening on the lateral and posterior walls (asterisk in b), mixed flat and semispherical thickening (hollow arrow in c), and complete opacification (asterisk in d); and patent (asterisk in a) or obstructed (triangle in a) primary maxillary ostium.

The sinuses with the mucosal status (1) were categorised as sinuses without mucosal thickening, while those with the status (2)-(5) were categorised as sinuses with mucosal thickening.

For the identified mucosal thickening, their locations (inferior, superior, lateral, medial, and posterior walls) in the maxillary sinus were assessed for subsequent evaluations of the association of smoking-related factors and dental status with mucosal thickening at specific locations.

Mucosal morphological thickening in the ethmoid and sphenoid sinuses

The ethmoid[7] and sphenoid[8] sinuses with the mucosal status (2)-(5) were categorised as sinuses with mucosal thickening, while those with the status (1) were categorised as sinuses without mucosal thickening (Figure 2).

The primary and accessory maxillary ostia

The status of the primary maxillary ostium (PMO) and the presence of an accessory maxillary ostium (AMO) were evaluated.[21] The PMO status of each sinus was classified as either radiologically patent or obstructed. The presence of an AMO was classified as either radiologically absent or present.

The status of maxillary posterior teeth

The teeth in the bilateral posterior maxilla were assessed separately. The status of the maxillary posterior dentition, distal to the maxillary canine and excluding the third molar, was classified as either completely/partially edentulous or dentate. Based on whether the teeth in the maxillary posterior dentition penetrated the sinus floor, they were further classified into penetration and nonpenetration groups (Figure 3).

The assessments on the MRI were performed with reference to both axial and coronal T1- and T2-weighted images by an experienced on-board dentist who had over 10 years of experience in general dentistry and 6 years of experience in oral-maxillofacial imaging with the consultation from 2 certified radiologists with over 3 years of experience in head and neck and oral-maxillofacial radiology. To test intraobserver reproducibility, all the assessments were repeated on 20% of the included MRI scans.

Patient demographics and cigarette consumption

Patient demographics, history of cigarette consumption (current, former, or nonsmoker), daily cigarette consumption, and years of smoking at the time of imaging were retrieved from the patients' medical records. The pack-years of smoking, a highly recommended cumulative exposure indicator for estimating patients' risk of developing smoking-related conditions in clinical practice, research, and healthcare

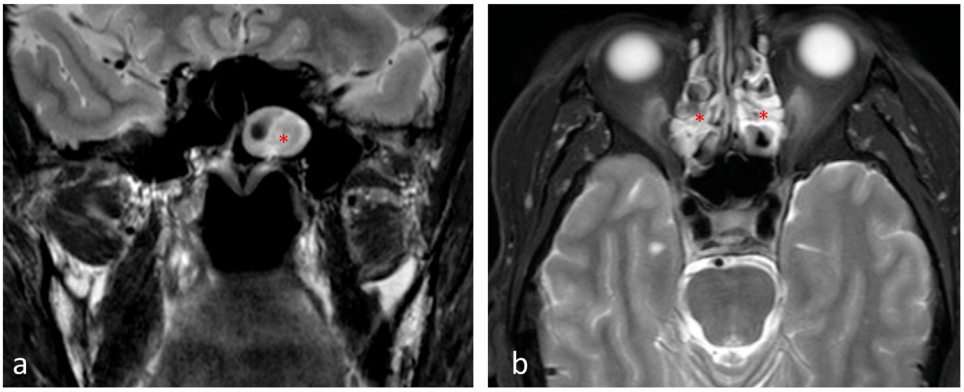

Fig. 2 – Representative T2-weighted MR images on the coronal (a) and axial (b) planes demonstrating mucosal morphological thickening (asterisks) in the sphenoid sinus (a) and ethmoid sinus (b).

policies, was calculated for each patient using the following formula[22]:

$$\frac{\textit{Number of daily cigarettes consumption} \times \textit{Number of years of smoking}}{20}$$

Statistical analysis

Differences among current, former, and nonsmokers in the presence and/or location of mucosal thickening in the maxillary, ethmoid, and sphenoid sinuses, the ostia of the maxillary sinus, and the status of the maxillary posterior teeth were assessed using Pearson chi-square tests. Pairwise comparisons were performed with Bonferroni adjustment. The analyses of potential dose-response association of the intensity, duration, and pack-years of smoking with mucosal thickening in the paranasal sinuses were performed using independent samples *t*-tests. Intraobserver reproducibility was evaluated using Cohen kappa values. The significance level chosen for all of the statistical tests mentioned above was set at 0.05. All analyses were performed using SPSS (Version 29.0, IBM Corp.).

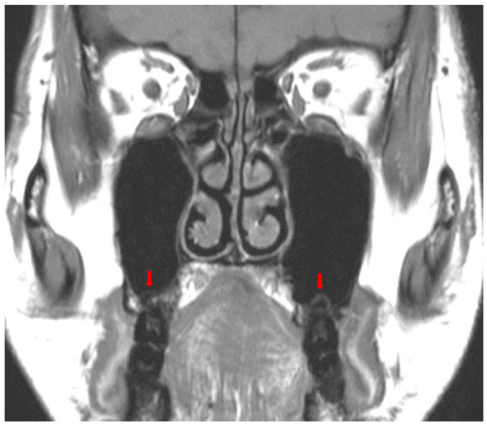

Fig. 3 – A representative T1-weighted MR image on the coronal plane demonstrating maxillary molar roots penetrating the sinus floor (arrows).

Results

A total of 1094 maxillary, ethmoid, and sphenoid sinuses from 547 eligible patients were included in the assessments. Among these patients, 324/547 (59.2%) were nonsmokers, 141/547 (25.8%) were current smokers, and 82/547 (15%) were former smokers. The mean age was 56.1 years for nonsmokers, 54.3 years for current smokers, and 55.7 years for former smokers. The patient's average age, daily cigarette consumption, years of smoking, pack-years of smoking, mucosal thickening in the maxillary, ethmoid, and sphenoid sinuses, as well as the status of maxillary sinus ostia and maxillary posterior teeth are shown in Table 1. Mucosal thickening was observed in 517/1094 (47.3%) maxillary sinuses, 135/1094 (12.3%) ethmoid sinuses, and 27/1094 (2.5%) sphenoid sinuses.

In maxillary sinuses, the most common type of mucosal change was flat/irregular thickening (332/1094, 30.3%), followed by semispherical/cystic thickening (92/1094, 8.4%), mixed flat and semispherical thickening (54/1094, 4.9%), and complete/partial opacification (39/1094, 3.6%). The PMO obstruction and AMO presence were observed in 45/1094 (4.1%) and 25/1094 (2.3%), respectively. Complete/partial edentulism in the posterior maxilla and maxillary posterior tooth roots penetrating the sinus floor was observed in 300/1094 (27.4%) and 87/1094 (8%), respectively. The intraobserver reproducibility for identifying mucosal thickening in the paranasal sinuses, ostia of the maxillary sinus, and maxillary posterior dentition on MRI was substantial to excellent, with kappa values ranging from 0.72 to 0.93.

Cigarette consumption and mucosal thickening in the paranasal sinuses

Compared with nonsmokers, current and former smokers showed significantly higher rates of mucosal thickening in the maxillary and ethmoid sinuses ($P < .001$) but not in the sphenoid sinus ($P = .08$) (Table 1). Flat/irregular thickening and mixed flat and semispherical thickening in the maxillary sinus were more commonly observed in current and former smokers than in nonsmokers ($P < .001$) (Table 1). No differences in rates of cystic thickening and complete/partial opacification among the smoking groups ($P = .672$ and .354, respectively) (Table 1). More maxillary sinuses with an obstructed PMO were found in current and

Table 1 – Differences among current, former, and nonsmokers regarding average age, daily cigarette consumption, years of smoking, mucosal morphological thickening in the sinuses, maxillary sinus ostia, and the status of maxillary posterior dentition.

	Nonsmoker (a)	Current smoker (b)	Former smoker (c)	All	*P* value
Demographics and smoking habits					
Number (%) of subjects	324 (59.2%)	141 (25.8%)	82 (15%)	547 (100%)	
Average age (range) (years)	56.1 (39-68)	54.3 (40-67)	55.7 (41-68)	55.6 (39-68)	
Average years of smoking	NA	27.5	16.5	NA	
Average daily cigarette consumption	NA	14.3	13.9	NA	
Average pack-years of smoking	NA	20.6	13	NA	
Image assessment					
Mucosal morphological thickening in the maxillary sinus					
(i) Healthy/shallow thickening of <2 mm	400 (61.7%)	116 (41.1%)	61 (37.2%)	577 (52.7%)	**<.001** ($a > b = c$)
(ii) Flat/irregular thickening of >2 mm	151 (23.3%)	114 (40.1%)	68 (41.5%)	332 (30.3%)	**<.001** ($a < b = c$)
(iii) Semispherical/cystic thickening	53 (8.2%)	27 (9.6%)	12 (7.3%)	92 (8.4%)	.672
(iv) Complete/partial opacification exceeding 50% of the sinus	21 (3.2%)	9 (3.2%)	9 (5.5%)	39 (3.6%)	.354
(v) Mixed flat and semispherical thickening	23 (3.5%)	17 (6%)	14 (8.5%)	54 (4.9%)	**.019** ($a < c$)
Ethmoid sinus					
Mucosal morphological thickening (ii)-(v)	43 (6.6%)	64 (22.7%)	28 (17.1%)	135 (12.3%)	**<.001** ($a < b = c$)
Sphenoid sinus					
Mucosal morphological thickening (ii)-(v)	12 (1.9%)	12 (4.3%)	3 (1.8%)	27 (2.5%)	.08
Ostia of the maxillary sinus					
PMO obstruction	15 (2.3%)	19 (6.7%)	11 (6.7%)	45 (4.1%)	**<.001** ($a < b = c$)
AMO presence	15 (2.3%)	7 (2.5%)	3 (1.8%)	25 (2.3%)	.903
Maxillary posterior dentition					
Complete/partial edentulism in the posterior maxilla	175 (27%)	80 (28.4%)	45 (27.4%)	300 (27.4%)	.912
Maxillary posterior tooth roots penetrating the sinus floor	49 (7.6%)	26 (9.2%)	12 (7.3%)	87 (8%)	.656

NA, not applicable; PMO/AMO, primary/accessory maxillary ostium.
Pearson chi-square test was performed; $P < .05$ in bold; pairwise comparisons with Bonferroni adjustment.

former smokers than in nonsmokers ($P < .001$) (Table 1). No significant differences were observed in the presence of an AMO and the status of maxillary posterior teeth among the smoking groups ($P = .656$ to $.912$) (Table 1). Compared with nonsmokers, significantly higher rates of mucosal thickening were observed on the medial and superior walls of the maxillary sinus in the current and former smokers ($P < .001$) (Table 2). No significant differences in the rates of mucosal thickening were found in other locations of the maxillary sinus among the three groups ($P = .749$) (Table 2).

Dose-response association between cigarette consumption and mucosal thickening in the paranasal sinuses

Compared with patients without mucosal thickening in the maxillary and ethmoid sinuses, those with mucosal thickening in these sinuses had significantly higher daily cigarette consumption and longer years of smoking ($P < .01$). Additionally, a greater pack-years of smoking was observed specifically in patients with mucosal thickening in the ethmoid sinus ($P = .004$) (Table 3). No significant

Table 2 – Comparison of mucosal morphological thickening in different locations of the maxillary sinus among current, former, and nonsmokers.

Mucosal morphological thickening in the maxillary sinus (ii)-(v)	Nonsmoker (a)	Current smoker (b)	Former smoker (c)	All	*P* value
All locations	248 (38.3%)	166 (58.9%)	103 (62.8%)	517 (47.3%)	**<.001** ($a < b = c$)
The superior and medial walls of the sinus	119 (18.4%)	114 (40.4%)	68 (41.5%)	301 (27.5%)	**<.001** ($a < b = c$)
The locations other than the superior and medial walls of the sinus	129 (19.9%)	52 (18.4%)	35 (21.3%)	216 (19.7%)	.749

Pearson chi-square test was performed; $P < .05$ in bold; pairwise comparisons with Bonferroni adjustment.

Table 3 – Analyses of dose-response association between cigarette consumption and mucosal morphological thickening in the maxillary, ethmoid, and sphenoid sinuses.

			Pack-years of smoking (mean ± SD)	*P* value	Number of daily cigarette consumption (mean ± SD)	*P* value	Year of smoking (mean ± SD)	*P* value
Maxillary sinus								
Mucosal morphological thickening (ii)-(v)	All locations	Present	18.1 ± 14	.711	7.4 ± 9.4	**<.001**	12.1 ± 14.1	**<.001**
		Absent	17.4 ± 13.8		4.3 ± 7.9		7.2 ± 12.4	
	The superior and medial walls of the sinus	Present	18.4 ± 14.1	.799	8.7 ± 9.8	**<.001**	14.4 ± 14.4	**<.001**
		Absent	17.4 ± 13.8		4.6 ± 8.1		7.7 ± 12.5	
	The locations other than the superior and medial walls of the sinus	Present	17.4 ± 13.9	.893	5.6 ± 8.4	.53	9.1 ± 13	.226
		Absent	17.9 ± 13.9		5.8 ± 8.8		9.7 ± 13.5	
Ethmoid sinus								
Mucosal morphological thickening (ii)-(v)		Present	19.7 ± 16.5	**0.004**	9.9 ± 10.9	**<0.001**	16.7 ± 14.7	**0.003**
		Absent	17.3 ± 13.1		5.2 ± 8.2		8.6 ± 12.9	
Sphenoid sinus								
Mucosal morphological thickening (ii)-(v)		Present	18.4 ± 14.8	0.675	6.5 ± 7.8	0.631	16 ± 16.5	**0.008**
		Absent	17.8 ± 13.9		5.7 ± 8.8		9.4 ± 13.3	

Independent samples *t* test was performed; $P < .05$ in bold; SD, standard deviation.

Pack-years of smoking is a measurement unit that represents a person's cumulative smoking exposure over time, calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.

Table 4 – Associations of mucosal morphological thickening in different locations of the maxillary sinus with the status of the maxillary posterior teeth.

	Maxillary posterior dentition			Roots of the maxillary posterior teeth		
Mucosal morphological thickening (ii)-(v)	Completely or partially edentulous	Dentate	*P* value	Penetrating the sinus floor	Not penetrating the sinus floor	*P* value
The sinus floor	131 (43.7%)	256 (32.2%)	**<.001**	31 (35.6%)	356 (35.4%)	.958
The locations other than the sinus floor	34 (11.3%)	96 (12.1%)	.73	12 (13.8%)	118 (11.7%)	.566

Chi-square test was performed; $P < .05$ in bold.

differences in the pack-years of smoking and daily cigarette consumption were found between patients with and without mucosal thickening in the sphenoid sinus, but patients with mucosal thickening in the sphenoid sinus showed longer years of smoking compared with those without mucosal thickening in the sphenoid sinuses ($P = .008$) (Table 3).

Maxillary posterior teeth and mucosal thickening in the maxillary sinuses

Compared to patients with dentate maxillary posterior dentition, those with completely or partially edentulous posterior dentition exhibited a significantly higher rate of mucosal thickening on the maxillary sinus floor (43.7% vs 32.2%, $P < .001$). However, no significant differences were found in the rate of mucosal thickening in locations other than the maxillary sinus floor between dentate and edentulous patients ($P = .74$) (Table 4). Regarding the penetration of tooth roots into the maxillary sinus floor, no significant differences were found in the mucosal thickening on the maxillary sinus floor or in locations other than the maxillary sinus floor between patients with and without tooth roots penetrating the sinus floor ($P = .958$ and .566, respectively).

Discussion

This study assessed the impact of cigarette consumption, including the status (current and former smokers and nonsmokers), intensity (daily cigarette consumption), duration (years of smoking), and pack-years of smoking on mucosal thickening in 1094 maxillary, ethmoid, and sphenoid sinuses from 547 patients using MRI. The results exhibited that compared to nonsmokers, current and former smokers showed higher rates of mucosal thickening in the maxillary and ethmoid sinuses but not in the sphenoid sinus. These findings suggest that cigarette smoking may have a greater impact on mucosal thickening in the maxillary and ethmoid sinuses than in the sphenoid sinus, possibly due to the location of the sinus ostium relative to the nasal cavity. The primary ostia of both maxillary and ethmoid sinuses open into the middle meatus of the nasal cavity, while the ostium of the sphenoid sinus opens into the sphenoethmoidal recess, a small space above the superior nasal concha in the superior meatus.[23] As a result, the maxillary and ethmoid sinuses may be more susceptible to the effect of inhaled smoke due to their direct connection to the middle meatus of the nasal cavity, while the sphenoid sinus ostium, located relatively posterior and superior, may limit its direct exposure to inhaled smoke. Moreover, this study found no significant differences in the rates of mucosal thickening in all sinuses between current and former smokers, suggesting that the effect of cigarette consumption on mucosal thickening in the sinuses may be long-term and persist even after individuals quit smoking. It is worth noting that the history of cigarette consumption was only associated with flat/irregular thickening of the sinus mucosa but not cystic thickening. This finding may be attributed to the different pathophysiological mechanisms underlying these mucosal thickening, with smoking causing chronic inflammation and irritation that leads to flat/irregular thickening, while cystic thickening may be more likely to occur due to blockage of the ducts of the small mucous glands in the sinus mucosa.[1,24]

Although some studies have assessed the association between smoking status and chronic rhinosinusitis, few have investigated the dose-response effect.[25] In those studies evaluating the dose-response association, daily cigarette consumption and years of smoking were mainly used as cigarette exposure indicators.[9,25] However, it is unclear which exposure indicators provide a more reliable estimate of the risk of developing specific smoking-related conditions.[22] This study included various indicators (the intensity, duration, and pack-years of smoking) in the analysis, and results showed that patients with greater intensity, duration, and pack-years of smoking were more likely to have mucosal thickening in the ethmoid sinus. Despite no significant differences in the pack-years, patients with greater intensity and duration of smoking were more likely to have mucosal thickening in the maxillary sinus. However, when assessing the lesions at different locations of the maxillary sinus, the intensity and duration of smoking were associated only with the lesions on the superior and medial walls of the sinus but not at other sinus locations. Interestingly, the findings exhibited that patients with longer smoking duration were more likely to have mucosal thickening in the sphenoid sinus. This finding might suggest that individuals who smoke over a long time period are more likely to develop sphenoid sinus lesions than those smoking at a higher intensity, which should be further evaluated in specifically designed studies.

There have been largely inconsistent findings regarding the association between cigarette consumption and mucosal thickening in the maxillary sinus, which may be due to small sample sizes of less than 100 subjects.[13-17] This study with large sample size showed that mucosal thickening at the

superior and medial walls of the sinus was significantly more common in current and former smokers than in nonsmokers. However, when looking only at the lesions located in other sinus regions rather than the sinus superior and medial walls, no significant differences were found among individuals with different histories of cigarette consumption. Pathological changes in the maxillary sinus mucosa may be attributed to several causative factors, including dental-origin infections. Previous studies that investigated the association between the maxillary posterior teeth and maxillary sinus mucosal changes have reported inconsistent findings. Some studies report a positive association,[26-28] while others find no correlation.[29,30] Notably, most of these studies have evaluated sinus lesions in all maxillary sinus locations and some did not consider whether the apices of the assessed teeth were close to or even penetrating the sinus floor. However, lesions on the sinus floor may be more directly influenced by dental infections due to their close proximity, particularly in the case of teeth with apices near the sinus floor. In this study, patients with completely or partially edentulous posterior dentition showed a significant association with mucosal thickening on the sinus floor but not at other locations besides the sinus floor. The absence of these teeth might imply possible previous severe dental issues leading to their extraction. These findings suggest that locations of mucosal thickening in the maxillary sinus could be influenced by various causative factors, with cigarette smoking more likely to impact the sinus mucosa in the regions near the primary ostium, while the dentition status is likely to affect the mucosa of the sinus floor. In addition, this study found that tooth root penetration did not demonstrate a significant association with mucosal thickening on the sinus floor or at other locations besides the sinus floor, suggesting that the proximity of healthy maxillary posterior teeth to the sinus floor may have a lesser impact on the sinus mucosa compared to dentoalveolar pathologies.

This study also showed that former and current smokers had significantly higher rates of PMO obstruction than nonsmokers. The higher PMO obstruction rates in former and current smokers also support the hypothesis that cigarette consumption has a greater impact on the sinus mucosa in the regions near the primary ostium. The results exhibited that the presence of an AMO was not associated with the history of cigarette consumption.

The influence of seasonal changes on sinus membrane abnormalities remains an ongoing debate.[31,32] While certain studies suggest an association between seasonal variations and an increased number of sinus membrane abnormalities,[33,34] others find no significant differences in the prevalence and size of these abnormalities across different months and seasons.[32,35] Some studies indicate a higher incidence of sinus membrane abnormalities in early spring and autumn,[36] others during the summer,[37] while some find no seasonal variations at all, including during winter,[38] Notably, it has been proposed that the individual's living and working environment, such as indoor administrative officers in centrally heated buildings versus outdoor labour workers exposed to colder weather, may have a greater impact on the prevalence of sinus membrane abnormalities than the season itself.[32] In this study, the scans included were not limited to a specific season and information regarding the subjects' living and working environments was unavailable. Future studies, specifically designed to investigate the influence of seasonal variations on sinus mucosa in subjects with similar living and working conditions, would be necessary to validate this hypothesis.

This study has some limitations. First, as the study subjects were exclusively selected from a pool of healthy control males participating in previous cancer screening studies,[39-42] the association between cigarette consumption and mucosal thickening in the paranasal sinuses in females remains unknown. Second, the MRI protocol may not be optimal for the evaluation of AMO, which is a tiny anatomical structure usually evaluated by fine-cut computed tomography (CT). However, the incidence of AMO in our study was consistent with that previously reported in the CT studies.[21] Third, as the information regarding the chronic rhinitis or nose allergies of the screened patients was not retrievable, it is uncertain whether individuals with these conditions were included in this study. Moreover, due to the retrospective nature of the MRI scans included in our study, which were not taken for dental indications and the unavailability of dental treatment records, this study could only investigate the status of the maxillary posterior teeth but not the presence of dental origin infections at the time of imaging. Nevertheless, these findings could contribute valuable information for future studies to further explore the associations between different causative factors and sinus mucosal lesions at varying sinus locations. Furthermore, when investigating the association between a patient's smoking status and the presence of a lesion in a specific sinus, the dependent variable was assessed at the patient level, while the independent variables were evaluated at the sinus/side level. Although this statistical analysis approach has been widely used in the literature, it could potentially introduce bias. Future studies should adopt advanced statistical analysis methods, such as multilevel modelling or generalised estimating equations, to minimise potential bias arising from within-patient correlation.

Conclusions

Based on the findings of this study, the following conclusions were drawn:

1. Compared to nonsmokers, current and former smokers may be more likely to exhibit mucosal thickening in the maxillary and ethmoid sinuses;
2. Impact of cigarette consumption on mucosal thickening in the sinuses may be long term and persist even after individuals quit smoking;
3. Both daily cigarette consumption and years of smoking may have a significant impact on the mucosa of the maxillary and ethmoid sinuses; and
4. Smoking appears to have a greater impact on areas near the primary maxillary ostium, while dental status seems to more significantly affect the sinus floor mucosa.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Author contributions

Conception or design of the work: Hung and Ai; acquisition of data: Tse, Leung, King, and Lam; analysis of data: Hung, Shan, and Ai; interpretation of data: Hung, Shan, Leung, Chen, and Ai; drafted the work or revised it critically for important intellectual content: Hung and Ai; approved the version to be published: all authors.

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