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Association between obesity and chronic rhinosinusitis with nasal polyps: a national population-based study

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ABSTRACT

Objectives We performed a cross-sectional analysis of data from the nationwide Korea National Health and Nutrition Examination Survey to evaluate the association between obesity and chronic rhinosinusitis with nasal polyps (CRSwNP) or without nasal polyp (CRSsNP).

Design Retrospective cross-sectional analysis of health survey data.

Setting Voluntary survey of representative South Korean populations.

Participants In total, 32 384 individuals aged 19 years or older with available data on CRS and obesity were included.

Primary and secondary outcome measures Diagnosis of CRSwNP or CRSsNP was performed by trained otolaryngologists through sinus endoscopy and surveys of medical history. General and central obesity was diagnosed using body mass index (BMI) and waist circumference (WC), respectively.

Methods A multivariate logistic regression analysis was used to clarify the association between CRSwNP or CRSsNP and obesity according to BMI and WC. Non-obese individuals were recruited as controls.

Results The prevalence of CRSwNP was higher in the general (OR, 1.438; 95% CI, 1.170 to 1.768; p<0.001) and central (OR, 1.251; 95% CI, 1.031 to 1.520; p=0.033) obesity groups than in the control group. Prevalence of CRSsNP was not correlated with obesity. In a logistic regression analysis, olfactory dysfunction (OR, 1.329; 95% CI, 1.137 to 1.553; p<0.001) and purulent discharge (OR, 1.383; 95% CI, 1.193 to 1.603; p<0.001) showed a higher incidence in the central obesity group than in the control group.

Conclusions We demonstrated an association between CRSwNP and general and central obesity. Further investigations on the mechanism underlying this correlation are necessary for an improved understanding of the pathogenesis of CRSwNP.

Strengths and limitations of this study

- ► This study used data from the Korea National Health and Nutrition Examination Survey, a representative national population-based study in Korea.
- ► The study sample included a large number of participants (N=32 384) who had a wide age range (19–86 years).
- ► This study examined general and central obesity, which are important public health concerns.
- ► The association between chronic rhinosinusitis and obesity was explored after adjusting for age, sex, diagnosis of hypertension and other relevant clinical characteristics.
- ► The causal relationship between obesity and chronic rhinosinusitis phenotypes was not assessed in this study.

INTRODUCTION

Obesity is a rapidly increasing global health problem. Over 1.9 billion people are classified as overweight and over 650 million are categorised as obese, globally.[1] Diseases related to obesity include asthma, cardiovascular diseases and diabetes mellitus. Certain types of cancers are also associated with obesity due to chronic inflammation.[2 3] There have been reports suggesting that obesity can potentially result in chronic inflammatory diseases.[4] However, controversies remain about the effect of obesity in sinonasal inflammatory diseases.[5 6]

Chronic rhinosinusitis (CRS) is defined as persistent inflammation of the sinonasal mucosa due to multifactorial causes.[7] *Staphylococcus aureus* or *Alternaria fungi* have been reported as critical agents for CRS,[8] and antibody deficiencies in primary immunodeficiencies may predispose certain patients to recurrent sinus infections.[9]

CRS is categorised as CRS with nasal polyps (CRSwNP) and CRS without nasal polyps (CRSsNP) based on the sinus endoscopy.[10] CRSwNP is typically characterised as a type 2 inflammatory response, exhibiting enhanced tissue eosinophilia and higher levels of eosinophilic granule protein,[11 12] eosinophil chemotactic proteins, interleukin (IL)-5 and

IL-13.[13 14] In general, patients with CRSwNP have more severe nasal obstruction than patients with CRSsNP, and effective medical treatments for patients with CRSwNP are limited.[15 16] In addition, patients with CRSwNP have a significantly lower health-related quality of life compared with healthy controls.[17]

Several studies have reported that obesity is a potential chronic inflammatory condition and leads to chronic systemic inflammation.[4 18] However, to our knowledge, no previous studies have reported the association of CRSwNP with general and central obesity under the influence of various confounding factors in large nationwide studies. As a representative measure of obesity, body mass index (BMI) is the most widely used parameter for general obesity diagnosis and is correlated with various chronic diseases and mortality risk. However, a limitation of BMI is that it does not represent the body fat distribution.[19] Waist circumference (WC) has been commonly used as a simple and clinically applicable method to evaluate central obesity.[20] Thus, using both of these measurements is appropriate when investigating the relationship between general and central obesity and CRSwNP. In this study, we aimed to investigate this association using data retrieved from the Korea National Health and Nutrition Examination Survey (KNHANES), with subgroup analysis and stratification for major confounding factors.

MATERIALS AND METHODS

Study design and population

The data for the study were collected by the KNHANES between 2008 and 2012. The KNHANES was a national cross-sectional survey conducted by the Ministry of Health and Welfare of South Korea. Specially trained medical investigators used well-designed questionnaires to evaluate representative South Korean populations. The evaluation included physical examination, health-related surveys and nutritional status assessment. In the current study, 34 670 participants aged ≥19 years were included. After excluding 2286 participants with missing data on CRS or obesity, the data of 32 384 participants were analysed. All eligible participants provided written informed consent. The study followed the Ethical Principles for Medical Research Involving Human Subjects based on the Declaration of Helsinki.

General characteristics

Of the total KNHANES study population, only the participants aged ≥19 years were included in this study. Socio-demographic and economic variables of the participants were acquired with a self-reported questionnaire, which collected information on cigarette smoking habits, household income, alcohol consumption, education level and region of residence. Alcohol drinkers were categorised as heavy (>30 g/day), mild (1–30 g/day) and non-drinkers. Smokers were categorised as non-smokers or ex-smokers and current smokers. Education level was categorised as graduation from high school (≥13 years) or not. Household income was classified into quartiles by the number of family members, following recommendations of the WHO.

The medical evaluation included assessment of blood pressure, height, weight and WC. Hypertension was confirmed when the systolic or diastolic blood pressure measurements were repeatedly over 140/90 mm Hg. Height (cm) was measured using a stadiometer (Seca 225, Seca, Germany) while the participants had their upper back parallel to the wall, were facing forward and were without shoes. Weight (kg) was measured by a digital medical weighing scale (GL-6000–20, G-Tech International, South Korea) with participants wearing light clothes. Results were recorded by trained examiners and presented up to the second decimal. The physical examination devices were replaced with recently calibrated devices once a year to maintain the accuracy of results. BMI was calculated by dividing the weight (kg) by the squared height (m^2). WC was measured at the midpoint level between the costal margin and the iliac crest at the end of a normal expiration. Chronic diseases, such as stroke, bronchial asthma, allergic rhinitis, depression and pulmonary tuberculosis, previously diagnosed by a physician were also investigated throughout the survey.

Diagnosis of CRSwNP and CRSsNP

CRS was diagnosed by trained otolaryngologists using sinus endoscopy and surveys of medical histories following the European Position Paper on Rhinosinusitus and Nasal Polyps (EPOS) 2012 guidelines for epidemiological studies.

Each participant was evaluated by a single otolaryngologist, and overall, approximately 200 trained otolaryngologists were involved in the KNHANES. CRS was defined as inflammation of the nose and paranasal sinuses with the presence of two or more of the following symptoms for over 12 weeks: (1) nasal blockage/congestion/obstruction; (2) sinonasal discharge; (3) facial pressure/pain; and (4) reduction or loss of smell.[21] An objective diagnosis of either CRSsNP or CRSwNP was made using sinus endoscopy. The participants' socio-demographics, general health characteristics and comorbidities were compared with determine the risk factors for CRS.

Definition of general obesity according to the BMI

The WHO Regional Office for the Western Pacific Region has announced the following BMI standards: normal, 18.5–22.9 kg/m^2; overweight, 23–24.9 kg/m^2; and obese ≥25 kg/m^2.[22] This definition for obesity was followed by the Korean Society for the Study of Obesity and was used in the present study to define general obesity.[23]

Definition of central obesity according to the WC

The Korean Society for the Study of Obesity recommends that the WC cut-off level for diagnosing central obesity be 90 cm for men and 85 cm for women aged ≥19 years. These cut-off values were defined by receiver operating characteristics curve analysis, based on the representative

sample data acquired during the KNHANES in 1998.[24] The same standards were applied in this study to define central obesity in the participants.

Statistical analysis

All statistical analyses were conducted with the SAS software, V.9.4. Differences with p values below 0.05 were considered statistically significant. SAS/STAT survey analyses were conducted because the KNHANES had applied a stratified cluster sampling method when selecting participants from 2008 to 2012. Descriptive statistics have been presented as means (SEs) for categorical variables and weighted percentages (SEs) for continuous variables and frequencies. A Pearson's χ^2 test was used to compare categorical variables, and an independent samples t-test was used to compare continuous variables. The SAS PROC SURVEYREG was used to analyse continuous variables, and the PROC SURVEYFREQ was used for categorical variables. A multiple logistic regression analysis was conducted by the SAS PROC SURVEYLOGISTIC, after adjusting for multiple variables, to clarify the association between CRS phenotypes and general and central obesity (BMI-related and WC-related, respectively). Models were run after adding age and sex for model 1; smoking and severe drinking for model 2; and family income, residence, hypertension, stroke, bronchial asthma, influenza vaccination, allergic rhinitis, and pulmonary tuberculosis for model 3.

Patient and public involvement

The study population was not involved in the development of the research questionnaires, in the analysis or in drawing conclusions from the results.

RESULTS

Demographics of the study participants

Among the total participants, 32 384 were categorised as normal, overweight or obese, according to BMI. The demographic and clinical characteristics of these participants are presented in table 1. The mean age was 42.65 (0.23) years for participants with normal weight, 47.29 (0.24) years for overweight and 47.05 (0.22) years for obese participants. Factors with significant differences among the groups were age, sex, smoking status, severe drinking, family income, residence, hypertension, depression, bronchial asthma, influenza vaccination, allergic rhinitis and stroke. Using the WC standard, 23 260 participants were considered normal and 8690 participants were diagnosed with central obesity. Table 2 shows the clinical characteristics of the participants according to the presence of WC-based central obesity.

Prevalence of CRS in relation to general and central obesity

The overall prevalence of CRS in the participants categorised by BMI was 5.81 (0.31)% in the normal weight group, 5.91 (0.37)% in the overweight group and 6.70 (0.36)% in the obese group. The normal and obese groups differed significantly when assessed by regression analysis (OR, 1.164; 95% CI, 1.009 to 1.342; p=0.036). The prevalence of CRSwNP in the normal and obese groups was 2.20 (0.16)% and 3.43 (0.25)%, respectively; while that of CRSsNP was 3.36 (0.21)% and 3.50 (0.29)%, respectively.

Among the groups categorised by WC, the overall prevalence of CRS was 5.89 (0.24)% in the normal group and 6.81 (0.39)% in the centrally obese group. The prevalence of CRSwNP was 3.44 (0.26%) in the

Table 1 Participants characteristics according to body mass index-based general obesity

	Normal		Overweight		Obese		
	n=14 432		n=7657		n=10 295		
Variable	n	Weighted % (SE)	n	Weighted % (SE)	n	Weighted % (SE)	P value
Age	42.65 (0.23)		47.29 (0.24)		47.05 (0.22)		<0.001
Sex (female)	9235	57.87 (0.53)	4054	45.15 (0.64)	5520	44.16 (0.55)	<0.001
Severe drinking	2761	20.67 (0.46)	1707	25.51 (0.65)	2316	26.87 (0.57)	<0.001
Current smoking	2936	24.64 (0.48)	1600	26.92 (0.70)	2204	28.61 (0.58)	<0.001
Residence (rural area)	3307	18.15 (1.18)	1771	18.86 (1.27)	2519	20.57 (1.37)	<0.001
Family income (intermediate/high)	8665	58.48 (0.75)	4658	59.99 (0.82)	5930	56.67 (0.79)	0.003
Allergic rhinitis	1198	16.43 (0.61)	501	13.17 (0.71)	670	13.44 (0.62)	<0.001
Hypertension	1979	10.03 (0.31)	1748	17.50 (0.48)	3249	25.38 (0.54)	<0.001
Stroke	227	1.09 (0.09)	165	1.40 (0.12)	249	1.62 (0.12)	0.006
Bronchial asthma	410	2.63 (0.16)	251	2.91 (0.25)	388	3.34 (0.20)	0.027
Influenza vaccination	9123	70.98 (0.56)	4566	68.33 (0.69)	6117	68.01 (0.61)	<0.001
Depression	543	3.42 (0.19)	299	3.45 (0.23)	452	3.85 (0.23)	0.271
Pulmonary tuberculosis	904	5.66 (0.23)	368	4.49 (0.28)	360	3.12 (0.19)	<0.001

Table 2 Participants characteristics according to waist circumference-based central obesity

	Normal		Obese		
	n=23 260		n=8690		
Variable	n	Weighted % (SE)	n	Weighted % (SE)	P value
Age	43.17 (0.21)		50.37 (0.29)		<0.001
Sex (female)	11 315	51.02 (0.39)	4124	49.04 (0.68)	0.023
Severe drinking	4167	23.19 (0.41)	1563	25.99 (0.65)	<0.001
Current smoking	4318	26.81 (0.44)	1419	26.85 (0.68)	0.956
Residence (rural area)	4418	17.74 (1.29)	1989	23.33 (1.71)	<0.001
Family income (intermediate/high)	11 754	58.92 (0.71)	4086	57.61 (0.91)	0.154
Allergic rhinitis	1890	15.62 (0.49)	486	11.86 (0.64)	<0.001
Hypertension	3832	12.10 (0.28)	3183	30.73 (0.65)	<0.001
Stroke	386	1.10 (0.07)	262	2.06 (0.15)	<0.001
Bronchial asthma	672	2.62 (0.13)	388	3.95 (0.25)	<0.001
Depression	866	26.13 (0.97)	429	30.40 (1.66)	0.023
Pulmonary tuberculosis	1288	4.87 (0.17)	351	3.66 (0.22)	<0.001

centrally obese group and 2.40 (0.14)% in the normal group. The prevalence of CRSsNP was 3.66 (0.21)% in the centrally obese group and 3.61 (0.32)% in the normal group (figure 1).

Correlation between CRSwNP prevalence and general and central obesity

Among the groups classified by the BMI, CRSwNP was more prevalent in the general obesity group than in the normal group (OR, 1.582; 95% CI, 1.294 to 1.934; p<0.001), but did not differ between the normal and overweight groups (OR, 1.120; 95% CI, 0.894 to 1.404; p=0.324). Multivariate regression analysis was performed under various confounding factors with models 1, 2 and 3, showing a higher prevalence of CRSwNP in the general obesity group than in the normal group (OR, 1.438; 95% CI, 1.170 to 1.768; p<0.001 in model 3). When participants were classified by the WC, univariate logistic regression analysis showed that the prevalence of CRSwNP in the central obesity group was higher than in the normal group (OR, 1.447; 95% CI, 1.208 to 1.733; p<0.001). Multivariate regression analysis using the three models resulted in a similar outcome (OR, 1.251; 95% CI, 1.031 to 1.502; p=0.023 in model 3) (table 3).

Correlation between CRSsNP prevalence and general and central obesity

The correlation between CRSsNP and obesity was investigated. We found no difference in CRSsNP prevalence between the overweight and normal groups (OR, 1.019; 95% CI, 0.864 to 1.203; p=0.820), but a significant difference was noted between general obesity and normal groups (OR, 1.164; 95% CI, 1.009 to 1.342; p=0.036). Multivariate regression analysis with models 1, 2 and 3 found no difference in CRSsNP prevalence between the normal and overweight (OR, 0.986; 95% CI, 0.832 to 1.169; p=0.872 in model 3) or in the normal and obese (OR, 1.130; 95% CI, 0.975 to 1.310; p=0.104 in model

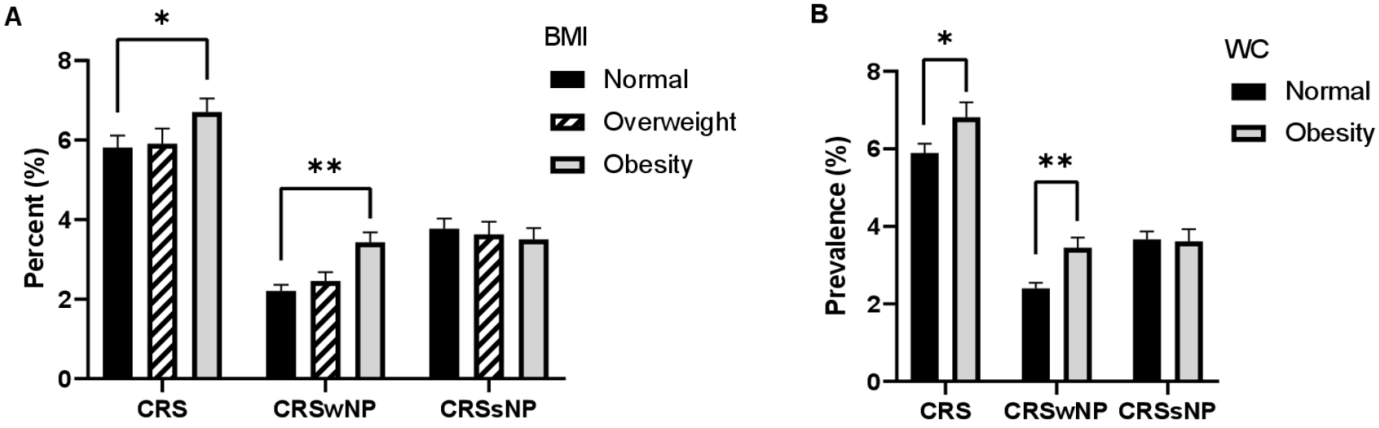

Figure 1 Prevalence of CRSwNP or CRSsNP according to the presence of obesity. (A) Prevalence of CRSwNP or CRSsNP according to general obesity. (B) Prevalence of CRSwNP or CRSsNP according to central obesity. BMI, body mass index; CRS, chronic rhinosinusitis; CRSsNP, CRS without nasal polyps; CRSwNP, CRS with nasal polyps; WC, waist circumference. *p<0.05, **p<0.01 (univariate logistic regression analysis).

Table 3 Association between CRSwNP and obesity according to BMI and WC

Model	Variable	OR (95% CI)	P value
Unadjusted	Overweight vs normal	1.120 (0.894 to 1.404)	0.324
	Obesity vs normal	1.582 (1.294 to 1.934)	0.000*
Model 1†	Overweight vs normal	0.959 (0.765 to 1.204)	0.378
	Obesity vs normal	1.382 (1.129 to 1.693)	0.002*
Model 2‡	Overweight vs normal	0.944 (0.731 to 1.220)	0.660
	Obesity vs normal	1.465 (1.179 to 1.821)	<0.001*
Model 3§	Overweight vs normal	0.968 (0.762 to 1.228)	0.787
	Obesity vs normal	1.438 (1.170 to 1.768)	<0.001*
Unadjusted	Central obesity vs normal	1.447 (1.208 to 1.733)	<0.001*
Model 1†	Central obesity vs normal	1.226 (1.016 to 1.480)	0.033*
Model 2‡	Central obesity vs normal	1.257 (1.021 to 1.547)	0.030*
Model 3§	Central obesity vs normal	1.251 (1.031 to 1.520)	0.023*

*Differences with p<0.05 were considered statistically significant.
†Model 1: adjusted for age and sex.
‡Model 2: adjusted for age, sex, current smoking and severe drinking.
§Model 3: adjusted for age, sex, current smoking, severe drinking, family income, residence, hypertension, stroke, bronchial asthma, influenza vaccination, allergic rhinitis and pulmonary tuberculosis.
BMI, body mass index; CRSwNP, chronic rhinosinusitis with nasal polyps; WC, waist circumference.

3) groups. When grouped by WC, the prevalence of CRSsNP in the central obesity group was higher than in the normal group on univariate analysis (OR, 1.168; 95% CI, 1.020 to 1.338; p=0.024 in model 3) but not in the multivariate regression analysis using the three models (OR, 1.144; 95% CI, 0.993 to 1.318; p=0.063 in model 3) (table 4).

Association of sinonasal symptoms with general and central obesity

In the logistic regression analysis, olfactory dysfunction (OR, 1.329; 95% CI, 1.137 to 1.553; p<0.001) and purulent discharge on sinus endoscopy (OR, 1.383; 95% CI, 1.193 to 1.603; p<0.001) showed a higher incidence rate in the central obesity group than in the normal group.

Table 4 Association between CRSsNP and obesity according to BMI and WC

Model	Variable	OR (95% CI)	P value
Unadjusted	Overweight vs normal	1.019 (0.864 to 1.203)	0.820
	Obesity vs normal	1.164 (1.009 to 1.342)	0.036*
Model 1†	Overweight vs normal	0.956 (0.810 to 1.129)	0.598
	Obesity vs normal	1.091 (0.944 to 1.262)	0.237
Model 2‡	Overweight vs normal	0.960 (0.811 to 1.136)	0.634
	Obesity vs normal	1.110 (0.960 to 1.285)	0.159
Model 3§	Overweight vs normal	0.986 (0.832 to 1.169)	0.872
	Obesity vs normal	1.130 (0.975 to 1.310)	0.104
Unadjusted	Central obesity vs normal	1.168 (1.020 to 1.338)	0.024*
Model 1†	Central obesity vs normal	1.124 (0.980 to 1.291)	0.095
Model 2‡	Central obesity vs normal	1.137 (0.989 to 1.306)	0.071
Model 3§	Central obesity vs normal	1.144 (0.993 to 1.318)	0.063

*Differences with p<0.05 were considered statistically significant.
†Model 1: adjusted for age and sex.
‡Model 2: adjusted for age, sex, current smoking and severe drinking.
§Model 3: adjusted for age, sex, current smoking, severe drinking, family income, residence, hypertension, stroke, bronchial asthma, influenza vaccination, allergic rhinitis and pulmonary tuberculosis.
BMI, body mass index; CRSsNP, chronic rhinosinusitis without nasal polyp; WC, waist circumference.

Table 5 Sinonasal symptoms according to presence of general and central obesity

Symptoms	Variable	OR (95% CI)	P value
Rhinorrhoea/PND	General obesity vs normal	0.736 (0.511 to 1.058)	0.097
	Central obesity vs normal	0.917 (0.763 to 1.101)	0.349
Nasal obstruction	General obesity vs normal	1.302 (0.927 to 1.828)	0.126
	Central obesity vs normal	1.097 (0.928 to 1.297)	0.276
Olfactory dysfunction	General obesity vs normal	1.011 (0.722 to 1.416)	0.948
	Central obesity vs normal	1.329 (1.137 to 1.553)	<0.001*
Daily life disability	General obesity vs normal	1.454 (1.083 to 1.952)	0.012*
	Central obesity vs normal	1.121 (0.948 to 1.326)	0.179
Pale mucosa	General obesity vs normal	0.746 (0.591 to 0.942)	0.013*
	Central obesity vs normal	0.870 (0.760 to 0.996)	0.043*
Purulent discharge	General obesity vs normal	1.400 (0.987 to 1.988)	0.059
	Central obesity vs normal	1.383 (1.193 to 1.603)	<0.001*

*Differences with p<0.05 were considered statistically significant.
PND, postnasal drip; WC, waist circumference.

Daily life disability (OR, 1.454; 95% CI, 1.083 to 1.952; p=0.012) was more prevalent in the general obesity group than in the normal group. Pale nasal mucosa on sinus endoscopy was more prevalent in the normal group than in the central (OR, 0.870; 95% CI, 0.760 to 0.996; p=0.043) and general (OR, 0.746; 95% CI, 0.591 to 0.942; p=0.013) obesity groups. The presence of rhinorrhoea/postnasal drip and nasal obstruction did not differ between the normal and obesity groups (table 5).

DISCUSSION

In the present study, the prevalence of CRSwNP was significantly higher in the BMI-based general obesity and WC-based central obesity groups compared with the normal group. The association between CRSsNP and either general or central obesity was not significant. In addition, common sinonasal symptoms seen in CRS such as purulent discharge and olfactory dysfunction were more frequent in the central obesity group.

Previous studies investigating the association between obesity and sinus infections have shown inconsistent results. Obesity was reported to not affect the severity of sinonasal diseases in patients with asthma.[5] While childhood obesity was associated with the occurrence of acute otitis media, an association between obesity and allergic rhinitis or CRS was not observed.[6] Other studies have shown that a high BMI was associated with the prevalence of CRS.[25 26] However, these studies did not classify the prevalence of CRS based on the presence of nasal polyps; therefore, the effect of adiposity on CRSwNP has not been determined.

In this study, the association between general obesity and CRSsNP was insignificant in contrast to that with CRSwNP, after adjusting for various confounding factors. This result reveals that factors related to general obesity might be closely related to the polypogenesis process in sinonasal inflammatory disease, the mechanism for which has not been fully identified yet. Inflammatory pathophysiological processes are associated with obesity and certain respiratory diseases.[27] Several mechanisms involved in the contribution of obesity to asthma progression have been studied. The increased production of cytokines, adipocytokines, adiponectin and other mediators by the adipose tissue is considered to have important implications in immune responses.[28 29] There are reports indicating that increased levels of inflammatory mediators[30] and metabolic hormones[31] could be the possible causes for immune system dysfunction in obese patients. Therefore, the significant association between CRSwNP and obesity found in this study could be explained by nasal polyps being strongly related to recurrent and severe CRS in most cases,[30] which is possibly due to decreased immune function seen in obesity.

The association between central obesity and CRSwNP was significant after adjusting for various confounding factors, in contrast to CRSsNP, in this study. This difference suggests that mechanisms related to central obesity might impact the pathogenesis of nasal polyps in CRS. The prevalence of central obesity, defined as excessive fat accumulation around the abdomen, is increasing and is associated with elevated mortality risk.[32] Visceral fat is correlated with elevated production of inflammatory factors, leading to increased metabolic complications.[33] Studies have reported that the connection between obesity and inflammatory diseases is mediated by oxidative stress and inflammation.[32] Compared with other fat deposits, visceral fat has more pronounced inflammatory effects, including increased levels of free fatty acids, adipocytokines and inflammatory molecules, all playing critical roles in inflammatory diseases.[34] Superfluous adiposity in the upper trunk has been reported to be a risk factor for dyslipidaemia, type 2 diabetes,

hypertension, cardiovascular diseases and hyperinsulinaemia.[35] The metabolic and endocrine effects of obesity, and the changes in production of hormones including insulin, adipocytokines, insulin-like growth factor-1 and insulin-like growth factor binding protein-3 secreted by adipose tissue have been reported as potential causes for the development of adenomatous polyps and cancer in the colon.[36 37] The mechanism of polypogenesis in the nasal cavity should be further investigated from a metabolic prospective.

We also found that sinonasal symptoms such as olfactory dysfunction and purulent discharge showed a higher prevalence in the central obesity group. This suggests that central obesity might be associated with an increased risk of chronic rhinosinusitis, showing a higher incidence of local sinonasal inflammatory signs. Generally, the mucosa during allergic inflammation is pale and swollen, while it is mostly reddish during acute infections and in overuse of topical nasal sprays.[38] In our study, pale mucosa, which is more prevalent in allergic rhinitis, showed a higher prevalence in the normal group, consistent with a previous report which showed that obese patients have a higher incidence of non-allergic rhinitis.[39] Further investigations on the mechanism behind these observations is warranted.

This cross-sectional study is limited by its inability to evaluate the causal relationship between obesity and the CRS phenotypes. In addition, since the KNHANES is based on self-reported questionnaires in reporting parameters on confounding factors, response biases may have occurred.

Besides the confounders used in the multiple regression analysis, psychiatric conditions,[40] gastro-oesophageal reflux disease,[41] autoimmune disease[9] and various environmental exposures[42] are potential risk factors for CRS. However, these factors were not included as confounders due to unavailability of data. Furthermore, obesity is an important risk factor for asthma in adults,[43] and the possible correlation between severity of asthma and obesity could not be considered in this analysis. Nevertheless, the present study has notable advantages in that it has evaluated the correlation between CRS phenotypes and general and central obesity using a well-defined representative sample of adults in South Korea. In addition, endoscopic examinations and medical interviews were conducted by trained otolaryngologists, and confounding factors related to the association between CRS and obesity were adjusted for in the statistical analysis.

CONCLUSION

This study demonstrated a distinct association between CRSwNP and general and central obesity in a large nationwide population sample. Among sinonasal symptoms, the incidences of olfactory dysfunction and purulent discharge were higher in the central obesity group than in the control group. These results suggest a possible role of obesity in the development of CRSwNP, which is mostly refractory to medical treatment and often recurs even after surgery. Further investigations on the mechanism of this correlation is anticipated for elucidating the pathogenesis of CRSwNP.

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Contributors J-SN and H-JC designed the study and wrote the manuscript. WAF, J-GH and H-EN contributed to data collection. J-SN, WAF and YHR performed the statistical analysis. J-HY, C-HK and H-JC interpreted the results. All authors read and approved the final manuscript.

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