

Allergic Phenotype of Chronic Rhinosinusitis Based on Radiologic Pattern of Disease

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Objectives/Hypothesis: Polypoid edema of the middle turbinate is a marker of inhalant allergy. Extensive edematous changes may result in limited central nasal and sinus disease, which has been called central compartment atopic disease (CCAD). Radiologically, this is seen as soft tissue thickening in the central portion of the sinonasal cavity with or without paranasal sinus involvement. When the sinuses are involved, the soft tissue thickening spares the sinus roof or lateral wall (centrally limited). This centrally limited radiological pattern was assessed among chronic rhinosinusitis (CRS) patients and compared to allergy status.

Study Design: Diagnostic cross-sectional study.

Methods: This study included consecutive CRS patients without prior sinus surgery. Computed tomography (CT) scans of the paranasal sinuses were blindly assessed and allergy status was confirmed by serum or skin testing. Individual sinus cavities were defined as either centrally limited or diffuse disease. The radiological pattern that may predict allergy was determined, and its diagnostic accuracy was calculated.

Results: One hundred twelve patients diagnosed to have CRS, representing 224 sides, were assessed (age 46.31 ± 13.57 years, 38.39% female, 41.07% asthma, Lund-Mackay CT score 15.88 ± 4.35 , 56.25% atopic). The radiological pattern defined by centrally limited changes in all of the paranasal sinuses was associated with allergy status (73.53% vs. 53.16%, $P = .03$). This predicted atopy with 90.82% specificity, 73.53% positive predictive value, likelihood positive ratios of 2.16, and diagnostic odds ratio of 4.59.

Conclusions: A central radiological pattern of mucosal disease is associated with inhalant allergen sensitization. This group may represent a CCAD subgroup of patients with mainly allergic etiology.

Key Words: Chronic rhinosinusitis, paranasal sinuses, allergic rhinitis, aeroallergens, computed tomography.

Level of Evidence: 3b

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INTRODUCTION

Aeroallergen deposition into the nasal cavity causes immunoglobulin E (IgE)-mediated inflammation resulting in middle turbinate edema. Middle turbinate edema is a specific endoscopic sign of aeroallergen sensitization.^{1,2} Brunner and colleagues³ have also defined the high association between allergic rhinitis and middle turbinate polyposis, as a distinct clinical entity to sinonasal polyposis. DelGaudio et al.⁴ reported that this allergic edema may extend to involve the superior turbinate and upper septum, and the resulting mechanical obstruction gives a characteristic centrally limited disease picture called central compartment atopic disease (CCAD) (Fig. 1).⁴ CCAD is a disease process where the primary pathology is allergic edema of the turbinate mucosa, which may secondarily obstruct the sinus ostia with minimal lateral sinus changes, giving rise to a chronic rhinosinusitis (CRS) picture. Radiologically, this is seen as soft-tissue thickening in the central portion of the sinonasal cavity. Sinus involvement spares the roof or lateral wall of the sinus cavity.

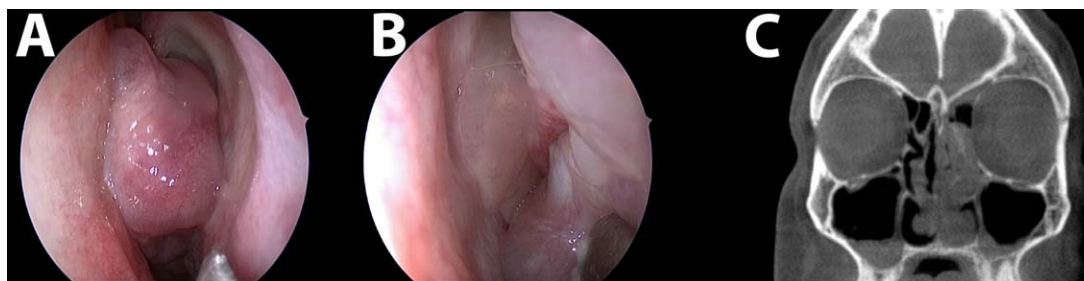


Fig. 1. Left nasal endoscopy and CT scan of a patient with central compartment atopic disease. (A) Left middle turbinate polyps. (B) The left upper septum is involved. (C) Coronal CT scan demonstrated sinus obstruction from the middle turbinate polyposis. The diseased sinuses presented with a central focus, with relatively normal roof and lateral wall of the sinuses. CT = computed tomography. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

This centrally limited disease has been described as middle turbinate polyp with secondary sinus involvement.³ In this study, allergy was suspected as an underlying pathophysiology, as this entity differed from classic sinonasal polyposis with allergic rhinitis over-represented (83% vs. 34%, $P < .001$).³ In contrast, eosinophilic CRS is primarily a non-IgE-mediated inflammatory disorder of the sinuses, classically occurring in adults with or without history of prior inhalant allergy.⁵ These patients tend to present with severe recalcitrant disease. A radiological comparison between individual patients either with eosinophilic CRS or CCAD are presented in Figure 2.

Although an initial description of CCAD was reported by DelGaudio and colleagues,⁴ the potential of a radiologic pattern in CRS disease, to predict an allergic etiology, requires cross-sectional analysis. We hypothesize that a centrally limited pattern of sinus disease is more likely to be associated with inhalant allergen sensitization.

MATERIALS AND METHODS

A cross-sectional diagnostic study was performed on patients diagnosed with CRS who had both allergology and radiological

assessment. This study had obtained prior approval from the local human research ethics committee (HREC-SVH09/083). Informed consent was obtained from all participants.

Study Population

Adults (≥ 18 years old) diagnosed with CRS, either with or without nasal polyps and without prior sinus surgery, were assessed at a tertiary referral clinic. Consecutive patients who had both available allergy testing and radiological examinations were included. The diagnosis of CRS was based on the 2012 European position paper on rhinosinusitis and nasal polyps (EPOS) guidelines.⁵ Allergy assessments were performed by either epicutaneous testing or serological detection for specific IgE. Those with a positive allergy assessment were classified into the allergen-sensitized group. Radiological assessment was performed with a computed tomography (CT) scan. All allergy assessments (epicutaneous or serological) occurred within 3 months of their radiologic assessment.

Patients who were diagnosed with other sinus conditions such as barosinusitis, mucocele, recurrent acute rhinosinusitis, fungal sinusitis, and sinusitis from dental origin, or had prior sinus surgery were excluded. Patients with systemic conditions (immunodeficiency, vasculitis, cystic fibrosis, or granulomatous

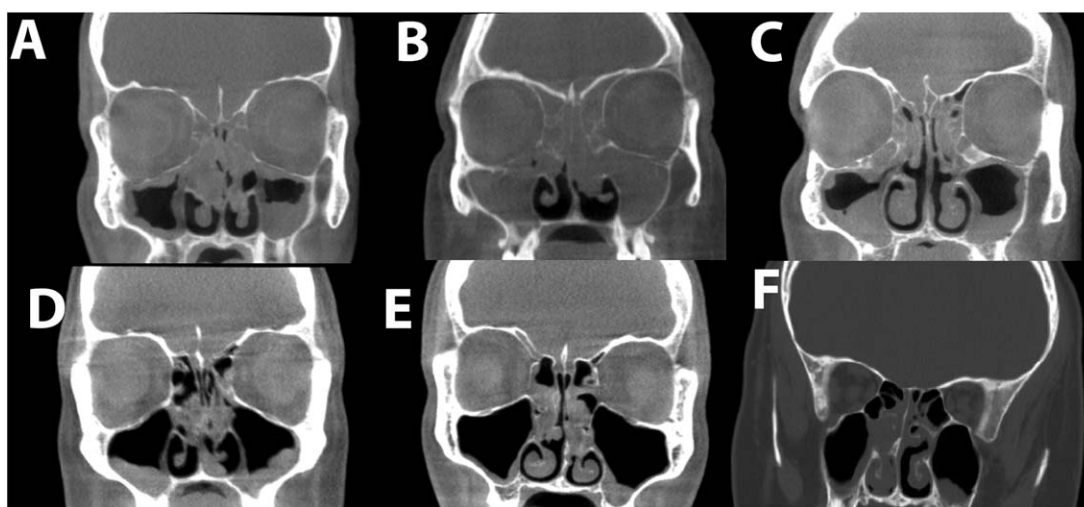


Fig. 2. Radiological characteristic of eCRS and central compartment atopic disease (CCAD). Representative CT scan images of eCRS, with complete opacification of ethmoids and diffuse mucosal involvement of the maxillary sinuses (A–C). This is in contrast to CCAD where there is a central focus of the mucosal disease due to middle turbinate polyposis. If the sinuses are involved, the roof or lateral wall tends to be relatively normal (D–F). CCAD = central compartment atopic disease; CT = computed tomography; eCRS = eosinophilic chronic rhinosinusitis.

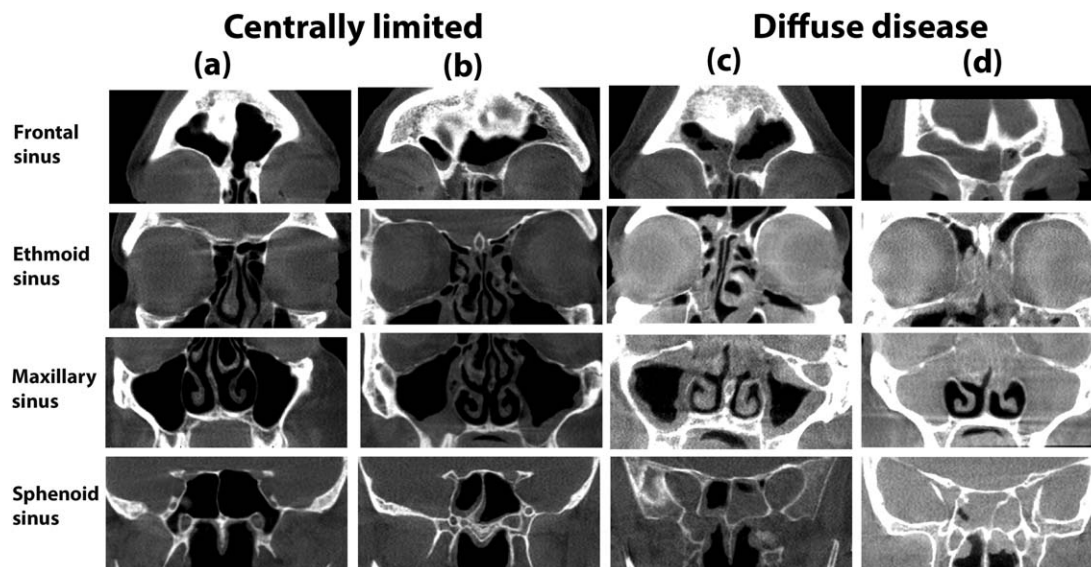


Fig. 3. Reference images used to grade each individual paranasal sinus. The paranasal sinuses were assessed from serial images (0.4-mm slices) on coronal, axial, and sagittal views, and each were classified as either centrally limited or diffuse disease. Centrally limited: (a) normal sinus or (b) mucosal thickening, which involves the floor or medial wall of sinuses. Diffuse disease: (c) mucosal thickening involving all four sinus walls or (d) completely opacified sinus cavity.

disease) that were thought to potentially affect mucosal tissue were also excluded.

Asthma status required additional criteria of either a 15% change in forced expiratory volume in 1 second on spirometry from challenge testing or β -agonist use, or if using regular inhaled bronchodilator/corticosteroid therapy. Smokers were defined as any patient currently smoking or who had ceased within the last 12 months.

All patients filled out a sinonasal symptom questionnaire⁶ at the first scheduled clinic visit. Three rhinologic problems, with relevance to allergy (need to blow nose, sneezing, and runny nose) scored on a scale of 0 to 5 (0 = no problem, 1 = very mild problem, 2 = mild or slight problem, 3 = moderate problem, 4 = severe problem, and 5 = problem bad as can be) were further evaluated.

Allergy Status

Allergen sensitization was determined by either epicutaneous testing or serological assessment. Patients refrained from antihistamines for at least 72 hours prior to testing. Epicutaneous testing was performed using allergens in a 50% glycerin solution. Allergens were applied to the volar forearm with a Multi-test II device. The aeroallergen panel used comprised of dust mites (*Dermatophagoides farina*, *Dermatophagoides pteronyssinus*), molds (penicillium, *Cladosporium* sp. Mix (*Cladosporium cladosporioides*, *Cladosporium herbarum*), *Aspergillus* sp. Mix (*Aspergillus fumigatus*, *Aspergillus nidulans*, *Aspergillus niger*, *Alternaria alternata*), animal epithelium (cat, dog), and grass (7-grass mix [Kentucky Blue/June, meadow, rye, sweet vernal, cocksfoot, timothy], Bermuda grass, Bahia grass, rye grass). Glycerin was used as negative control and histamine 10 mg/mL as a positive control. The wheal size was measured after 15 minutes of application. A positive skin test result was defined as a wheal of more than 3 mm to any one of the allergens with a nonreactive negative control.

Serum-specific IgE toward four allergen mixes that corresponded to the epicutaneous test panel were evaluated (house dust, mold, animal, and grass) by automated immunoassay. A

serum-specific IgE value of 0.35 KU/L or more for any of the mixed airborne antigen mixes was considered positive. Patients were grouped as allergen sensitized if either serology or epicutaneous test was positive. Patients were grouped as nonatopic if they tested negative for both tests.

Radiologic Assessment

Radiological assessment was performed using a compact, upright volume cone beam volumetric tomography scanner (MiniCAT IQ; Xoran Technologies, Ann Arbor, MI). The scans were taken during the first scheduled clinic visit. The paranasal sinuses were assessed from serial images (0.4-mm slices) on coronal, axial, and sagittal views. An ear, nose and throat (ENT) specialist (A.W.H.) blinded to the allergy status scored the scans using the Lund-Mackay scoring system.⁷ The date of the CT scan was also noted. CT scans done during pollen season was defined as scans done during a time that coincided with high temperate grass pollen count (September–March) in New South Wales.⁸

Classification of Individual Sinuses

The same blinded observer classified the frontal, maxilla, anterior ethmoids, posterior ethmoids, and sphenoid sinuses individually guided by reference images (Fig. 3). The classifications were predefined by two ENT specialists (R.J.H. and A.W.H.) based upon a previous radiological description of CCAD.⁴ This classification system was designed to identify a central sinus phenomenon in which inhalant allergy is thought to primarily affect the nasal cavity and turbinates with polypoid edema and only a secondary sinus obstructive phenomenon. Each sinus was classified as either centrally limited or diffuse disease. Centrally limited sinus was defined as having normal sinus mucosa or mucosal thickening involving only the floor or medial wall of the sinus (with either normal roof or lateral wall). Diffuse disease was defined as mucosal thickening involving the roof and lateral wall or all four sinus walls or a completely opacified sinus. Not all sinuses had evidence of disease. Sinus cavities



Fig. 4. Computed tomography (coronal and sagittal views) images of central radiological phenotype. This radiological phenotype was defined from grouping the individual sinuses (previously classified as centrally limited or diffuse disease). (A and B) In this central radiological pattern, all the paranasal sinuses must be centrally limited.

without any thickening were included as centrally limited as this reflects patients with the original description of early CCAD.

Defining the Radiological Pattern of CCAD: Grouping the Sinuses

The above classified individual sinuses were combined to give a radiological pattern that represents central mucosal disease seen in CCAD. This was done per side. We defined the CCAD radiological phenotype as centrally limited disease in all of the paranasal sinuses. This best fitted with the original CCAD description with early sinus involvement⁴ (Fig. 4).

Statistical Analysis

Statistical analyses were performed using SPSS version 24.0 (IBM Corp., Armonk, NY). The Student *t* test was used to compare continuous baseline values between the allergen sensitized and the nonatopic groups. χ^2 analysis was applied for comparisons of proportions. Individual sinus outcomes were dichotomous (either centrally limited or diffuse disease) and tested against allergy using χ^2 analysis. The baseline characteristics of patients with CCAD radiological phenotype and its remaining other (all other paranasal sides without CCAD radiological pattern) were also compared. This statistical analysis was done by sides. The association between CCAD radiological phenotype and allergy were tested using the χ^2 test. The individual symptom scores (need to blow nose, sneezing, and runny nose) were ordinal and were compared between groups using Kendall's τ B test. Data were presented as the proportion of those with a moderate or worse problem. The diagnostic accuracy of the CCAD radiological phenotype was calculated using the status of allergen sensitization as the reference test (sensitivity, specificity, positive predictive value [PPV], negative predictive value [NPV], likelihood ratios positive [LR+] and negative [LR-], diagnostic odds ratio [DOR], and diagnostic accuracy [DA]). Binomial logistic regression was also performed to test contributions of baseline characteristics and individual sinus classifications to predict allergen sensitization. A *P* value $< .05$ was considered significant.

RESULTS

Population

There were 112 patients diagnosed with CRS, representing 224 sides included in this study (age = 46.31 ± 13.57 years, 38.39% female, and 56.25% allergen sensitized). Among these, 41.07% had asthma, 66.07% were diagnosed as chronic rhinosinusitis with nasal polyps (CRSwNP), and 8.93% smoked. Among the allergen sensitized, 53.97 were polysensitized, 68.25% were

sensitized to grass, 65.08% to dust, 28.57% to molds, and 25.45% to animal epithelium. The mean total Lund-Mackay score was 15.88 ± 4.35 and ranged from 5 to 24. The nasal symptoms (as percent moderate problem or worse) were as follows: need to blow nose (64.5%), sneezing (38.2%), and runny nose (51.4%). Among the CT scans, 51% were performed during pollen season. CT scans done during pollen season and out of pollen season were similar among the grass allergen sensitized group (58% vs. 48%, *P* = .29) as were Lund-Mackay scores (15.76 ± 5.24 vs. 16.28 ± 3.76 , *P* = .60). The baseline characteristics between the nonatopic and allergen-sensitized group were also similar, except for age, where the allergen-sensitized group was significantly younger (44.03 ± 13.61 years vs. 49.23 ± 13.07 years; *P* = .04). The use of intranasal steroids was also more frequent among the allergen-sensitized group (Table I).

Classification of Individual Sinus and Allergen Sensitization

Centrally limited disease classification was found in the sphenoid sinuses (67.86%), frontal sinuses (58.93%), posterior ethmoid sinuses (50.0%), maxillary sinuses (

TABLE I.
Baseline Characteristics of Allergen-Sensitized and Nonatopic Populations

Factor	Allergen-Sensitized	Nonatopic	<i>P</i> Value
No.	63	49	
Age, mean $\pm$ SD	44.03 ± 13.61	49.23 ± 13.07	.04
Gender, % female	34.92	42.86	.39
Asthma, %	40.32	46.65	.58
CRSwNP, %	68.25	63.27	.58
Smoking, %	8.33	10.2	.74
Lund-Mackay score	15.60 ± 4.51	16.22 ± 4.13	.29
Intranasal steroids use, %	44.26	22.45	$< .01$
Oral steroids use, %	8.20	8.16	.99
Individual rhinologic score, % moderate problem or more			
Need to blow nose, %	66.1	62.5	.96
Sneezing, %	41.9	33.3	.17
Runny nose, %	51.6	51.1	.20

CRSwNP = chronic rhinosinusitis with nasal polyposis; SD = standard deviation.

TABLE II.

Baseline Characteristics of the Central Radiological Phenotype Compared to the Remaining Non-CCAD Radiological Group (Other)

Factor	CCAD Radiological Phenotype	Other	P Value
No.	34	190	—
Allergen-sensitized, %	73.53	53.16	.03
Age, mean $\pm$ SD	42.17 $\pm$ 11.96	47.05 $\pm$ 13.70	.05
Gender, % female	32.35	39.47	.43
CRSwNP, %	38.24	71.05	<.01
Asthma, %	44.12	40.53	.70
Smoking, %	8.82	8.94	.98
Total Lund-Mackay score	10.94 $\pm$ 2.74	16.76 $\pm$ 3.99	<.01
Intranasal steroids use, %	50.0	31.72	.04
Oral steroids use, %	8.82	8.06	.88
Individual rhinologic score, % moderate problem or more			
Need to blow nose, %	60.6	65.24	.27
Sneezing, %	33.3	39.0	.64
Runny nose, %	51.5	51.4	.95

CCAD = central compartment atopic disease; CRSwNP = chronic rhinosinusitis with nasal polyposis; SD = standard deviation.

44.64%) and anterior ethmoids sinuses (32.59%). However, there was no association between any individual centrally limited sinus cavity and allergen sensitization. The frequency of allergen sensitizations between the centrally limited sinus and the diffuse disease sinus cavity was similar (sphenoid: 59.21% vs. 50.0%, $P=.20$, frontal sinus: 58.33% vs 53.26%, $P=.45$, posterior ethmoid: 59.82% vs. 52.6%, $P=.28$, maxillary sinus: 62% vs 51.61%, $P=.12$, and anterior ethmoid: 63.01% vs. 52.98%, $P=.16$).

The Radiological Patterns of CCAD and Allergen Sensitization

The CCAD radiologic phenotype was associated with allergen sensitization (Table II). The baseline characteristics of the central radiological types were similar to the remaining other non-CCAD group. There was no difference in the individual rhinologic scores (need to blow nose, sneezing, and runny nose) between the CCAD radiologic phenotype and the other non-CCAD radiological patterns of disease. However, proportionally fewer CRSwNP patients were grouped as central radiologic phenotype. The Lund-Mackay score for the central radiological types was lower, and intranasal steroid use was more frequent among the CCAD radiologic phenotype group (Table II).

Dust sensitization was more frequent among the CCAD radiologic phenotype compared the other non-CCAD radiological examinations (58.8% vs. 32.6%, $P<.01$), but not for the other aeroallergen (grass: 44.1% vs. 37.4%, $P=.50$; mold: 26.5% vs. 14.2%, $P=.07$; animal epithelium: 27.6% vs. 23.5%, $P=.10$). Multiple aeroallergen sensitization versus monosensitization was not

TABLE III.

Two-by-Two Tables of the Central Radiological Phenotype

Test	Sensitization		Total
	Sensitized (+ve)	Nonatopic (–ve)	
CCAD radiological phenotype	25	9	34
Other	101	89	190
Total	126	98	224

CCAD = central compartment atopic disease.

found to be associated with CCAD radiologic phenotype (41.2% vs. 28.4%, $P=.14$).

Diagnostic Accuracy of CCAD Radiological Phenotype to Predict Allergen Sensitization

CCAD radiological phenotype gave a sensitivity of 19.84%, specificity of 90.82%, PPV of 73.53%, NPV of 46.84%, LR + of 2.16, LR– of 0.47, DOR of 4.59, and DA of 50.89% to diagnose allergen sensitization. The 2×2 table is presented in Table III.

Contributions of Baseline Characteristics and Individual Sinus Sparing in Predicting Allergen Sensitization

Baseline characteristics and individual sinus classifications were included in a binomial regression model to predict allergen sensitization. In this model, younger age was associated with increased probability of having aeroallergen sensitization by 3% ($P<.01$). No individual sinus classification predicted allergy (Table IV).

TABLE IV.

Binary Logistic Regression Model to Predict Allergen Sensitization Among Patients With Chronic Rhinosinusitis

Variables	OR (95% CI)	Significance
Constant	6.96	<.01
Individual sinus grades, base: centrally limited		
Frontal sinus	0.93 (0.47-1.83)	.83
Maxillary sinus	0.77 (0.42-1.40)	.39
Anterior ethmoid sinuses	0.78 (0.38-1.60)	.50
Posterior ethmoid sinuses	0.99 (0.49-1.99)	.98
Sphenoid sinus	0.91 (0.46-1.81)	.78
Age	0.97 (0.95-0.99)	.01
Asthma, base: normal airway	1.03 (0.58-1.84)	.93
χ^2	$P=.12$	
–2LL	289.98	
Nagelkerke R^2	0.07	
Cox and Snell R^2	0.05	
Hosmer-Lemeshow test	0.21	
Classification accuracy	56.4%	

CI = confidence interval; OR = odds ratio.

DISCUSSION

The radiologically seen central mucosal changes defined by CCAD radiological phenotype were found to be significantly associated with aeroallergen sensitization. This was in keeping with the original description of CCAD by DelGaudio et al.⁴ CCAD was reported as a type of allergic disease with allergic edema involving the central nasal structure of ethmoid origins. It is a disease process primarily due to strong nasal allergy leading to obstructive edema (involving the middle and superior turbinates and posterosuperior nasal septum). Radiologically, it is characterized by soft tissue density in the central nasal cavity with or without sinus involvement. The sinus disease is postulated to occur secondary to simple ostia obstruction and advances from a medial to lateral progression. Thus, the paranasal sinuses in this study were classified to reflect either a centrally limited disease or diffuse disease where mucosa beyond the medial wall or sinus floor was involved. These classified sinuses were then grouped to form the CCAD radiologic phenotype defined as centrally limited disease in all sinuses. Normal sinuses were included as centrally limited, as this reflects the nature of CCAD in early stages. Therefore, although there may be normal sinuses in the CCAD radiologic phenotype group, the centro-nasal portion had mucosal edema. Furthermore, in our study, all patients had at least a diseased ethmoid, which reflects their CRS disease. In this study, data are provided to support the CCAD disease proposed by DelGaudio et al.⁴

Recently, a study on middle turbinate polyposis described the endoscopic equivalent of what is radiologically described as CCAD.³ Brunner et al. described this group as distinct from true nasal polyposis as they were younger (35.4 ± 12.5 years vs 53.4 ± 16.4 years, $P < .001$), more likely to have allergic rhinitis (83% vs. 34%, $P < .001$), less likely to have aspirin exacerbated respiratory disease (16% vs. 0%, $P = .024$), and had less symptom burden. More importantly, they showed that this group had a lower overall radiologic burden based on Lund-Mackay score (2.4 ± 2.8 vs 14.9 ± 6.4 , $P = .008$). These findings are very similar to the lower overall burden on CRS seen between the CCAD group and others in this study (Table II).

Dust was found to be the main allergen associated with CCAD radiologic phenotype. A study by Berrettini et al.⁹ studied 40 patients with perennial allergic rhinitis monosensitized to dust, and reported that 67.5% of these patients had sinus mucosal changes on CT scan. Similarly, patients with allergic rhinitis have been described to be more likely to have sinus radiographic changes compared to the nonallergic rhinitis group.¹⁰ In CCAD, it has been postulated that aeroallergen deposition triggers allergic inflammation, leading to edema and subsequent secondary mechanical obstruction of the sinuses.⁴ The sinus changes on radiology may simply represent ostial occlusion and mucus trapping in patients who may have uncontrolled allergic rhinitis. In this current study, although dust sensitization was associated with CCAD radiological pattern of disease, whether this sensitization is responsible for the allergic

edema and mucosal disease among this CRS group of patients is uncertain. Prior studies have shown that 32% of CRSwNP patients have detectable nasal-specific IgE that was commonly perennial allergens.¹¹ However, this may present the same problem as serum, in which it may be a prior sensitization and not the cause of nasal allergic inflammation. Another study found that 50% of CRSwNP patients sensitized to grass pollen responded to nasal allergen provocation compared to 100% of patients diagnosed with allergic rhinitis.¹² However, some may argue that the presence of large nasal polyps may have attenuated the response. The causal relationship between allergen sensitization and CRS still needs to be further investigated.

Pollen was not found to be associated with this central disease radiological pattern. This may be due to the study design, which does not take into account the pollen season. In contrast, previous studies have proven that pollens may induce radiological changes of the paranasal sinuses among patients with allergic rhinitis. Sinus mucosal thickening has been reported in CT scans or sinus radiographs during pollen season¹³ or following nasal allergen challenge with pollen.^{14,15} In the latter studies, nasal challenges led to new or worsened soft-tissue thickening, mainly within the ostiomeatal complex, maxillary sinus, or ethmoid sinus. Whether these changes would progress with repeated pollen exposure remains an intriguing question. A future study focusing on patients with seasonal allergic rhinitis and investigating the association between central radiological phenotype and pollen allergy in and out of season would complement the presented findings.

The Lund-Mackay CT score was found to be similar between the allergen-sensitized and nonatopic patients among our CRS population (Table I). Other studies also did not find a statistically significant association between atopy status and CRS or differences in the Lund-Mackay CT score between the atopic and nonatopic group.^{16,17} The Lund-McKay score does not differentiate diseased sinuses between 5% opacified sinus and 95% opacified sinus, and does not differentiate location of disease. It is not surprising that an absolute Lund-Mackay score did not differentiate these groups. A more specific instrument is needed to describe the location of disease, as this is the more important factor in CCAD.

When the baseline characteristics were compared between the CCAD radiological phenotype and the remaining group, CCAD were less often defined in our clinic as CRSwNP. This was not surprising, as CRSwNP is a term we use to describe eosinophilic inflammation and true sinonasal polyposis. This relationship might represent a bias, as the authors do not use the term CRSwNP to describe middle turbinate polypoid changes, but use it only for true polyps arising from the middle meatus. Intranasal steroid use was greater in the CCAD subtype of CRS (50.0% vs 31.72%, $P = .04$). Whether patients remained more compliant with intranasal corticosteroid use because their condition was primarily allergic or nasal cavity in origin might be an area for future study.

This study has a cross-sectional design that is representative of only one point in time, whereas CT scan features may change with the season, disease state, and medications. There was also no difference in individual rhinologic symptoms between CCAD radiologic phenotype and other radiological pattern (Table II). Unfortunately, these simple scores did not differentiate those with clinically significant allergic symptoms. Allergic rhinitis symptoms among CRS patients might be better assessed by formal allergic history taking (nasal symptoms triggered in the presence of offending allergens or seasonality, allergic comorbidities), nasal endoscopy (middle turbinate edema, turbinate hypertrophy), and appropriate questionnaires for allergic rhinitis. Ultimately, a combination of a radiological CCAD pattern of disease, endoscopic findings of middle turbinate edema, and allergic symptomatology might best define an allergically predisposed CRS patient. This would need further research

CONCLUSION

A centrally limited pattern of mucosal disease, defined by centrally limited changes in all paranasal sinuses on radiology, is associated with inhalant allergen sensitization. This central radiological pattern may represent a CCAD subgroup of patients for which further allergology investigations could be of benefit.

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