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ORIGINAL RESEARCH

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Does chronic rhinosinusitis relate to systemic hypoxemia?

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Abstract

Objectives: Determine if chronic rhinosinusitis (CRS) is associated with systemic hypoxemia.

Methods: Outpatient otolaryngology visits for 12 months were analyzed, identifying patients with a diagnosis of CRS, without a comorbid diagnosis of pulmonary disease, and an oxygen saturation measurement within 14 days of the otolaryngology visit. Oxygen saturation (SpO_2) measures (average SpO_2, minimum SpO_2 and proportion abnormal SpO_2) were compared between CRS patients (with nasal polyps [NP] and without NP) and a control cohort of otology patients, also without pulmonary disease with univariate and multivariate analysis.

Results: Among 640 unique CRS patients with 3105 encounters, the mean and minimum SpO_2 measurements were 97.6% (97.5%-97.7%) and 97.3% (97.2%-97.5%), respectively. Among 3613 control patients with 25 073 encounters, the mean and minimum SpO_2 measurements were 97.3% (97.3%-97.4%) and 97.1% (97.1%-97.2%), respectively. When comparing mean and minimum SpO_2 among CRSsNP (97.5% and 97.2%), CRScNP (97.3% and 97.0%) and control patients (97.3% and 97.1%), no statistically significant differences were found among the 3 groups in mean and minimum SpO_2 adjusting for age and sex (P = .183 and P = .464, respectively, ANOVA). With respect to the presence of an abnormally low oxygen saturation ($SpO_2 \leq 94\%$), 4.4% of the CRSsNP, 10.9% of the CRScNP and 7.3% of the control patients demonstrated a low oxygen saturation (P = .013).

Conclusion: CRS alone does not objectively contribute to systemic hypoxemia, although a subset of CRScNP patients may have abnormally low SpO_2, possibly warranting SpO_2 assessment in this group of patients.

Level of evidence: 3.

KEYWORDS

asthma, chronic rhinosinusitis, dyspnea, hypoxemia, hypoxia, unified airway

1 | INTRODUCTION

Chronic rhinosinusitis (CRS) is a chronic inflammatory condition involving the nose and paranasal sinuses.[1] Although the pathophysiology of CRS remains elusive, several analogies have been drawn between the inflammation of the paranasal sinuses in CRS and the inflammation occurring in the lower airways, including the inflammation that accompanies asthma. In broad terms, this has led to the

unified airway hypothesis, in which similar and bidirectional disease processes may occur in both the paranasal sinuses and lungs.[2-4]

Not uncommonly, patients with CRS will report of a sense of dyspnea accompanying their sinusitis symptoms. In a significant proportion of CRS patients, such symptoms of shortness of breath and a sense of "air hunger" may be attributable to a comorbid diagnosis of asthma, which often accompanies CRS.[5] However, it is not clear why such symptoms of dyspnea might arise in CRS patients who *do not have* a comorbid diagnosis of asthma. We hypothesized that CRS, even in the absence of asthma, may have an effect on systemic oxygenation, thus potentially contributing to a patient's sense of dyspnea or the not uncommonly self-described sensation by patients of "not getting enough oxygen". In order to test this hypothesis, we constructed a cross-sectional cohort study of CRS patients vs a control cohort of otologic patients comparing their peripheral oxygen saturation levels.

2 | METHODS

A retrospective medical records review was conducted for outpatient visits to our major teaching hospitals' otolaryngology clinics for calendar year 2019. This study was approved by the Partners Healthcare institutional review board. Three historical cohorts were constructed. The first two cohorts consisted of those adult patients receiving an ICD-10 diagnoses of chronic rhinosinusitis without nasal polyposis (J32.* without J33.*) and those patients with sinonasal polyposis (J33.*), at their otolaryngology (ORL) visit and who had a recorded peripheral oxygen saturation (SpO_2) obtained with a standard pulse oximeter within 14 days of that visit. This oxygen saturation measurement could have occurred either at the otolaryngology visit or at another clinical venue. The third comparison (control) cohort consisted of adult patients who were seen and diagnosed with a primary otologic diagnosis (ICD-10 codes H60.*-H70.*) and who similarly had a recorded peripheral SpO_2 within 14 days of the clinic visit. The electronic medical record was further queried, and those patients with a comorbid diagnosis of asthma, chronic obstructive pulmonary disease, chronic bronchitis or bronchiectasis (collectively, pulmonary disease) within 1 year at any medical visit were excluded from the subsequent analysis for each cohort.

For each patient in each cohort, the SpO_2 was recorded along with standard demographic information. For patients who had more than one encounter identified with a corresponding SpO_2, their average SpO_2 and lowest recorded SpO_2 (min SpO_2) were utilized. O_2 saturations less than 80% were considered spurious and excluded from the analysis. Data were imported into SPSS (version 25.0, IBM Corporation, Armonk, New York) and analyzed. Standard demographic data and distributions were computed. Univariate testing was conducted, comparing the average SpO_2 and minimum SpO_2 between gender, age, CRS patients (CRSsNP and CRScNP combined) vs control patients, and CRSsNP vs CRScNP patients with Student's *t*-test.

Further analyses were conducted segregating the CRS patients with (cNP) and without (sNP) polyps and comparing SpO_2 among the three cohorts (CRSsNP, CRScNP and controls) using analysis of variance (ANOVA), adjusting for age and sex differences between the three cohorts. Finally, the proportion of patients with an abnormally low SpO_2 (≤94%) was determined for each cohort and compared with chi-square. Statistical significance was set at $P = .05$. Data are reported as mean value or proportion and associated 95% confidence interval (CI).

3 | RESULTS

Overall, 5156 ORL encounters were identified with a CRS diagnosis and an oxygen saturation measurement within 14 days of the ORL visit. After exclusion of patients with a co-morbid diagnosis of pulmonary disease, 3105 encounters remained, and 640 unique CRS patients (53.6% female, mean age, 59.2 years) were ultimately identified. Among CRS patients, 119 had NP vs 521 without NP. Similarly, 25 073 ORL encounters with an otology diagnosis with an oxygen

TABLE 1 Univariate analysis of oxygen saturations

	O2 Saturation (mean)					O2 Saturation (minimum)				
Variable	**Value**	**95% CI**		**p-value**		**Value**	**95% CI**			**p-value**
Age										
<60	97.8	97.7	97.9	<0.001		97.6	97.5	97.7		<0.001
>=60	97.2	97.1	97.2			96.9	96.8	97.0		
Sex										
Female	97.5	97.4	97.6	<0.001		97.3	97.2	97.4		<0.001
Male	97.2	97.1	97.3			97.0	96.9	97.0		
Patient Group										
Chronic rhinosinusitis	97.6	97.5	97.7			97.3	97.2	97.5		
with polyps	97.3	97.0	97.6	0.038	<0.001	97.1	96.8	97.4	0.065	0.002
without polyps	97.6	97.5	97.8			97.4	97.2	97.5		
Control	97.3	97.3	97.4			97.1	97.1	97.2		

TABLE 2 Analysis of variance for oxygen saturations among groups[a]

	O2 Saturation (mean)					O2 Saturation (minimum)			
Patient Group	**Value**	**95% CI**		**p-value**		**Value**	**95% CI**		**p-value**
Chronic rhinosinusitis without polyposis	97.5	97.3	97.6	0.183		97.2	97.1	97.4	0.464
Chronic rhinosinusitis with polyposis	97.3	97.0	97.6			97.0	96.7	97.4	
Control	97.3	97.3	97.4			97.1	97.1	97.2	

*Adjusted for age and sex

saturation measurement within 14 days of the visit were identified. After exclusion of patients with a comorbid diagnosis of pulmonary disease, 21 220 encounters remained with 3613 unique otology patients (57.2% female; mean age 66.5 years) identified. The oxygen saturation measurements were recorded on average 3.9 and 4.7 days apart from the ORL visit in the CRS and otology cohorts, respectively.

Table 1 presents the univariate oxygen saturation data comparisons by gender, age and between CRS and control patients. On univariate analysis, CRS patients actually exhibited a slightly higher mean O_2 saturation and minimum O_2 saturation vs the otology patients ($P < .001$, and $P < .001$, respectively). With respect to subgroup analysis for CRS with and without polyps, patients with CRS with NP did exhibit lower mean and minimum O_2 saturations relative to the CRS without NP cohort (Table 1).

When comparing mean and minimum O_2 saturations among CRSsNP (97.5 and 97.2%), CRScNP (97.3% and 97.0%) and control patients (97.3% and 97.1%), no statistically significant differences were found among the three groups in mean and minimum O_2 saturations when adjusted for age and sex ($P = .183$ and $P = .464$, respectively, ANOVA, Table 2). Finally, with respect to the presence of an abnormally low oxygen saturation ($SpO_2 \leq 94.0\%$), 4.4% of the CRSsNP, 10.9% of the CRScNP and 7.3% of the control patients demonstrated a low oxygen saturation measurement ($P = .013$).

4 | DISCUSSION

It is generally well recognized that CRS imparts a symptom and health burden that extends far beyond the paranasal sinuses themselves.[1] CRS is implicated in higher rates of depression, anxiety, absenteeism, bodily pain and more difficult to manage and refractory asthma disease levels.[1,6,7] One of the critical tasks for the otolaryngologist diagnosing and treating patients with CRS is to be able to ascribe with some reasonable certainty which symptoms and comorbid illnesses may be directly related to the presence of CRS. Given the complex and yet to be elucidated bidirectional interactions between the sinonasal cavity and the lower airways, it is conceivable that chronic rhinosinusitis may have an influence on oxygenation and transport even in the absence of comorbid asthma.[8] As such, it would be somewhat natural to describe a symptom of dyspnea or "air hunger" to underlying CRS, but objective data are lacking.

We found that after adjustment for age and sex and in the absence of pulmonary disease, CRS patients did not exhibit lower measures of O_2 saturation relative to a cohort of non-CRS patients. These data would suggest that CRS does not seem to directly affect peripheral oxygen saturation or hypoxemia. In the absence of objective evidence, the source for the dyspnea that sometimes arises in CRS patients remains unclear. One might also be tempted to ascribe the dyspnea as being secondary to the nasal obstruction that might accompany CRS, such as in cases of substantial sinonasal polyposis with nasal obstruction. In fact, although we did not demonstrate a significant difference in mean or minimum O_2 saturation among the three cohorts, we did identify a ~11% rate of abnormally low O_2 saturation among CRScNP patients. Thus, there may be an explanation for air hunger or shortness of breath particularly in patients with NP. The current data suggest that there might be some screening value for decreased O_2 saturation among patients with NP even in the absence of a concurrent diagnosis of asthma or COPD. This finding warrants further study.

Several limitations of the current study merit mention. First, the oxygen saturation measurements were not taken on the exact same day as the ORL visit in many of the cases, rather they were recorded from other clinical venues. However, they were recorded within approximately 4 to 5 days of the ORL visit for each cohort, which would be a relatively short time period between visits to account for variability at least by comparison between the cohorts. Second, it is possible that there were undiagnosed cases of asthma or COPD within 1 of the cohorts. Given the finding of a significant percentage of low oxygen saturation measurements in the CRS with NP group, this would be worthy of further study and we plan to make this the basis of a prospective cohort study. Finally, it is not the intent to dissociate the symptom of dyspnea (which patients may truly be feeling) in the CRS setting from the CRS diagnosis completely. Rather, we did not find an objective correlation of the symptom in terms of measurable differences relative to a control group. This implies that further exploration of this symptom and its cause is still warranted.

5 | CONCLUSION

Adult patients with CRS diagnoses and without pulmonary disease did not demonstrate significantly different oxygen saturation levels when compared to a control cohort of patients. CRScNP patients did however exhibit a substantial rate of abnormally low oxygen saturation levels which may explain such symptoms of dyspnea or "air hunger" described by a portion of this group of CRS patients.

CONFLICT OF INTEREST

The author declares no potential conflict of interest.

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