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Sleep Quality and Disease Severity in Patients with Chronic Rhinosinusitis

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Abstract

Objective—To evaluate sleep quality in patients with chronic rhinosinusitis (CRS) using a validated outcome measure and compare measures of CRS disease severity with sleep dysfunction.

Study Design—Cross-sectional evaluation of a multi-center cohort

Methods—Patients with CRS according to the 2007 Adult Sinusitis Guidelines were prospectively enrolled from four academic, tertiary care centers across North America. Each subject completed the Pittsburgh Sleep Quality Index (PSQI) instrument, in addition to CRS-specific measures of quality-of-life (QOL), endoscopy, computed tomography (CT), and olfaction. Patient demographics, comorbid conditions, and clinical measures of disease severity were compared between patients with “good” (PSQI; ≤ 5) and “poor” (PSQI; >5) sleep quality.

Results—Patients (n=268) reported a mean PSQI score of 9.4(range: 0–21). 75.0% of patients reported PSQI scores above the traditional cut-off indicating poor sleep quality. Patients with poor sleep quality were found to have significantly worse QOL scores on both the Rhinosinusitis Disability Index ($p<0.001$) and 22-item Sinonasal Outcome Test ($p<0.001$). No significant differences in average endoscopy, CT, or olfactory function scores were found between patients with good or poor sleep quality. Tobacco smokers reported worse average PSQI total scores compared to non-smokers($p=0.030$). Patients reporting poor sleep were more likely to have a history of depression, even after controlling for gender ($p=0.020$).

Conclusion—The majority of patients with CRS have a poor quality of sleep as measured by the PSQI survey. Poor sleep quality is significantly associated with CRS-specific QOL, gender, comorbid depression, and tobacco use but not CT score or endoscopy grade.

MeSH Key Words

Sinusitis; chronic disease; sleep; quality of life; rhinitis; cross-sectional studies

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INTRODUCTION

Very little is known about sleep quality in patients with chronic rhinosinusitis (CRS). Although prior studies suggest that sleep-related complaints may be common, few investigations have explored sleep quality beyond single questions. From a pathophysiologic standpoint, sleep impairment in CRS remains highly plausible and could be related to many factors including nasal obstruction, depression, gender, pain, direct neural signaling, or by systemic or local neural-immune signaling via pro-inflammatory somnogenic cytokines.[1–6] Consistent poor sleep can have staggering impacts on an individual's performance, overall quality-of-life (QOL), and even mortality. Given that upwards of 13% of adults in the United States have CRS[7], sleep dysfunction would have important implications for patients, physicians, and public policymakers alike.

The goal of this study was to evaluate sleep quality in a multi-institutional cohort of patients with CRS utilizing a validated sleep assessment instrument. Differences in demographics, comorbid conditions, clinical measures of disease severity, and disease-specific QOL were compared between those patients with normal and reduced sleep quality.

METHODS

Patient Population and Data Collection

Adult patients (≥18 years) with CRS were enrolled into an ongoing prospective, observational cohort investigation within four, academic, tertiary rhinology practices including: Oregon Health & Science University (Portland, OR.), the Medical University of South Carolina (Charleston, SC.), University of Calgary (Calgary, Alberta, Canada), and Stanford University (Palo Alto, CA.). Study subjects underwent standard clinical examinations consisting of physical evaluations, computed tomography (CT) imaging of the coronal plane, and bilateral sinonasal endoscopy.

Inclusion criteria consisted of a current diagnosis of symptomatic, refractory CRS as defined by the 2007 Adult Sinusitis Guidelines[8], previous treatment with oral, broad spectrum or culture directed antibiotics (≥2 weeks) and either topical nasal corticosteroid sprays (≥3 weeks) or a five-day trial of systemic steroid therapy. Patients were required to complete all study questionnaires and provide informed consent in English. The Institutional Review Board at each site monitored and approved all investigational protocols.

Patients were asked to provide demographic, social, and medical history data including: age, gender, current tobacco use, nasal polyposis, depression, asthma, allergies (either patient history, confirmed skin prick, or radioallergosorbent testing), acetylsalicylic acid (ASA) intolerance, cystic fibrosis, and history of prior sinus surgery. Patients diagnosed with a current exacerbation of recurrent acute sinusitis were excluded from final analyses. Patients diagnosed with obstructive sleep apnea by either testing or via medical history (n=34) were also excluded from all patients enrolled between February, 2011 and September, 2012.

Measurement of Sleep Quality

All patients completed the Pittsburgh Sleep Quality Index (PSQI) at enrollment with the assistance of a trained research assistant. The PSQI is an 18-item, self-reported measure of sleep quality and duration during the four week time period preceding survey completion. The PSQI yields seven component or sub-domain scores. Sub-domain component scores (range:0–3) are assessed using a publicly available scoring algorithm and summarized to obtain a total score (range:0–21). Higher PSQI scores suggest greater sleep disturbance. A PSQI score ≤5 is considered "good" sleep quality whereas a score >5 is associated with "poor" sleep quality.[3]

Disease-Specific Quality of Life Measures

Study participants also completed two CRS-specifics QOL instruments: the Rhinosinusitis Disability Index (RSDI) and the 22-item Sinonasal Outcome Test (SNOT-22). The RSDI (range: 0–120) is a 30-item, disease-specific survey instrument consisting of three subscales that evaluate the impact of CRS on a patient's physical (range: 0–44), functional (range: 0–36), and emotional (range: 0–40) subdomains.[9] Higher sub-domain and total scores indicate greater impacts of chronic sinonasal disease. The SNOT-22 is a validated, treatment outcome measure applicable to chronic sinonasal conditions (range: 0–110). Lower total scores on the SNOT-22 suggest better QOL and symptom severity.[10] These two instruments were chosen because they capture CRS-specific health impacts in a complementary fashion. The enrolling physicians at each site were blinded to all survey responses for the study duration.

Disease Severity Measures

Computed tomography images were evaluated and staged in accordance with the Lund-Mackay bilateral scoring system (range: 0–24) where higher scores represent higher severity of disease.[11] Endoscopic exams were scored using the Lund-Kennedy endoscopy staging system (range: 0–20) where higher scores represent worse disease severity.[12] This staging system quantifies the bilateral severity of nasal polyposis, discharge, edema, scarring, and crusting. All visualizations were scored by the enrolling physician at each site at the time of enrollment.

Olfactory function was measured at the initial enrollment period using The Brief Smell Identification Test (B-SIT; Sensonics,Inc., Haddon Heights,NJ). The B-SIT is a validated 12-item, standardized, non-invasive quantitative test of olfactory function that employs 12 microencapsulated odorant strips in a "scratch-'n-sniff" format (range: 0–12) with higher scores indicating a better sense of smell. Complete B-SIT scores ≥ 9 are defined as "normal" for healthy males and females of all ages.[13]

Statistical Analysis

Statistical comparisons were performed using a commercially available statistical software (SPSS ver.21, IBM Corp., Chicago, IL). Descriptive analytics (means, standard deviations (SD), frequencies, and ranges) were completed for demographic variables, clinical measures of CRS disease severity, and sleep quality data. Assumptions of normality and linearity were verified for continuous measures using graphical analysis. Two-tailed independent sample t-tests and Mann-Whitney U tests were used to evaluate differences between sleep quality subgroups and patient characteristics where appropriate. Pearson's chi-square tests were used to evaluate frequency differences in sleep quality subgroups. Due to the ordinal nature of the PSQI sub-domain scores, we used Spearman's correlation coefficient (r_s) to evaluate nonparametric bivariate correlations between all clinical measures of disease severity and PSQI total and sub-domain scores. Simple logistic regression was utilized to identify and adjust for significant, independent cofactors or effect modification associated with poor sleep quality and to identify possible collinear measures. Crude and adjusted odds ratios (OR) with corresponding 95% confidence intervals (CIs) are reported where appropriate.

RESULTS

A total of 268 patients completed all eligibility requirements for study enrollment including the PSQI questionnaire. Patients with CRS reported a mean PSQI total score of 9.4(4.4) with range 0 – 21. The breakdown of PSQI sub-domain and PSQI total scores by patient characteristic is provided in Table 1. Females reported significantly worse total PSQI scores compared to men. In addition, patients with depression reported worse PSQI scores

compared to those patients without a history of depression. Patients who currently smoked had worse PSQI total mean scores compared to non-smokers.

Each patient's sleep condition was evaluated and dichotomized into those reporting "poor" (n=201;75.0%) and "good" (n=67;25.0%) sleep quality. Demographic and patient characteristics are outlined in Table 2 stratified by severity of sleep impairment. There was a higher prevalence of poor sleep quality in women than men (58.7% vs. 41.3%) and depression was found to be more prevalent in patients reporting poor sleep quality.

Disease Severity as Measured by Endoscopy, CT, and B-SIT

The mean Lund-Kennedy endoscopy score for the patient group with poor sleep was 6.4(4.1) and ranged 0–20, while the mean Lund-Mackay CT score was 12.2(5.7) and ranged 1–24. Mean olfactory function B-SIT scores were 9.2(3.1) and ranged 1–12. Bivariate analysis found no significant differences ($p \geq 0.190$) in mean endoscopy, CT, or olfactory function B-SIT scores between patients reporting good sleep quality and poor sleep quality (Table 3).

Disease Severity as Measured by QOL Instruments

Disease-specific QOL scores as measured by the RSDI and SNOT-22 instruments are reported in Table 4. Patients with poor sleep quality reported significantly worse scores on both the total RSDI and all sub-domains of the RSDI as compared to those with good sleep quality. In addition, subjects with poor sleep quality reported significantly worse average SNOT-22 scores as compared to those patients reporting good sleep quality.

Moderate correlation coefficients were found between PSQI total scores and both the RSDI total ($r_s=0.54;p<0.001$) and SNOT-22 total scores ($r_s=0.63;p<0.001$;Table 5). Furthermore, weak and moderate correlations were found between the RSDI and SNOT-22 total scores and all sub-domains of the PSQI. No significant correlations were found between PSQI measures and other measure of disease severity such as CT, endoscopy, or olfactory function scores, with the exception of a weak correlation between PSQI sleep medication sub-domain score and CT score ($r_s=0.15;p=0.012$). Both the RSDI physical sub-domain and SNOT-22 instruments contain specific survey items directly pertaining to sleep quality and function which may, at least in part, account for the significant associations with the PSQI survey instrument. Removal of these survey items did not fundamentally change the correlation between total scores from the PSQI and RSDI ($r_s=0.53;p<0.001$) or SNOT-22 ($r_s=0.55;p<0.001$) instruments.

Logistic Regression Modeling for Poor Sleep Prevalence

Due to the fact that we found a higher prevalence of female subjects with a history of depression (23.1%) compared to male subjects (12.4%) and a higher prevalence of females reported poor sleep quality (80.3%) compared to males (68.6%), we used simple logistic regression modeling to further assess the relationship between these patient cofactors and sleep quality. A history of depression (OR:3.48, 95% CI: 1.32, 9.17;p=0.012) and gender (OR:1.86, 95% CI: 1.07, 3.26;p=0.029) were both found to be an independent risk factors for poor sleep quality in patients with CRS. After adjusting for gender prevalence, depression was still a significant independent predictor of poor sleep quality (OR:3.19, 95% CI: 1.20, 8.48;p=0.020). Furthermore, worse total RSDI scores and SNOT-22 score were independently associated with worse sleep outcomes ($p<0.001$), however collinearity was found between both QOL measures and our clinical measure of depression, a trend similarly identified in previous investigations.[6] Ultimately, QOL measures were strongly associated with worse PSQI total scores after controlling for depression.

DISCUSSION

Patients with CRS reported impaired quality of sleep across all subdomains of the PSQI survey with the majority of patients reporting PSQI scores above the traditional cut-off indicating poor sleep quality. Patients reporting poor sleep were more likely to have comorbid depression compared to patients reporting good sleep quality, and this difference persisted after controlling for gender. Significantly worse sleep was also found in patients who were current smokers. Poor sleep quality was significantly correlated with CRS-specific QOL as measured by both the RSDI and the SNOT-22 instruments, even after eliminating sleep-related questions from these instruments. No significant differences in endoscopy score, CT, or olfactory function were found between patients with good or poor sleep quality. These significant findings persisted after excluding those patients diagnosed with obstructive sleep apnea from the analysis.

This study suggests that patients with symptomatic CRS have a high prevalence of pathological sleep dysfunction, much greater than that typically identified in the general population (15–35%).[14] These findings coincide with previous studies which have suggested that sleep may be impaired in patients with CRS. Sleep dysfunction is common in other chronic diseases, including but not limited to, fibromyalgia,[15] rheumatoid arthritis,[16,17] ankylosing spondylitis,[18] myasthesia gravis,[19] multiple sclerosis,[20] and cystic fibrosis.[21,22] However, the prevalence of sleep dysfunction in our cohort was greater than that typically identified by the PSQI in these chronic disease populations(range: 50–59%).

Reduced quality of sleep is likely multifactorial in patients with CRS and may be due in part to gender or depression. There is a strong established correlation between sleep disorders and depression[2], with depressed patients reporting diminished sleep quality.[20] Both depression and sleep disorders have been found to be more prevalent in women. Poor sleep quality has been shown to be highly prevalent in women measured both subjectively and objectively.[3,21] Depression has been found to be a common comorbidity in patients with CRS with a prevalence as high as 25%[23,24] and with associated worse disease-specific QOL.[25] We found that women with CRS report worse sleep quality. Therefore, due to the fact there was a higher prevalence of depression and reported poor sleep quality in females, logistic regression modeling was performed, which demonstrated that a history of depression is an independent risk factor for poor sleep quality after controlling for gender. Disturbed sleep may ultimately reveal a link in this complex two-way relationship between female patients with depression and CRS.

Smoking has been associated with delayed sleep onset, nighttime arousals, and daytime sleepiness,[26] while cessation can improve quality of sleep.[27] Objective measures of sleep using polysomnography found that smoking was independently associated with sleep fragmentation.[28,29] Nicotine consumption increases vigilance, sleep latency and fragmentation, and daytime sleepiness, while decreasing sleeping time, slow wave sleep with reduced sleep efficiency.[29] Additionally, nicotine replacement therapy given for smoking cessation causes disturbed sleep.[30] Several important factors related to smoking besides nicotine consumption include nasal irritation, nasal congestion, increased airway resistance and reduced inspiratory flow,[31] which may be contributing to sleep disordered breathing in our patient population. Our data adds to the current literature further suggesting smoking is associated with poor sleep quality. However, questions still remain unanswered by these findings. Further studies need to be done to determine the mechanism by which smoking and/or nicotine affects sleep quality in patients with CRS. Ultimately nicotine concentrations peripherally and centrally need to be evaluated and correlated with objective sleep measures.

Subjective QOL and sleep quality is reduced in many chronic diseases[16–18,20–22,32] and results of prior studies suggest that quality of sleep seems to play a primary role impairing overall QOL. Furthermore, poor sleepers have a higher prevalence of chronic disease severity, with sleep quality directly relating to disability.[22] In stable heart failure patients, sleep quality is related to the severity of heart failure, as classified by the New York Heart Association,[33] and the global PSQI has been shown to be an independent predictor of QOL.[20] We found that patients with worse disease-specific QOL as measured by the RSDI and the SNOT-22 correlated significantly with subjective sleep quality. However, in our cohort poor sleep quality was not found to be associated to disease severity as measured by endoscopy, CT score, or olfactory function. This is consistent with prior published work that has repeatedly demonstrated no association between CRS disease severity, CT score, or endoscopy scores. It is unknown if improving disease-specific QOL or disease severity can improve sleep quality. The relationship between disease severity, QOL and poor sleep is likely bidirectional; disability predicts worse sleep and worse sleep may be a predictor of QOL.

It can be hypothesized that nasal obstruction contributes to sleep dysfunction in CRS patients by impairing nasal airflow and promoting sleep disordered breathing. Prior studies have shown that nasal polyps are associated with nasal obstruction and sleep impairment with a 2-fold higher risk ratio of sleep disturbances.[1,34] As such, patients with CRS with nasal obstruction due to nasal polyposis had significant decrease in mean value of excessive daytime sleepiness, measured by Epworth Sleepiness Scale (ESS)[35] following surgery; however, no change was seen in the apnea hyponea index measured by polysomnogram. This suggests impaired nasal airflow may not be the sole determinant of sleep quality in patients with CRS. We similarly found no difference in sleep quality between patients with and without nasal polyposis. We did not however objectively measure nasal airflow, and thus it is not assured that patients with polyps had poorer airflow as compared to those without polyps.

There have been many plausible hypotheses concerning the pathophysiology of sleep in both health and disease. Our study demonstrates that sleep quality is diminished in patients with CRS, but the underlying etiology and pathophysiology of this sleep dysfunction is unknown. We posit that sleep function is regulated in the central nervous system (CNS) by highly interconnected neuronal groups that are characterized by altered input-output signaling as proposed by Krueger et al.[36] This is controlled via local signaling by growth factors and cytokines, which can influence neurons to adjust, and even change, the input-output properties of the neuronal groups, both in sleep centers and in the cortex of the CNS. Interestingly, CRS is a chronic inflammatory disease associated with changes in cytokines, their receptors, and downstream products. Cytokines up-regulated by infection or inflammation induce sickness behavior including but not limited to increased sleep, via signaling through the hypothalamic-pituitary-axis.[37] In humans, sleep loss and altered pro-inflammatory cytokine levels are associated but not limited to fatigue,[38,39] pain,[40] depression,[41] impaired cognition,[42] and memory.[43] There is evidence linking pro-inflammatory cytokines such as TNF-α and IL1-β in CRS and sleep regulation.[44] The mechanisms by which systemic inflammatory cytokines might signal the central nervous system in patients with CRS is unknown; although preliminary evidence suggests they may act in the brain to stimulate sleep through stimulation or alteration of afferent transmission, transport across the blood brain barrier, altering the level or activity of another substance that signals the brain, and/or direct passage across the blood-brain barrier.[45,46] The fact that cytokines act in the brain to induce physiological adaptations may begin to help explain the pathophysiology of CRS and commonly associated pathologies including depression, fatigue, impaired cognition, memory and sleep disturbance. Additional inquiries into the

associations between sleep and sleep regulatory substances, and how they signal the CNS in patients with CRS, should give us insight into the pathophysiology of sleep dysfunction.

The strengths of this study include its prospective, multi-institutional design, and utilization of a validated instrument to assess sleep dysfunction. However, patients were enrolled from tertiary rhinology practices and care should be taken fully extrapolating these findings to all patients with CRS. The PSQI has not been specifically validated for patients with CRS although it has been validated in mixed-age healthy controls, older men, patients with major depression, and sleep clinic patients.[3,47,48] The associations between the PSQI, the ESS, and objective measures are still unclear.[48,49] The PSQI does not always positively correlate to polysomnography-measured sleep[22,50] or daytime sleepiness as measured by ESS.[21] Similarly, the ESS does not always correlate to objective instruments of sleep dysfunction.[51]

CONCLUSION

Patients with CRS have a high prevalence of poor sleep quality as measured by the PSQI survey. Poor sleep quality is significantly associated with CRS-specific QOL, depression, and tobacco use, but does not correlate to disease severity as measured by endoscopy or CT staging. Sleep dysfunction should be considered in patients with CRS, along with the potential contributions from depression and tobacco. Future studies are needed to further elucidate both the etiology and pathophysiology of sleep dysfunction in patients with CRS.

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Table 1

Mean sub-domain and total scores of the Pittsburgh Sleep Quality Index (PSQI) across patient characteristics

	Sleep Quality	Sleep Latency	Sleep Duration	Sleep Efficiency	Sleep Disturbance	Sleep Medication	Daytime Dysfunction	PSQI Total	
Characteristics:	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value**
Gender									
Males	2.2 (1.0)	1.2 (1.0)	0.9 (1.0)	0.8 (1.0)	1.7 (0.7)	2.0 (1.1)	1.3 (0.8)	8.1 (4.1)	
Females	2.5 (0.9)	1.7 (1.0)	1.0 (1.0)	1.2 (1.1)	2.1 (0.7)	2.3 (1.0)	1.4 (0.8)	10.4 (4.4)	<0.001
Nasal polyposis									
With	2.2 (1.1)	1.4 (1.0)	1.1 (1.1)	0.9 (1.0)	1.9 (0.7)	2.1 (1.1)	1.2 (0.8)	8.9 (4.5)	
Without	2.5 (0.9)	1.5 (1.0)	0.9 (1.0)	1.1 (1.1)	1.9 (0.7)	2.2 (1.0)	1.4 (0.8)	9.6 (4.3)	0.179
Depression									
With	2.5 (0.9)	1.7 (1.0)	1.0 (1.1)	1.3 (1.2)	2.1 (0.7)	2.1 (1.1)	1.8 (0.8)	11.1 (4.2)	
Without	2.4 (1.0)	1.4 (1.1)	0.9 (1.0)	1.0 (1.1)	1.9 (0.7)	2.2 (1.0)	1.3 (0.8)	9.0 (4.4)	0.002
Asthma									
With	2.3 (1.0)	1.4 (1.0)	1.0 (1.0)	1.0 (1.1)	1.9 (0.7)	2.3 (1.0)	1.3 (0.9)	9.2 (4.9)	
Without	2.4 (0.9)	1.5 (1.1)	0.9 (1.0)	1.0 (1.1)	1.9 (0.7)	2.1 (1.1)	1.4 (0.8)	9.5 (4.2)	0.633
Allergy by history									
With	2.2 (1.0)	1.3 (1.0)	0.6 (0.9)	0.8 (1.0)	1.9 (0.6)	2.3 (0.9)	1.1 (0.6)	8.2 (4.2)	
Without	2.4 (1.0)	1.5 (1.0)	1.0 (1.0)	1.1 (1.1)	1.9 (0.7)	2.1 (1.1)	1.4 (0.9)	9.5 (4.5)	0.087
Allergy by testing									
With	2.5 (0.9)	1.4 (1.1)	1.0 (1.0)	1.1 (1.2)	1.9 (0.7)	2.2 (1.0)	1.4 (0.9)	9.6 (4.2)	
Without	2.4 (1.0)	1.5 (1.0)	0.9 (1.0)	1.0 (1.1)	1.9 (0.7)	2.2 (1.1)	1.3 (0.8)	9.3 (4.5)	0.656
ASA Intolerance									
With	2.4 (0.9)	1.5 (1.2)	1.1 (1.1)	1.2 (1.2)	2.0 (0.7)	2.6 (0.7)	1.4 (0.9)	9.9 (4.6)	
Without	2.4 (1.0)	1.5 (1.0)	0.9 (1.0)	1.0 (1.1)	1.9 (0.7)	2.1 (1.1)	1.3 (0.8)	9.3 (4.4)	0.584
Current smoker									
Yes	2.6 (0.9)	1.9 (0.9)	0.9 (1.1)	1.1 (0.9)	2.3 (0.7)	2.3 (1.1)	2.1 (0.7)	11.7 (3.9)	
No	2.4 (1.0)	1.5 (1.1)	1.0 (1.0)	1.0 (1.1)	1.9 (0.7)	2.2 (1.0)	1.3 (0.8)	9.2 (4.4)	0.030
Prior sinus surgery									

	Sleep Quality	Sleep Latency	Sleep Duration	Sleep Efficiency	Sleep Disturbance	Sleep Medication	Daytime Dysfunction	PSQI Total	
Characteristics:	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value**
Yes	2.4 (0.9)	1.5 (1.1)	0.9 (1.0)	1.0 (1.1)	2.0 (0.7)	2.2 (1.1)	1.4 (0.8)	9.5 (4.5)	
No	2.3 (1.0)	1.5 (1.0)	1.0 (1.0)	1.1 (1.1)	1.9 (0.6)	2.1 (1.0)	1.4 (0.9)	9.3 (4.3)	0.690
Cystic fibrosis									
With	2.6 (0.8)	2.0 (0.8)	0.6 (1.0)	1.0 (1.2)	2.0 (0.6)	2.0 (1.3)	1.7 (0.5)	9.7 (3.3)	
Without	2.4 (1.0)	1.5 (1.1)	1.0 (1.0)	1.0 (1.1)	1.9 (0.7)	2.2 (1.0)	1.3 (0.9)	9.4 (4.5)	0.834

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; ASA, acetylsalicylic acid intolerance. Allergy by patient history was self-reported. Allergy by testing was indicated if subjects underwent skin prick or mRAST testing. P-values are reported for differences in PSQI total score for each patient characteristic.

Table 2

Differences in patient characteristics between patients with and without poor sleep quality

	PSQI "Poor" Sleep Quality (> 5; n=201)		PSQI "Good" Sleep Quality (≤ 5; n=67)		
Patient Characteristics:	**Mean(SD)**	**n (%)**	**Mean(SD)**	**n (%)**	**p-value**
Age	48.7 (14.3) Range: [20–86]		50.7 (15.9) Range: [18–75]		0.345
Gender: Males		83 (41.3%)		38 (56.7%)	
Females		118 (58.7%)		29 (43.3%)	0.028
Nasal polyposis		67 (33.3%)		28 (41.8%)	0.210
Depression		44 (21.9%)		5 (7.5%)	0.008
Asthma		67 (33.3%)		27 (40.3%)	0.301
Allergy by history		23 (11.4%)		11 (16.4%)	0.289
Allergy by testing		52 (25.9%)		16 (23.9%)	0.746
ASA Intolerance		17 (8.5%)		5 (7.5%)	0.797
Current smoker		15 (7.5%)		2 (3.0%)	0.254
Prior sinus surgery		106 (52.7%)		32 (47.8%)	0.480
Cystic fibrosis		7 (3.5%)		0 (0.0)	0.269

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; ASA, acetylsalicylic acid intolerance.

Table 3

Mean differences in measures of CRS disease severity between patients with and without poor sleep quality

	PSQI "Poor" Sleep Quality (> 5; n=201)		PSQI "Good" Sleep Quality (≤ 5); n=67)			
Disease Severity Measures:	**Mean(SD)**	**Range [LL, UL]**	**Mean(SD)**	**Range [LL, UL]**	**t**	**p-value**
Lund-Mackay CT score	12.2 (5.7)	[1, 24]	12.8 (6.5)	[1, 24]	0.671	0.503
Lund-Kennedy Endoscopy score	6.4 (4.1)	[0, 20]	6.7 (3.8)	[0, 14]	0.498	0.619
B-SIT olfactory score	9.2 (3.1)	[1, 12]	8.5 (3.5)	[2, 12]	−1.313	0.190

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; LL, lower limit; UL, upper limit; CT, computed tomography; B-SIT, Brief Modified Smell Identification Test

Table 4

Mean disease-specific QOL measures and sleep quality

	PSQI "Poor" Sleep Quality (> 5; n=201)		PSQI "Good" Sleep Quality (≤ 5; n=67)			
Survey Measures:	**Mean(SD)**	**Range [LL, UL]**	**Mean(SD)**	**Range [LL, UL]**	**t**	**p-value**
RSDI physical	20.0 (8.9)	[1, 44]	11.9 (6.9)	[0, 28]	−7.658	<0.001
RSDI functional	16.4 (9.0)	[0, 36]	9.3 (7.5)	[0, 32]	−6.287	<0.001
RSDI emotional	14.6 (9.6)	[0, 40]	6.6 (6.9)	[0, 25]	−7.427	<0.001
RSDI total	50.9 (25.3)	[1, 116]	27.9 (19.0)	[1, 77]	−7.864	<0.001
SNOT-22 total	55.9 (19.0)	[4, 99]	35.8 (14.3)	[9, 69]	−9.114	<0.001

PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; LL, lower limit; UL, upper limit; RSDI, Rhinosinusitis Disability Index; SNOT-22, 22-item Sinonasal Outcome Test

Table 5

Spearman's correlation coefficients between clinical measures of disease severity and PSQI total and sub-domain scores

Measures:	Sleep Quality	Sleep Latency	Sleep Duration	Sleep Efficiency	Sleep Disturbance	Sleep Medication	Daytime Dysfunction	PSQI Total
RSDI physical	0.35*	0.44*	0.21**	0.30*	0.51*	0.17**	0.51*	0.56*
RSDI functional	0.27*	0.28*	0.13**	0.23*	0.44*	0.15**	0.61*	0.47*
RSDI emotional	0.30*	0.30*	0.16**	0.27*	0.42*	0.13**	0.63*	0.50*
RSDI total	0.33*	0.36*	0.18**	0.28*	0.50*	0.16**	0.62*	0.54*
SNOT-22	0.39*	0.43*	0.31*	0.38*	0.56*	0.17**	0.52*	0.63*
CT Score	0.03	−0.03	0.12	0.04	0.09	0.15**	−0.02	0.04
Endoscopy score	−0.01	−0.04	0.06	−0.07	0.09	0.02	−0.04	−0.04
B-SIT olfactory score	0.06	−0.03	0.03	0.04	−0.01	0.02	−0.03	0.02

* indicates p-value (2-tailed) significance less than 0.001.

** indicates p-value (2-tailed) less than 0.05.

PSQI, Pittsburgh Sleep Quality Index; RSDI, Rhinosinusitis Disability Index; SNOT-22, 22-item Sinonasal Outcome Test; CT, computed tomography; B-SIT, Brief Modified Smell Identification Test