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Treatment of Obstructive Sleep Apnea in Heart Failure Patients

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Introduction

A common sleep disorder known as obstructive sleep apnea (OSA) is when a person sleeps, their upper airway repeatedly closes completely or partially. About 13% of middle-aged men and 6% of middle-aged women are thought to have moderate to severe disease if they experience at least 15 apneas or hypopneas per hour of sleep. OSA is associated with a wide range of adverse health outcomes, including cardiovascular disease, diabetes, neurodegenerative disorders, hypertension, and mortality. Frequently cited as OSA symptoms are loud snoring, witnessed apneas, and nocturnal gasping. Other symptoms include excessive sleepiness, sleep disturbances, and general fatigue. Due to the fact that OSA etiology, symptoms, and outcomes vary greatly from patient to patient, atypical symptom profiles are under-recognized. Consequently, a number of recent studies have attempted to better define OSA patients' clinical subtypes in terms of demographics, disease severity, symptoms, and comorbidities. In order to tailor and improve clinical administration, a more precise depiction and determination of OSA is essential [1].

Description

The Icelandic Sleep Apnea Cohort (ISAC)'s moderate-to-severe OSA patients were the subject of the first major attempt to identify clinical presentation subtypes. The three distinct clusters that were found were as follows: a group with excessive sleepiness, a group with few symptoms, and a group with insomnia and restless sleep. Later research in Greece, Italy, France, and Europe suggested OSA clusters. These studies cannot be easily compared due to differences in sample characteristics (such as the inclusion of controls) and cluster-defining variables (such as the inclusion of the apnea hypopnea index, or AHI). However, several studies found clusters of patients with relatively low symptom burden, as well as clusters of patients with predominant symptoms of sleep disturbance or daytime sleepiness, in the original ISAC study. To better understand individual differences in clinical presentation, this article focuses on the clinical symptoms of patients with moderate-to-severe OSA [2].

Race and ethnicity play a role in OSA's etiology, according to recent research. Asian patients, for instance, are less likely to be obese and have more prevalent craniofacial risk factors when compared to Caucasians with similar disease severity. Similar to Caucasians, young African-Americans are found to have OSA with a higher severity than Caucasians. Due to these differences in disease risk factors and etiology, which may result in distinct symptom profiles and disease outcomes among ethnicities, the robustness and generalizability of OSA clusters previously identified within a single ethnic group are questioned. Also important is figuring out if the findings in Iceland are typical of OSA or unique to Iceland, possibly due to cultural norms or referral patterns. As a result of this knowledge, both our comprehension of OSA and clinicians' capacity to identify the most significant disease characteristics in particular patient populations would improve. The current study first attempted to confirm the existence of the three OSA clinical clusters that were initially identified by ISAC in a brand-new Icelandic cohort and a diverse international cohort of ethnic origin in order to accomplish this objective. Second, we attempted to determine the optimal number of clinical groups within the larger ethnically diverse partner outside of Iceland. We assumed that the ethnically diverse international cohort would exhibit similar OSA clusters and that the previously identified OSA clusters would be confirmed in Iceland [3].

The current international clinical sample of OSA patients was used to select the study's participants from the Sleep Apnea Global Interdisciplinary Consortium (SAGIC) study cohort (http://www.med.upenn.edu/sleepctr/sagic.html). The primary objective of SAGIC is to establish a large, multinational cohort with thorough phenotyping in order to comprehend common and ethnicity-specific OSA presentations and risk profiles. SAGIC participants were selected from the following eight sleep centers located in six nations: Germany (Charité University Hospital), Brazil (Médicado Instituto do Sono), Taiwan (Chang Gung Memorial Hospital), Iceland (Landspitali—The National University Hospital of Iceland), the United Kingdom (University of Pennsylvania and The Ohio State University), Australia (University of Sydney and Sir Charles Gairdner Hospital, Perth), and the United States (University of Pennsylvania and The Ohio State University). Members selected outside of Iceland were independently examined in light of the current review's objectives. The examination was restricted, like the first ISAC study, to participants with moderate-serious OSA (defined as AHI 15 times per hour) and self-detailed side effect recurrence data; The analyses did not include participants who did not respond to more than five of the symptom questions. Based on these criteria, 972 SAGIC participants, 757 from the remaining sites, and 215 from Iceland were included in the analyses. The study protocol was approved by the University of Pennsylvania's Institutional Review Board (IRB), and additional IRB approval was required for each site. Informed consent was given by all participants [4].

Sleep studies

688 (70.8%) of the patients in the analysis sample used for clustering (N = 972) underwent home-based sleep studies, while 284 (29.2%) underwent laboratory-based polysomnography. Standard operating procedures were implemented at each site to guarantee uniform data collection. For both in-laboratory and home studies, we observed significant inter-rater agreement among the eight sleep centers regarding the scoring of respiratory events. The American Academy of Sleep Medicine (AASM) manual's scoring criteria were used to evaluate sleep stages, arousals, and respiratory events. Hypopneas were defined as a 10% oxygen desaturation and a 30% decrease in airflow compared to baseline. The average number of apneas and hypopneas per hour of sleep was used to calculate the AHI. Apneas were defined as an absence of airflow on the oronasal thermistor or nasal pressure cannula for ten seconds. Total analysis time for home studies was calculated by using the sleep technologist's review of the study and patient questionnaires regarding sleep onset and wake times; the study did not include upright time. It was presumptuous to believe that this interval represented a full night's sleep. In the Icelandic sample, OSA was primarily diagnosed through in-home studies in accordance with standard clinical procedure [5].

This study is an essential next step in comprehending OSA clinical presentation clusters, building on the ISAC's initial descriptions of the clusters. Using an international sample of apneics recruited from sleep clinics in the SAGIC, we demonstrate the reproducibility of the disturbed sleep, minimally symptomatic, and excessively sleepy OSA clusters in an independent sample from Iceland and in a sample from outside of Iceland with a greater ethnic diversity. Despite the fact that the three clinical subtypes were generalizable, our findings suggested more specific OSA clinical clusters within the international SAGIC sample, enhancing our comprehension of OSA heterogeneity. The best solution, in particular, produced five OSA clusters, three of which were analogous to the ISAC-defined clusters of minimally symptomatic, excessively sleepy, and disturbed sleep. Two new subtypes were associated with a lack of other symptoms, and these clusters were referred to as disturbed sleep, minimal symptoms, and upper airway symptoms with sleepiness, respectively. The terms "upper airway symptoms dominant" and "sleepiness dominant" were used to describe these subtypes. In

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general, this study provides a novel approach for better describing patients with OSA presenting at rest facilities worldwide. This information can assist in the development of personalized medicine approaches to OSA treatment by enabling clinicians to focus interventions on the OSA symptoms and outcomes that are most relevant to each individual patient [2].

The fact that the clinical clusters defined by ISAC replicate in terms of symptom characteristics and prevalence in an independent Icelandic sample and generalize to other ethnicities provides strong evidence that these are genuine disease clusters among OSA patients. There were clear parallels to the ISAC solution, which had three clusters, despite the fact that the SAGIC samples from outside of Iceland had five OSA clusters—which we determined to be the optimal number from a statistical standpoint. With either solution, people were found with disturbed sleep, no traditional symptoms, and the typical OSA presentation of excessive sleepiness and upper airway symptoms; A significant number of patients remained similarly defined in both the three- and five-cluster solutions. Therefore, in clinical practice, using three or five clusters may provide comparable benefits. Clinically, the five OSA clusters appear to have additional advantages, such as increased precision regarding primary symptom complaints; the new clusters of sleepiness dominant and upper airway symptoms dominant included patients with a narrower symptom spectrum. If these primary symptoms could be recognized more quickly, then decisions regarding treatment could be made more effectively [5].

Conclusion

This study replicated and expanded on the clinical presentation clusters of OSA patients with disturbed sleep, minimal symptoms, and excessive sleepiness. To replicate the symptoms and prevalence of these clusters in the initial investigation, a separate Icelandic ancestor sample was used. In addition, it was demonstrated that a population of more ethnically diverse international sleep centers contained these clusters, albeit at a different prevalence. We extended the initial result to five clusters in this ethnically diverse sample and discovered two more specific OSA clusters with sleepiness dominant and upper airway symptoms dominant, in addition to clusters that were similar to the three clusters found in ISAC. In the end, our findings suggest that, regardless of the number of clusters, OSA patients with similar disease severity are distinguished by the absence of insomnia-related complaints, excessive sleepiness, and these symptoms. Despite the fact that these new clusters may help clinicians recognize primary symptoms more easily, this is the case. As suggested by differences in cluster prevalence and associations with ethnicity or demographics among the subtypes, symptom reporting and perceptions of OSA's consequences may differ across ethnic groups, just as they do with the etiology of the condition. Future research ought to focus on the targeted interventions implied by OSA clusters as well as the usefulness of incorporating biological or genetic factors when defining them. It is necessary to develop clinical tools that effectively group a new OSA patient who comes to the sleep clinic into the appropriate cluster. In the end, this information ought to make it simpler to implement personalized medicine strategies for OSA patients.

Acknowledgement

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Conflict of Interest

None.

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