

Clinical Subtypes of Obstructive Sleep Apnea are Correlated with Variations in the Prevalence of Cardiovascular and Metabolic Diseases

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Introduction

Obstructive sleep apnea (OSA) is a common sleep disorder in which the upper airway repeatedly closes completely or partially while a person is sleeping. At least 15 apneas or hypopneas per hour of sleep are considered to be indicative of moderate-severe disease in approximately 13% of middle-aged men and 6% of middle-aged women. OSA is linked to a wide range of negative health outcomes, including mortality, diabetes, neurodegenerative disorders, hypertension and cardiovascular disease, and more. Upper airway narrowing symptoms such as loud snoring, witnessed apneas, and nocturnal gasping, excessive sleepiness, sleep disturbances, and general fatigue are frequently cited as symptoms of OSA. Patients with atypical symptom profiles are under-recognized due to the fact that the etiology, symptoms, and outcomes of OSA vary greatly from patient to patient. As a result, a number of recent studies have attempted to better define the demographics, severity of the disease, symptoms, and comorbidities of OSA patients' clinical subtypes. More precise portrayal and determination of OSA is critical to customizing and working on clinical administration [1].

Description

Patients with moderate-to-severe OSA from the Icelandic Sleep Apnea Cohort (ISAC) were the subject of the first major attempt to identify clinical presentation subtypes. The following three distinct clusters were identified: an excessively sleepy group, a minimally symptomatic group, and a disturbed sleep group characterized by insomnia and restless sleep. OSA clusters have been suggested by subsequent research in Greece, Italy, France, and Europe. 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), these studies cannot be easily compared. However, the original ISAC study found clusters of patients with predominant sleep disturbance symptoms or daytime sleepiness, as well as clusters with relatively low symptom burden in several studies. This article focuses on the clinical symptoms of patients with moderate-to-severe OSA to better comprehend individual differences in clinical presentation [2].

According to recent research, OSA etiology varies by race and ethnicity. When compared to Caucasians with similar disease severity, Asian patients, for instance, are less likely to be obese and have more prevalent craniofacial risk factors. In a similar vein, compared to Caucasians, young African-

Americans are found to have OSA at a higher severity. The robustness and generalizability of OSA clusters previously identified within a single ethnic group is questioned because of these differences in disease risk factors and etiology, which may result in distinct symptom profiles and disease outcomes among ethnicities. In addition, it is essential to determine whether the findings in Iceland are characteristic of OSA or unique to Iceland, possibly as a result of cultural norms or referral patterns. Our understanding of OSA would grow as a result of this knowledge, as would clinicians' ability to identify the most significant disease characteristics in particular patient populations. In order to achieve this goal, the current study first attempted to verify the existence of the three OSA clinical clusters initially identified by ISAC in a new Icelandic cohort and a diverse international cohort of ethnic origin. Second, we tried to decide the ideal number of clinical bunches that exist inside the bigger ethnically different accomplice from beyond Iceland. We assumed that similar OSA clusters would be observed in the ethnically diverse international cohort and that the previously identified OSA clusters would be confirmed in Iceland [3].

Participants in the study were selected from the Sleep Apnea Global Interdisciplinary Consortium (SAGIC) study cohort (<http://www.med.upenn.edu/sleepctr/sagic.html>) for the current international clinical sample of OSA patients. In order to comprehend common and ethnicity-specific OSA presentations and risk profiles, the primary objective of SAGIC is to establish a large, multinational cohort with thorough phenotyping. Participants in SAGIC were selected from eight sleep centers in six countries, including the following: the United States (University of Pennsylvania and The Ohio State University), Australia (University of Sydney and Sir Charles Gairdner Hospital, Perth), Germany (Charité University Hospital), Brazil (Médicado Instituto do Sono), Taiwan (Chang Gung Memorial Hospital), Iceland (Landspítali—The National University Hospital of Iceland), and the United Kingdom (University of Pennsylvania and The Ohio State University). Given the objectives of the current review, members from Iceland were examined independently from members selected beyond Iceland. Like the first ISAC study, the examination test was confined to members with moderate-serious OSA [defined as AHI \geq 15 occasions for each hour] and data accessible on self-detailed side effect recurrence; Participants who didn't answer more than five of the symptom questions were left out of the analyses. 972 SAGIC participants, 757 from the remaining sites and 215 from Iceland, were included in the analyses based on these criteria. The University of Pennsylvania's Institutional Review Board (IRB) approved the study protocol, and additional IRB approval was required at each site. All participants gave their informed consent [4].

Sleep studies

In the analysis sample used for clustering (N=972), 688 (70.8 percent) patients were diagnosed with home-based sleep studies, while 284 (29.2 percent) patients were diagnosed with laboratory-based polysomnography. To ensure uniform data collection, each site implemented standard operating procedures. Within the eight sleep centers, we observed significant inter-rater agreement in the scoring of respiratory events for both in-laboratory and home studies. Sleep stages, arousals, and respiratory events were scored using criteria from the American Academy of Sleep Medicine (AASM) manual. Hypopneas were defined as a 30% reduction from baseline in airflow for 10s accompanied by at least a 4% oxygen desaturation. Apneas were defined as an absence of airflow on the oronasal thermistor or nasal pressure cannula for 10 s. The average number of apneas and hypopneas per hour of sleep was

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used to calculate the AHI. The sleep technologist's review of the study and patient questionnaires regarding sleep onset and wake times were used to calculate total analysis time for home studies; upright time was not included in the study. It was assumed that this interval represented the length of a night's sleep. In accordance with standard clinical procedure, OSA was diagnosed primarily through in-home studies in the Icelandic sample [5].

Discussion

Building on the ISAC's initial descriptions of OSA clinical presentation clusters, this study is an essential next step in the understanding of OSA clinical presentation clusters. We demonstrate the reproducibility of the disturbed sleep, minimally symptomatic, and excessively sleepy OSA clusters within an independent sample from Iceland and in a sample from outside of Iceland with a greater ethnic diversity using an international sample of apneics recruited from sleep clinics in the SAGIC. Our findings suggested more specific OSA clinical clusters within the international SAGIC sample, enhancing our comprehension of OSA heterogeneity despite the fact that the three clinical subtypes were generalizable. In particular, the best solution produced five OSA clusters, three of which were analogous to the ISAC-defined clusters of disturbed sleep, minimally symptomatic, and excessively sleepy. These clusters were referred to as disturbed sleep, minimal symptoms, and upper airway symptoms with sleepiness, respectively, and two new subtypes were associated with a lack of other symptoms. These subtypes were referred to as upper airway symptoms dominant and sleepiness dominant. By and large, this study gives an original way to deal with better describe patients with OSA introducing at rest facilities around the world. By enabling clinicians to focus interventions on the OSA symptoms and consequences that are most relevant to each individual patient, this information can assist in the development of personalized medicine approaches to OSA treatment [2].

Strong evidence that these are genuine disease clusters among OSA patients is provided by the observation 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. Even though the SAGIC samples from outside of Iceland had five OSA clusters, which we found to be the optimal number from a statistical standpoint, there were clear parallels with the ISAC solution with three clusters. People with disturbed sleep, no traditional symptoms, and the typical OSA presentation of excessive sleepiness and upper airway symptoms were found with either solution; In both the three- and five-cluster solutions, a significant portion of patients remained similarly defined. As a result, employing three or five clusters may offer comparable advantages in clinical practice. The five OSA clusters appear to have additional clinical benefits, including improved accuracy regarding primary symptom complaints; Patients with a narrower symptom spectrum were included in the new clusters of sleepiness dominant and upper airway symptoms dominant. The efficiency of both diagnosis and treatment decisions could be enhanced if these primary symptoms could be recognized more quickly [5].

Conclusion

The clinical presentation clusters of OSA patients with disturbed sleep, minimally symptomatic, and excessively sleepy were replicated and expanded upon in this study. An independent sample of Icelandic ancestry was used

to replicate the symptoms and prevalence of these clusters in the original investigation. In addition, it was demonstrated that these clusters were present, albeit at a different prevalence, in a population of international sleep centers that was more ethnically diverse. In addition to clusters that were similar to the three that were found in ISAC, 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. Our findings ultimately suggest that, regardless of the number of clusters, insomnia-related complaints, excessive sleepiness, and a lack of these symptoms are important characteristics that distinguish OSA patients with similar disease severity. This is despite the fact that these new clusters may provide some added clinical benefit by recognizing primary symptoms more easily. Similar to the etiology of OSA, symptom reporting and perceptions of OSA's consequences may differ across ethnic groups, as suggested by differences in cluster prevalence and associations with ethnicity or demographics among the subtypes. The usefulness of incorporating biological or genetic factors when defining OSA clusters should be the subject of future research, as should the targeted interventions implied by these clusters. Clinical tools that effectively classify a new OSA patient who presents to the sleep clinic into the appropriate cluster must be developed. In the end, this knowledge ought to make personalized medicine approaches for OSA patients easier to implement.

Acknowledgement

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Conflict of Interest

None.

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