



Insomnia features and patient-reported daytime sleepiness in patients with obstructive sleep apnea

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ABSTRACT

Previous preliminary evidence suggests insomnia features playing a major causative or confounding role in daytime sleepiness in obstructive sleep apnea (OSA) patients. We investigated further this hypothesis in a larger OSA patient cohort. In a cross-sectional study in a tertiary medical center, consecutive patients presenting with suspected OSA, but without other sleepiness-promoting comorbidities, and tested by in-lab polysomnography (PSG) were evaluated prospectively for excessive daytime sleepiness (EDS) using the Epworth Sleepiness Scale (ESS) and for insomnia using the Insomnia Severity Index (ISI) respectively. Two hundred and thirty patients (63 female; average age: 54.1 y) were included in this OSA treatment-naïve cohort. ISI values correlated best (Spearman's $\rho = 0.29$, $p < .001$) with the total ESS score than any PSG-associated metric did. Especially ISI item 7- (interference of sleep problems with daily functions, $r = 0.33$, $p < .001$) and item 2- (difficulty staying asleep, $r = 0.28$, $p < .001$) and to a lesser degree item 4- (satisfaction with own current sleep patterns, $r = 0.23$, $p = 0.025$) scores showed significant correlations. Notably, no single significant correlation was found between ESS score and any PSG-metric at all. In a multiple regression analysis, the ISI item 7 score emerged as the sole significant independent predictor of the ESS score. We conclude that insomnia may significantly impact patient-reported daytime sleepiness in OSA patients. We suggest that assessment of insomnia symptoms and features (e.g. by means of ISI) should always be performed in OSA patients reporting daytime sleepiness. We propose that these preliminary findings should be validated in larger and diverse cohorts of OSA patients.

1. Introduction

Reported excessive daytime sleepiness (EDS) is quite prevalent among patients suffering from obstructive sleep apnea (OSA) [1] and EDS is a very common precipitating factor for seeking initial treatment for OSA [2]. EDS was identified among 31.0 % of participants and 51.7 % reported sleepiness as a precipitating factor for seeking initial OSA treatment in a real-world cohort of patients with OSA [2]. When sleepiness was defined as an Epworth Sleepiness Scale (ESS) score >10 or a report of at least frequently feeling unrested or sleepy, 46 % percent of participants in a cohort of 1149 participants with moderate to severe OSA reported sleepiness [3]. The prevalence of EDS has been reportedly increasing with increasing severity of OSA: in a cohort of 1115 patients suspected of having OSA, EDS was prevalent in 40.5 % of patients without OSA (apnea-hypopnea index (AHI) < 5), in 46.5 % of patients with mild OSA, in 52.0 % of patients with moderate (AHI 15–29.9/h), and in 58.0 % of patients with severe OSA (AHI 30/h or above) [4]. Daytime sleepiness may be evaluated by means of patient-reported outcomes, such as the Epworth sleepiness scale (ESS) [5], or by much more sophisticated means such as in-lab multiple sleep latency test

(MSLT), among others.

Due to its simplicity in both implementation and interpretation and its favourable validation results, ESS became over the years the principal instrument to evaluate EDS in the clinical sleep medicine setting. Because of the quite significant impact of EDS both from an individual (alertness, concentration, participation in social activities, etc.) as well as from a societal (e.g. vehicle or other machine use) viewpoint, ESS attained a pivotal role in OSA trials, with ESS score being quite often the primary or major outcome measure and/or treatment target in OSA-related trials [6,7]. In a meta-analysis, continuous positive airway pressure (CPAP) therapy has been found to elicit small improvements in subjective sleepiness and objective wakefulness in people with mild to moderate OSAS [8]. This fact raises questions about the association between sleepiness and respiratory disturbance in OSA patients, especially those of mild or moderate severity.

There has been evidence that insomnia-related features may impact on EDS [2] and more specifically on subjective, patient-reported EDS, as depicted by the ESS metric. In addition, in OSA patients who did not tolerate positive airway pressure (PAP) therapy and were treated with hypoglossal nerve stimulation therapy, ISI (Insomnia Severity Index)

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scores correlated quite strongly with ESS scores both preoperatively (Spearman's $\rho = 0.46$) and postoperatively ($\rho = 0.79$) [9]. Also, a dissociation between ESS and standard polysomnography (PSG)-metrics has been observed, namely no correlations between ESS and AHI (apnea/hypopnea index), minimal SpO₂ and ODI (oxygen desaturation index) was proven and solely a weak positive correlation between arousal index and ESS was found in OSA patients [10,11].

As many as 39 %–58 % of OSA patients may report insomnia symptoms, and 29 %–67 % of insomnia patients may fulfil criteria for a diagnosis of OSA [12–14]. More specifically, in studies in which the Insomnia Severity Index (ISI) questionnaire has been applied to measure insomnia in patients with OSA as captured by polysomnography (PSG) a prevalence of 32 %–84 % has been reported [11,13].

Previous studies investigating the associations between insomnia symptoms and EDS in patients with OSA have found that the composite ESS score was significantly higher in patients with COMISA than in patients with OSA without any comorbid insomnia [15]. Also, a significant correlation between the composite ISI score and the composite ESS score has been previously reported in a cohort of OSA patients, irrespective of the existence of comorbid insomnia or not [15].

Further, there are pathophysiological associations between sleepiness and insomnia in people with OSA. For example, sleepiness may be one of the daytime symptoms of either insomnia and/or OSA, resulting in a positive association between insomnia symptoms and sleepiness. Alternatively, insomnia (sleeplessness) and EDS (sleepiness) may also be viewed as occurring at opposite ends of a 'sleepiness' continuum. Many people with insomnia do not report any symptoms of sleepiness, but rather an inability to fall asleep characterised by feelings of chronic alertness/psychological arousal and fatigue during the day. This could theoretically contribute to a negative association between insomnia and sleepiness in people with OSA.

Based on the above findings, on the aforementioned theoretical pathophysiological associations between sleepiness and insomnia in patients with OSA and on our clinical experience with OSA patients, especially those neither adhering to nor tolerating OSA-therapies of any kind who are commonly presenting with various comorbid insomnia traits, we investigated further the hypothesis that insomnia features may be associated with EDS in an OSA-treatment naïve patient cohort in a clinical sleep medicine setting.

More specifically, we aimed by means of a more granular approach at identifying specific insomnia symptoms or subtypes that could possibly relate to EDS and be predictive of the ESS score in OSA patients.

2. Methods

In a cross-sectional study in a tertiary medical center, consecutive patients presenting with suspected OSA, but without other sleepiness-promoting comorbidities, and tested by in-lab polysomnography (PSG) were evaluated prospectively for excessive daytime sleepiness (EDS) using the Epworth Sleepiness Scale (ESS) and for insomnia using the Insomnia Severity Index (ISI) respectively.

The aforementioned consecutive patients with suspected OSA were assessed by in-lab PSG in the sleep center of a tertiary medical center between July 2018 and February 2021. Only patients with suspected OSA who were OSA-treatment naïve and had filled in both the ESS and the ISI questionnaires as part of the clinical routine assessment were included. Patients already on psychotropic or other major central nervous system acting medication, or who had any history of psychiatric illness or any history of psychosomatic/behavioural therapy, clinical heart failure (NYHA II – IV), significant (GOLD III or IV) COPD were excluded. Other causes of EDS such as sleep deprivation, narcolepsy or hypersomnias were excluded on the basis of medical history. Data of participants with a clinical suspicion of any of the three above conditions were not included for analysis. Polysomnography was performed and scored according to AASM standards by experienced physicians in a sleep-accredited lab. All patients with suspected OSA were asked if their

standard routine assessment data could be used for research purposes. Written consent was provided after the participants were informed about the relevant study procedures and data protection policies. Local ethics/IRB approval was granted (Nr. T857150). All study procedures were in accordance with the principles of the Helsinki Declaration for studies in humans.

2.1. Statistical methods

Spearman's correlations coefficients (with respective p-values) have been computed for correlations between Epworth Sleepiness Scale total score and ISI scores (total and ISI item-specific) as well as PSG-based metrics (for details see Table 1). To deal with multiple comparisons, we applied the Bonferroni-Holm correction. In addition, after log-normalization of the resulting statistically significant independent parameters (as presented on Table 1) a multiple regression analysis was conducted to predict ESS as dependent variable from the significant ISI parameters as well as from age and sex of the participants. In addition, with the aim of a further sensitivity analysis, focused on AHI as an input, we did a subgroup Spearman' correlation analysis of ESS values with participants with AHI ≤ 15 /h and participants with AHI > 15 /h (Tables 2 and 3).

3. Results

Two hundred and thirty patients who presented with suspected OSA (63 female; average age: 54,1 y) were included in this cross-sectional treatment-naïve cohort. Their mean AHI (apnea/hypopnea index) was 35,6/h (range: 2/h – 109,7/h). Six participants had an AHI ≤ 5 events/h. Forty one participants had an AHI ≤ 15 events/h. Regarding insomnia, the prevalence of ISI scores ≥ 8 (mild to severe insomnia) in this cohort was 80 %. The prevalence of ISI scores ≥ 15 (moderate to severe insomnia) in this cohort was 47 %. Using a cutoff of AHI = 15/h for

Table 1

Correlation analysis between total Epworth Sleepiness Scale score and various variables (including PSG-metric values as well as ISI-item scores) in the entire patient cohort. Abbreviations: AHI, apnea/hypopnea index; AI, apnea index; Ari, arousal index; ESS, Epworth Sleepiness Scale; HI, hypopnea index; ISI, Insomnia Severity Index; ODI, oxygen desaturation index; SpO₂, arterial oxygen saturation (in %); PLMI, periodic leg movement index; PSG, polysomnography; SE, sleep efficiency; SWS, slow wave sleep; TST, total sleep time; t90, percentage of total sleep time with arterial oxygen saturation below 90 %. BH p-value refers to the p-values after the Bonferroni-Holm correction for multiple comparisons.

ESS score	with variable	Spearman ρ (rho)	probability > ρ	BH p-Value
	Age	–0,09	0,16	1,76
	Subjective sleep duration [h]	–0,15	0,03	0,45
	ISI – total (composite) score	0,29	p < .001	p < .001
	ISI – item 1 score	0,07	0,32	1,62
	ISI – item 2 score	0,28	p < .001	p < .001
	ISI – item 3 score	0,20	0,005	0,09
	ISI – item 4 score	0,23	0,001	.025
	ISI – item 5 score	0,20	0,004	0,07
	ISI – item 6 score	0,19	0,008	0,13
	ISI – item 7 score	0,33	p < .001	p < .001
	PSG – AI (events/h)	0,06	0,35	1,41
	PSG – HI (events/h)	0,01	0,83	1,66
	PSG – ODI (events/h)	0,12	0,07	0,81
	PSG – average SpO ₂	–0,01	0,91	0,90
	PSG – t90 (%)	0,07	0,29	1,71
	PSG – PLMI (events/h)	0,13	0,05	0,69
	PSG – AHI (events/h)	0,08	0,24	2,42
	PSG – TST	0,04	0,59	1,79
	PSG – SE (%)	0,07	0,27	1,90
	PSG – SWS (%)	0,07	0,26	2,08
	PSG – REM (%)	0,08	0,25	2,25
	PSG – Ari	0,13	0,05	0,74

Table 2
Spearman’s correlation analysis between total Epworth Sleepiness Scale score and various variables in the subgroup of our patient cohort with AHI ≤15 events/h. Abbreviations as on Table 1.

ESS score	with variable	Spearman ρ (rho)	probability > ρ	BH p-Value
	Age	−0,38	0,011	0,254
	Subjective sleep duration [h]	−0,10	0,68	3,43
	ISI - total score	0,23	0,23	4,03
	ISI - item 1 score	−0,07	0,51	4,60
	ISI - item 2 score	0,20	0,23	3,81
	ISI - item 3 score	0,10	0,48	4,89
	ISI - item 4 score	0,23	0,16	2,88
	ISI - item 5 score	0,31	0,048	2,02
	ISI - item 6 score	0,17	0,34	4,09
	ISI - item 7 score	0,19	0,35	3,85
	PSG -AI (events/h)	−0,02	0,86	1,72
	PSG -HI (events/h)	0,08	0,59	4,76
	PSG -ODI (events/h)	0,08	0,59	3,59
	PSG - average SpO2	0,17	0,26	4,04
	PSG - t90 (%)	0,06	0,69	2,76
	PSG - PLMI (events/h)	−0,08	0,59	4,17
	PSG-AHI	0,02	0,89	0,89
	PSG - TST	0,16	0,29	4,09
	PSG - SE (%)	0,15	0,33	4,39
	PSG - SWS (%)	−0,24	0,13	2,47
	PSG - REM (%)	0,04	0,76	2,30
	PSG - ARI	0,24	0,13	2,60

Table 3
Spearman’s correlation analysis between total Epworth Sleepiness Scale score and various variables in the subgroup with AHI >15 events/h. Abbreviation list as on Table 1.

ESS score	with variable	Spearman ρ (rho)	probability > ρ	BH p-value
	Age	−0,03	0,65	3,94
	Subjective sleep duration [h]	−0,15	0,03	0,53
	ISI - total (composite) score	0,31	<.001	0,001
	ISI - item 1 score	0,11	0,15	2,05
	ISI - item 2 score	0,29	<.001	0,002
	ISI - item 3 score	0,21	0,006	0,12
	ISI - item 4 score	0,22	0,003	0,08
	ISI - item 5 score	0,18	0,021	0,36
	ISI - item 6 score	0,19	0,014	0,26
	ISI - item 7 score	0,37	<.001	<.001
	PSG -AI (events/h)	0,05	0,43	3,45
	PSG -HI (events/h)	−0,01	0,91	3,66
	PSG -ODI (events/h)	0,11	0,14	1,98
	PSG - average SpO2	−0,02	0,74	3,71
	PSG - t90 (%)	0,06	0,41	3,72
	PSG - PLMI (events/h)	0,17	0,01	0,32
	PSG-AHI (events/h)	0,06	0,37	3,75
	PSG - TST	−0,01	0,93	2,81
	PSG - SE (%)	0,05	0,46	3,24
	PSG - SWS (%)	0,15	0,03	0,53
	PSG - REM (%)	0,09	0,20	2,29
	PSG - ARI	0,10	0,15	1,91

clinically significant (moderate – severe) OSA and a cutoff of ISI composite score = 15 points for clinically significant (moderate – severe) insomnia, it turned out that in this study’s cohort the prevalence of OSA only was 44 %, of insomnia only was 10 % and of COMISA was 38 %. In 8 % of the cohort neither OSA nor insomnia was present. The total (or composite) ISI score, item 2, item 4 and item 7 scores were best correlated with the ESS score. Item 3, 5 and 6 scores of the ISI showed significant, although less strong correlations with the ESS score in our patient cohort. On the contrary, standard PSG metrics did not show any meaningful correlation with ESS at all (Table 1).

3.1. Results of the subgroup analyses

In the subgroup of patients with mild OSA or no OSA at all (namely, with AHI ≤15) no significant correlation between the ESS score and any of the tested variables was found after Bonferroni-Holm correction (Table 2).

In the subgroup of patients with moderate and severe OSA (namely, participants with AHI >15) significant correlation between the total (composite) ESS score and some of the ISI item scores, including the total/composite ISI score were found, even after Bonferroni-Holm correction (Table 3).

3.2. Results of the multiple regression analysis

In order to perform a sensitivity analysis, we performed a multiple regression analysis, as described in the Methods. The model explained a significant portion of the variance in ESS, $R^2 = 0.13$, $F(6,193) = 4.91$, $p < .001$. The adjusted R^2 , which accounts for the number of predictors, was 0.11. Given the large number of tests conducted in the regression model, we applied a False Discovery Rate (FDR) correction using the Benjamini-Hochberg procedure to control for type I errors. Only the ISI 7 item score emerged as a significant predictor of the ESS score, with an adjusted p-value of $p = .002$.

4. Discussion

We provide preliminary evidence for a rather strong association of insomnia with patient-reported EDS scores in patients with suspected OSA. Although traditionally daytime sleepiness in OSA patients has been associated with the degree of respiratory disturbance, the evidence provided by the present report supports the hypothesis that insomnia-related features have a much stronger impact than PSG-metrics have on the patient-reported EDS, as captured by ESS, in OSA patients.

From a clinical viewpoint, these findings make sense. Insomnia with its associated sleep deficits and sleep pattern disruption has great potential to cause daytime sleepiness. Although comorbid insomnia and sleep apnea (COMISA) is quite prevalent, and therapy of this comorbid condition is quite challenging [16], the impact of insomnia specifically on EDS has been less investigated in a dedicated fashion in OSA patients.

Especially ISI items 2 and even more 7 appear to be quite strongly associated with ESS scores. Item 2 (i.e. difficulty staying asleep) may indeed be influenced by apneas and hypopneas in OSA patients causing arousals and sleep interruption. Nonetheless, the reasons causing awakenings lasting for many minutes (or hours) during the night may be closely related to (non-respiratory) psychological stressors.

Further, the very close association between ISI item 7 and ESS scores suggests that individuals with EDS have an increased self-awareness (or may even be significantly preoccupied) with their own sleep health issues. This fact may be due to increased psychological stress as perceived under the current patient-reported conditions.

Regarding a possible age effect, older veterans with OSA report lower ISI scores [17]. Our targeted statistical analysis showed no significant age effect on the observed ESS scores in the pooled results of the entire cohort (Table 1). Nonetheless, after performing a separate Spearman’s correlation analysis in the subgroup of people with mild OSA or no OSA at all (i.e. AHI ≤15/h) we found initially a significant correlation between age and ESS score, which lost significance after the Bonferroni-Holm correction (Table 2). In addition, in the multiple regression analysis age had no predictive value for ESS score prediction neither. Therefore, age may not be considered a predictor of the ESS score. Further, we computed a separate Spearman’s correlation of the relationship between age and each single ISI-item score in our cohort. A weak negative ($r = -0.226$), but significant ($p = .007$, after Bonferroni-Holm correction) correlation between age and solely ISI-item 5 (“How noticeable to others do you think your sleep problem is in terms of impairing the quality of your life?”) has been found. No other

ISI-item showed any significant correlation with age.

Strong evidence for a dose-response relationship between PAP-adherence use and reduction of EDS has been previously reported [6, 7]. A more favourable PAP-adherence and/or EDS reduction could have been due to less severe insomnia-related traits in these OSA patient cohorts.

Some authors report higher levels of daytime sleepiness in patients with COMISA than in patients with OSA alone [18]. Nonetheless, other authors have reported more EDS in patients with OSA without insomnia than in patients with COMISA [19].

4.1. Limitations

There are some limitations to this study's methodology that require attention and should be considered in future studies. A control group is missing. Also, the cross-sectional design of this study does not allow any causal associations. Regarding limitations to the interpretation of the study results, it should be mentioned that there is a well-documented variability in ESS responses over time, and there is a reported variability in the associations between subjective (ESS) and objective (Multiple Sleep Latency Test) measures of daytime sleepiness. These issues may influence the validity of using ESS data to infer any relationship between insomnia and objective functioning/alertness. Also, like many studies in sleep medicine, our study was mostly composed of male participants ($n = 168$; 73 % of our cohort) and due to a relative lack of data in females we did not consider applying sex-specific subgroup analysis. This is a study limitation and we have treated this by means of including sex in our multiple regression model. Further studies with higher sample size need to be conducted to study sex-specific differences of OSA and ISI relationship. Nonetheless, we have adjusted the ESS predictive model for sex in our multiple regression analysis; it turned out that sex was not any significant independent predictor of the ESS score.

Although the ISI questionnaire has been developed and validated for use in insomnia patient management and clinical research [20], the quite high prevalence of clinical comorbid insomnia among OSA patients, impacting both OSA diagnosis and treatment adherence [21], may be a reasonable argument to further evaluate use of ISI and consider insomnia features and possibly even subclinical insomnia in a more targeted and systematic fashion in OSA patient cohorts in the future.

5. Conclusions

Therefore, insomnia-related symptoms, especially those related with item 7 of the ISI questionnaire, emerge as potential key predictors of the patient-reported Epworth Sleepiness Scale score in treatment-naïve OSA patients. Of note, PSG-based metrics did not show any significant correlation to ESS scores in this cohort of patients with suspected OSA. In addition, we suggest that assessing insomnia symptoms related to any difficulty falling asleep (item 2 of the ISI questionnaire), the subjective overall satisfaction of the patients with their current sleep pattern (item 4) and the total ISI score could provide clinical added value in the diagnostic evaluation of OSA patients or patients with a suspicion of OSA who report daytime sleepiness. We propose that these preliminary findings should be validated in larger and diverse cohorts of OSA patients.

CRedit authorship contribution statement

Gouveris H.: Writing – original draft, Validation, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Deiß A.:** Writing – review & editing, Investigation, Data curation. **Hackenberg B.:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Bahr-Hamm K.:** Writing – review & editing, Investigation, Formal analysis, Data curation. **Huppertz T.:** Writing – review & editing, Validation, Investigation, Data curation. **Ludwig K.:** Writing – review & editing, Validation, Formal

analysis. **Matthias C.:** Writing – review & editing, Validation, Supervision. **Simon P.:** Writing – review & editing, Validation, Software, Methodology, Formal analysis, Data curation.

Data availability statement

The raw data used for generation of the results presented in this manuscript will be available after reasonable request.

Ethics approval statement

Local ethics/IRB approval was granted (Nr. T857150/Ethics Committee of the State Medical Association of Rheinland – Palatinate, Germany).

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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