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Comparison of obstructive sleep apnoea prevalence and severity across WHO pulmonary hypertension groups

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ABSTRACT

Introduction Pulmonary hypertension is classified into five groups in the WHO classification system. Patients with pulmonary hypertension often have comorbid obstructive sleep apnoea (OSA), yet the prevalence and severity of OSA in each of the WHO pulmonary hypertension groups have not been well established.

Methods To compare the prevalence and severity of OSA between WHO pulmonary hypertension groups, we performed a retrospective cohort study, including patients who had polysomnography or a home sleep study and confirmed pulmonary hypertension on right heart catheterisation. The primary outcomes of OSA prevalence and severity were measured by median apnoea hypopnea index (AHI) or respiratory event index (REI) and were compared by WHO pulmonary hypertension group. Multivariable negative binomial regression was used to evaluate the association between the outcome of OSA severity by AHI or REI and WHO group.

Results Among the cohort of 132 patients, OSA was common in all WHO pulmonary hypertension groups but was most common and most severe in WHO group II pulmonary hypertension. Median AHI or REI in WHO group II was 12.0 events/hour compared with 2.8 in group I, 3.7 in group III, 10.0 in group IV and 6.4 in group V. Multivariable negative binomial regression showed about a twofold increase in AHI or REI in WHO group II compared with WHO group I pulmonary hypertension.

Discussion Our findings demonstrate that OSA deserves greater consideration as a treatable comorbidity that may affect pulmonary haemodynamics and quality of life in patients with pulmonary hypertension across all WHO groups.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ There is a relationship between obstructive sleep apnoea (OSA) and pulmonary hypertension, but the prevalence and severity of OSA in patients with pulmonary hypertension are not well established.

WHAT THIS STUDY ADDS

⇒ We compared the prevalence and severity of OSA in 132 patients with haemodynamically confirmed pulmonary hypertension across the WHO pulmonary hypertension classification system. OSA was common in all WHO pulmonary hypertension groups but may be most common and most severe in patients with WHO group II pulmonary hypertension.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study emphasises that clinicians should screen for OSA in all patients with pulmonary hypertension as OSA is a treatable condition that may affect quality of life in this population.

INTRODUCTION

The relationship between obstructive sleep apnoea (OSA) and pulmonary hypertension is still being explored, but it is generally believed that overnight hypoxemia and repetitive large intrathoracic negative pressure swings attributable to upper airway obstruction may contribute adversely to pulmonary haemodynamics in patients with OSA.[1 2] Previous studies examining OSA in patients with pulmonary hypertension are limited, and estimates of prevalence in patients with pulmonary hypertension range widely.[3–8] While a previous prospective study of pulmonary haemodynamics on right heart catheterisation in 220 consective patients with OSA showed that OSA was only associated with mild elevation of mean pulmonary artery pressure,[9] the effect of OSA overlap on patients with pre-existing pulmonary hypertension from other aetiologies may be more consequential. OSA may overlap with other causes of pulmonary hypertension, such as left heart failure, chronic obstructive pulmonary disease or chronic thromboembolic disease; and the clinical impact of comorbid OSA on pulmonary haemodynamics may depend on the underlying aetiology of pulmonary hypertension.

Pulmonary hypertension is classified into five groups in the WHO classification system, the main clinical framework used to treat patients with pulmonary hypertension.[10] In this investigation, we compare the prevalence and severity of OSA by WHO pulmonary

hypertension group. We tested the hypothesis that OSA would be most prevalent and most severe among patients with WHO Group II pulmonary hypertension, as we expected a high burden of OSA in patients with cardiac disease and associated metabolic syndrome.

METHODS

Study design, setting and population

To compare the prevalence and severity of OSA across WHO pulmonary hypertension groups, we performed a retrospective cohort study of patients treated for pulmonary hypertension at a tertiary referral centre in New York from January 2000 to August 2019. During this period, patients with suspected pulmonary hypertension were consistently referred for a sleep study, either in-lab polysomnography or a type III home sleep study, if they reported symptoms of sleep apnoea (daytime somnolence, fatigue, snoring or frequent nocturnal awakenings) or if they had a Mallampati score of 3 or 4 on physical examination. Patients were identified for this study if they underwent a sleep study, either in-lab polysomnography or a type III home sleep study, followed by right heart catheterisation within 24 months. Among patients identified, all patients with a confirmed diagnosis of pulmonary hypertension based on mean pulmonary artery pressure >20 mm Hg on right heart catheterisation were included. Patients' WHO pulmonary hypertension groups were determined based on guidelines from the sixth World Symposium on Pulmonary Hypertension.[10] Apnoeas were defined by a 90% reduction in nasal flow for at least 10 s. Hypopneas were defined by a 4% desaturation with at least a 30% decrease in nasal flow for at least 10 s. The apnoea-hypopnea index (AHI) was defined as the number of apnoeas or hypopneas per hour of sleep on polysomnography. The respiratory event index (REI) was defined as the number of apnoeas or hypopneas per hour of recording time on type III home sleep study.

Patient involvement

Given the retrospective design of this study, patients were not involved in the design or recruitment process. However, the study was prompted by the investigators' observation that OSA was common in patients with pulmonary hypertension. We embarked on this study in an effort to work towards improving quality of life in patients with pulmonary hypertension. Once this study is published, patients following in our pulmonary hypertension practice will be informed of the results through the distribution of an information brochure highlighting the importance of sleep evaluation.

Data collection

Data collected included demographic factors, comorbid medical conditions and data from right heart catheterisation (mean pulmonary artery pressure, pulmonary artery occlusion pressure and pulmonary vascular resistance), pulmonary function testing, polysomnography and home sleep studies. The mean pulmonary artery pressure, pulmonary artery occlusion pressure (pulmonary artery wedge pressure) and pulmonary vascular resistance were recorded from right heart catheterisation reports. The AHI or REI, depending on whether the sleep study was a polysomnogram or a home sleep study, was recorded from sleep study reports. Data were collected retrospectively from the electronic medical record and stored in a secure database. Approval for the study was obtained from the institutional review board of Northwell Health, which waived the need to obtain informed consent from subjects. Research was conducted in accordance with the Declaration of Helsinki.

Statistical analysis

Data were analysed for the entire cohort, including patients who underwent either polysomnography or home sleep study, and then separately for the subset of patients who had polysomnography. Demographic and clinical characteristics for the entire study cohort and for the subset of patients who had polysomnography were compared across the five WHO pulmonary hypertension groups using X^2 tests for categorical variables and Kruskall-Wallis tests for continuous variables. The primary outcomes of OSA prevalence and OSA severity, the latter measured by median AHI or REI (AHI-REI) in the complete cohort and AHI in the subset of patient who had polysomnography, were compared by WHO pulmonary hypertension group using X^2 and Kruskall-Wallis tests. $P<0.05$ was considered statistically significant. OSA was defined using the threshold of AHI-REI ≥5 events/hour and was further stratified by severity into mild (5 ≥AHI or REI <15 events/hour), moderate (15 ≥AHI or REI <30 events/hour) and severe OSA (AHI or REI ≥30 events/hour). Separate multivariable negative binomial regressions, one for the entire cohort and one for the subset of patients who underwent polysomnography, were then performed to model the association between the outcome of AHI-REI (in the complete cohort) or AHI (in the subset who underwent polysomnography) and the independent variable of WHO pulmonary hypertensions group, controlling for body mass index (BMI), age, gender and in-lab or home sleep study type. Negative binomal models were used to model the association between WHO pulmonary group and AHI-REI because of the data's skew. Statistical analysis was performed using STATA statistical software.

RESULTS

The study cohort included 132 patients with pulmonary hypertension confirmed on right heart catheterisation who underwent sleep study with either polysomnography or home sleep study. There were 49 patients with WHO group I pulmonary hypertension, 53 patients with WHO group II pulmonary hypertension, 13 patients with WHO group III pulmonary hypertension, 13 patients with

Table 1 Clinical characteristics of the complete cohort*

	WHO group I	WHO group II	WHO group III	WHO group IV	WHO group V	P†
Number of patients (n=132)	49	53	13	13	4	–
Demographics						
Age, years	56 (42–65)	72 (60–76)	68 (56–73)	55 (47–61)	57 (44–69)	**<0.01**
Male gender	11 (22%)	12 (23%)	7 (54%)	5 (38%)	3 (75%)	**0.03**
Body mass index, kg/m^2	30 (26–33)	33 (27–39)	27 (26–32)	31 (29–31)	26 (24–29)	**0.04**
Chronic medical conditions						
Connective tissue disease	28 (57%)	5 (9%)	4 (31%)	1 (8%)	0 (0%)	**<0.01**
COPD	6 (12%)	3 (6%)	5 (38%)	0 (0%)	1 (25%)	**0.01**
Interstitial lung disease	11 (22%)	3 (5%)	8 (62%)	0 (0%)	0 (0%)	**<0.01**
Right heart catheterisation						
MPAP, mm Hg	35 (29–45)	42 (33–52)	31 (27–32)	38 (36–46)	26 (23–37)	**0.02**
PAOP, mm Hg	11 (9–14)	21 (17–27)	11 (10–12)	13 (11–14)	13 (11–14)	**<0.01**
PVR, wood units	3.3 (2.4–8.1)	3.2 (2.6–6.0)	4.1 (3.2–5.1)	4.3 (3.5–11.2)	2.6 (1.9–5.5)	0.30
Sleep study						
Home sleep study	12 (24%)	23 (43%)	2 (15%)	8 (62%)	4 (100%)	**<0.01**
Time with SpO_2<90%, %	7.6 (0.1–40.7)	16.5 (2.6–45.5)	2.3 (0.3–20.9)	40.3 (23.9–97.7)	4.6 (3.1–6.9)	**0.02**
AHI-REI ≥5	20 (41%)	40 (75%)	5 (38%)	9 (69%)	3 (75%)	**0.03**
5≤AHI REI < 15	12 (24%)	14 (26%)	5 (38%)	4 (31%)	3 (75%)	0.06
15≤AHI REI < 30	4 (8%)	13 (25%)	0 (0%)	3 (23%)	0 (0%)	0.07
AHI-REI≥30	4 (8%)	13 (25%)	0 (0%)	2 (15%)	0 (0%)	–
AHI-REI, events per hour	2.8 (0.9–9.5)	12.0 (6.1–29.2)	3.7 (2.1–8.2)	10.0 (2.2–19.4)	6.4 (2.6–10.1)	**<0.01**
Obstructive apnoeas	0 (0–5)	6 (0–33)	2 (0–8)	5 (0–13)	–	0.20
Central apnoeas	0 (0–1)	0 (0–3)	1 (0–9)	4 (0–5)	–	0.16
Mixed apnoeas	0 (0–1)	0 (0–2)	0 (0–0)	0 (0–1)	–	0.81
Hypopneas	7 (3–26)	63 (36–100)	3 (1–34)	64 (51–162)	–	**<0.01**

*Data are summarised as median (IQR) for continuous variables and N (%) for categorical variables.
†P values are calculated using the Kruskal-Wallis test for continuous variables and the X^2 test for categorical variables. P<0.05 was considered statistically significant. P-values <0.05 are indicated in boldface.
AHI, apnoea-hypopnoea index; COPD, chronic obsrructive pulmonary disease; MPAP, mean pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; PVR, pulmonary vascular resistance; REI, respiratory event index.

WHO group IV pulmonary hypertension and 4 patients with WHO group V pulmonary hypertension. Table 1 compares baseline characteristics in the complete subject cohort by WHO pulmonary hypertension group. Patients in WHO groups II and III were older. The majority of patients were men in WHO groups III and V and women in WHO groups I, II and IV. BMI also differed across groups, with patients in group II having the highest BMI. Comorbid medical conditions and pulmonary artery opening pressures were consistent with patients' pulmonary hypertension classifications. Mean pulmonary artery pressure was highest in patients with WHO group II pulmonary hypertension. Table 2 compares baseline characteristics in the subset of patients who underwent polysomnography and shows that the characteristics of this subset mirror those of the complete cohort.

Sleep study results for the complete cohort are summarised by WHO pulmonary hypertension group in table 1. The majority of patients in WHO groups I, II and III underwent polysomnography while the majority patients in WHO groups IV and V had home sleep studies. OSA was common in all WHO pulmonary hypertension groups. The prevalence of OSA differed by WHO pulmonary hypertension group and was 41% (20 of 49) in WHO group I, 75% (40 of 53) in WHO group II, 38% (5 of 13) in WHO group III, 69% (9 of 13) in WHO group IV and 75% (3 of 4) in WHO group V. OSA was most severe in patients with WHO group II pulmonary hypertension, for which median AHI-REI was 12.0 events/hour, followed by WHO group IV, for which AHI-REI was 10.0 events/hour. Median AHI-REI was 2.8 in WHO group I, 3.7 in WHO group III and 6.4 in WHO group V. Time with SpO_2<90% (T90) was higher in WHO groups IV and II compared with the other groups.

Sleep study results for the subset of patients who underwent polysomnography are shown in table 2. Similar to the results for the entire cohort, OSA was common in all WHO pulmonary hypertension groups. OSA was most

Table 2 Clinical characteristics of the subgroup of patients who underwent polysomnography*

	WHO group I	WHO group II	WHO group III	WHO group IV	P†
Number of patients (N=83)	37	30	11	5	
Demographics					
Age, years	57 (46–69)	65 (54–74)	68 (54–73)	55 (49–56)	**0.03**
Male gender	8 (22%)	8 (27%)	6 (55%)	3 (60%)	0.08
Body mass index, kg/m^2	30 (26–33)	35 (29–40)	26 (25–33)	30 (29–31)	0.06
Chronic medical conditions					
Connective tissue disease	22 (59%)	4 (13%)	3 (27%)	0 (0%)	**<0.01**
Chronic obstructive pulmonary disease	5 (14%)	1 (3%)	5 (45%)	0 (0%)	**<0.01**
Interstitial lung disease	9 (24%)	2 (7%)	6 (55%)	0 (0%)	**<0.01**
Right heart catheterisation					
MPAP, mm Hg	34 (27–44)	43 (33–54)	31 (28–40)	37 (37–42)	0.14
PAOP, mm Hg	11 (9–13)	19 (17–26)	12 (9–14)	14 (11–14)	**<0.01**
PVR, wood units	3.3 (2.4–7.4)	3.5 (2.5–7.4)	4.8 (3.2–5.8)	4.3 (4.0–6.1)	0.84
Sleep study					
Total sleep time, minutes	317.3 (251.8–348.4)	327.3 (242.0–377.4)	302.9 (238.2–350.5)	363.0 (274.0–382.3)	0.90
Time with SpO_2<90%, %	8.6 (0.1–41.0)	14.0 (3.1–39.4)	4.6 (0.4–39.3)	98.8 (90.7–99.6)	**<0.01**
AHI≥5	13 (35%)	27 (90%)	4 (36%)	5 (100%)	**<0.01**
5≤AHI < 15	8 (22%)	10 (33%)	4 (36%)	3 (60%)	0.29
15≤AHI < 30	1 (3%)	8 (27%)	0 (0%)	0 (0%)	**<0.01**
AHI≥30	4 (11%)	9 (30%)	0 (0%)	2 (40%)	**0.04**
AHI, events per hour	2.5 (0.9–8.6)	22.5 (6.2–32.1)	3.7 (2.1–8.2)	10.6 (10.0–31.7)	**<0.01**
Obstructive apnoeas	0 (0–5)	6 (0–33)	2 (0–8)	5 (0–13)	0.13
Central apnoeas	0 (0–1)	0 (0–3)	1 (0–9)	4 (0–5)	0.18
Mixed apnoeas	0 (0–1)	0 (0–2)	0 (0–0)	0 (0–1)	0.77
Hypopnoeas	7 (3–26)	63 (36–100)	3 (1–34)	64 (51–162)	**<0.01**

*Data are summarised as median (IQR) for continuous variables and N (%) for categorical variables.
†P values are caclulated using the Kruskal-Wallis test for continuous variables and the X^2 test for categorical variables. P<0.05 was considered statistically significant. P-values <0.05 are indicated in boldface.
AHI, apnoea-hypopnea index; MPAP, mean pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; PVR, pulmonary vascular resistance.

common in WHO group II, for which prevalence was 90% (27 of 30), and in WHO group IV, for which prevalence was 100% (5 of 5). The prevalence of OSA was 35% (13 of 37) in WHO group I and 37% (4 of 11) in WHO group III. OSA was most severe in patients with WHO group II pulmonary hypertension, for which median AHI was 22.5 events/hour, followed by WHO group IV, for which AHI was 10.6 events/hour. Median AHI was 2.5 events/hour in WHO group I and 3.7 events/hour in WHO group III. The majority of apnoeas observed on polysomnography were obstructive, though hypopneas were the most common events among all WHO groups. The hypopneas could not be further classified as obstructive or central in origin. T90 was higher in WHO groups IV and II compared with the other groups.

The results of the multivariable negative binomial regression model for the entire cohort are shown in table 3. Controlling for age, gender, BMI and sleep study type, AHI-REI was 87% higher in patients with WHO group II pulmonary hypertension compared with patients with WHO group I pulmonary hypertension. In contrast, patients with WHO group III pulmonary hypertension had an AHI-REI 56% lower than patients with WHO I group pulmonary hypertension. Male gender was associated and an increase in AHI-REI of 92% relative to female gender. A 1 point increase in BMI was associated with a 3% increase in AHI-REI. The results of the multivariable negative binomial regression for subset of patients who underwent polysomnography are also shown in table 3. Controlling for age, gender, BMI and sleep study type, in patients who underwent polysomnography, AHI was 104% higher in patients with WHO group II pulmonary hypertension and 161% higher in patients with WHO group IV pulmonary hypertension compared with patients with WHO group I pulmonary hypertension. Patients with WHO group III pulmonary hypertension had an AHI 55% lower than patients with WHO I group pulmonary hypertension. A 1-point increase in BMI was associated with a 4% increase in AHI.

Table 3 Results of multivariable negative binomial regressions examining the relationship between WHO pulmonary hypertension group and obstructive sleep apnoea severity

	All patients (N=132)				Polysomnography only (N=83)			
	Dependent variable: AHI-REI (events/hour)				Dependent variable: AHI (events/hour)			
	N	Rate ratio	95% CI	P*	N	Rate ratio	95% CI	P*
WHO group I	49	1 (Reference)	-	-	37	1 (Reference)	-	-
WHO group II	53	1.87	(1.17 to 3.00)	**<0.01**	**30**	2.04	(1.14 to 3.67)	**0.02**
WHO group III	13	0.44	(0.23 to 0.84)	**0.01**	**11**	0.45	(0.22 to 0.93)	**0.03**
WHO group IV	13	1.48	(0.79 to 2.79)	0.22	5	2.61	(1.06 to 6.39)	**0.04**
WHO group V	4	1.19	(0.31 to 4.47)	0.80	–	–	–	–
Age, years		1.02	(1.00 to 1.04)	0.05		1.02	(1.00 to 1.05)	0.08
Male gender		1.92	(1.21 to 3.03)	**<0.01**		1.44	(0.71 to 2.90)	0.31
BMI, kg/m^2		1.03	(1.00 to 1.06)	**0.02**		1.04	(1.00 to 1.07)	**0.03**
Home sleep study		0.70	(0.46 to 1.07)	0.10				

*P<0.05 was considered statistically significant. P-values <0.05 are indicated in boldface.
BMI, body mass index.

DISCUSSION

In this retrospective cohort study examining the prevalence and severity of OSA in patients with pulmonary hypertension confirmed on right heart catheterisation, we found that OSA was common in all WHO pulmonary hypertension groups. In the complete cohort (N=132), the prevalence of sleep apnoea based on AHI-REI ranged from 37% and 38%, respectively, in WHO groups I and III, to 75% and 69%, respectively, in WHO groups II and IV. OSA severity based on AHI or REI was nearly two times as high (87% higher) in patients with WHO group II pulmonary hypertension compared with patients with WHO group I pulmonary hypertension, controlling for age, gender, BMI and sleep study type (table 3). In the subset of patients who underwent polysomnography (N=83), multivariable analysis similarly showed about a twofold increase (104%) in AHI in patients with WHO group II compared with WHO group I pulmonary hypertension (table 3).

Our investigation is the first study of its kind comparing OSA by WHO pulmonary hypertension group in patients with pulmonary hypertension confirmed on right heart catheterisation. By comparing OSA across WHO pulmonary hypertension groups, we show that OSA is common in patients with pulmonary hypertension across the main classification system used in clinical practice and that there is a strong association between WHO pulmonary hypertension group and OSA severity. Our study demonstrates that among patients with pulmonary hypertension, OSA may be most common and most severe among patients with WHO group II pulmonary hypertension.

Previous literature examing the prevalence of OSA in patients with pulmonary hypertension did not compare patients using the WHO classification system; and studies enrolled mainly patients with WHO groups I and IV pulmonary hypertension. The estimated prevalence and severity of OSA varied across studies, likely due to variability in subject population, small sample size and disparate AHI thresholds used to define OSA.[3–8] Dumitrascu *et al*, the largest study in the literature, included 169 patients and showed that the prevalence of OSA in patients with pulmonary hypertension differed by underlying aetiology but excluded patients with pulmonary hypertension due to left heart disease.[8]

The overlap of comorbid OSA with pulmonary hypertension likely has an adverse effect on pulmonary haemodynamics. It is widely known that patients with COPD-OSA overlap with untreated OSA have worse outcomes, including increased hospitalisation for COPD exacerbation and mortality compared to patients with COPD alone.[11] It is therefore interesting that in our study, OSA was more common and more severe in WHO group II than in WHO group IIIpulmonary hypertension. Chaouat *et al* evaluated pulmonary hypertension based on right heart catheterisation in 220 patients with OSA and reported an average mean pulmonary artery pressure of 26±6 mm Hg, suggesting that OSA is typically associated only with mild to moderate elevation of pulmonary pressures.[9] Only 2 of 37 patients in Chaoat *et al* had mean pulmonary artery pressure >35 mm Hg. In contrast, in our cohort, mean pulmonary artery pressure was much higher, 35 mm Hg in WHO group I and 43 mm Hg in WHO group II. Fifty per cent of patients with WHO group II pulmonary hypertension had moderate or severe OSA. Both overnight hypoxic burden and repetitive large intrathoracic negative pressure swings attributable to upper airway obstruction are implicated in the development of pulmonary hypertension in patients with OSA.[1 2]

We as investigators recognise that overnight hypoxemia affects pulmonary pressures, especially in patients with previously diagnosed pulmonary hypertension. We were very interested in comparing overnight hypoxemia across the WHO groups using T90 and were impressed by

the finding that along with OSA prevalence and severity measured by AHI, T90 was also higher in WHO groups II and IV, compared with the other WHO groups. However, an important limitation of our study is that we were unable to quantify how many patients were using supplemental oxygen during our sleep studies, affecting our determination of T90. Given this limitation, we cannot draw further conclusions in comparing T90 across WHO groups and hope to further examine the relationship between T90 and pulmonary hypertension in a future prospective study. Though use of supplemental oxygen may have potentially lead to an underestimatation of hypopneas and T90 in some patients, there was still a high prevalence of OSA and nocturnal hypoxemia in our cohort.

Other limitations of our study include its retrospective design and the inclusion of some patients who underwent home sleep study instead of polysomnography. Fourty-nine of 132 patients in our cohort had home sleep study rather than polysomnography, either due to patients' refusal or the limitations of their medical insurance. For this reason, we controlled for sleep study type in the multivariable model, including the entire cohort, and performed a separate analysis including only patients who underwent polysomnography (tables 2 and 3). The proportion of patients who underwent home sleep study was highest among the patients with WHO group II pulmonary hypertension, and, therefore, we may be underestimating the finding that OSA is most severe in patients with WHO group II pulmonary hypertension, as REI in home sleep studies is reported based on total recording time rather than total sleep time.

Another limitation of our study is that there were fewer patients with WHO groups III (N=13), IV (N=13) and V (N=4) pulmonary hypertension than patients with WHO groups I (N=49) and II (N=53) pulmonary hypertension. Furthermore, we are aware that mean pulmonary artery pressure was higher in patients with WHO group II pulmonary hypertension than in patients with WHO group I pulmonary hypertension as there may have been some patients with severe pulmonary hypertension in the latter group who were too ill to undergo a sleep study. While patients with WHO group II had higher BMI, we controlled for this variable in our multivariable analyses. We believe that the above limitations do not compromise the integrity of our main findings: (1) OSA is common among patients across the WHO pulmonary hypertension classification system, (2) OSA appears to be most common and most severe in WHO group II and (3) that evaluation for OSA in patients with pulmonary hypertension deserves greater attention. Further research is needed to determine whether OSA is a modifiable comorbidity not only affecting pulmonary pressures in this population but also patient-centred outcomes such as fatigue and 6 min walk distance that affect quality of life in patients with pulmonary hypertension.

In this investigation, we demonstrated that OSA is a common comorbidity affecting patients with pulmonary hypertension across WHO groups, and that in the context of this classification, OSA may be most prevalent and most severe in patients with WHO group II pulmonary hypertension. Our study highlights that evaluation for OSA requires greater consideration in the care of patients with pulmonary hypertension. The authors hope that this study lays the groundwork for future prospective investigation to determine whether treatment of OSA and overnight hypoxemia improves pulmonary haemodynamics and quality of life in patients with the overlap of pulmonary hypertension and OSA.

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