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ORIGINAL ARTICLE

Screening for obstructive sleep apnoea in high-risk patients with mood disorders

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Abstract

OBJECTIVE: Our study aimed to screen for obstructive sleep apnoea (OSA) in a clinical population of psychiatric patients with affective disorders and risk factors for OSA using screening devices in psychiatric clinical environments.
METHODS: Inpatients admitted with mood disorders in an inpatient psychiatric department were selected via inclusion and exclusion criteria and assessed for the risk factors of OSA. The inclusion criteria were: a diagnosis of an affective disorder confirmed by two independent psychiatrists, snoring or apnoeic pauses witnessed during regular night check-ups by nurses, and BMI > 25 kg/m^2. The exclusion criteria were: a comorbid psychotic disorder, previously diagnosed OSA, intellectual disability, organic mental illness, acute coronary syndrome, acute or chronic heart failure, acute pulmonary diseases, a history of stroke, neuromuscular disorders, or a myorelaxant treatment. All included patients underwent overnight monitoring by a screening device SomnoCHECK Micro Cardio. A certified somnologist assessed obtained data.
RESULTS: A total of 32 subjects (23 women and nine men) were included in the study. The mean age was 49.8 ± 8.8 years. Most participants had major depressive disorder (n = 23); another nine individuals had bipolar disorder. Diagnostic criteria for OSA were found in 50% of the sample, specifically in 88% of men and 33% of women. The correlation analysis identified several risk factors and variables.
CONCLUSIONS: This pilot study showed an increased risk of OSA in patients with mood disorders. Psychiatric patients with identified risk factors should be routinely screened for obstructive sleep apnoea and referred to proper treatment.

INTRODUCTION

Sleep apnoea syndrome (SAS), with its most common variant obstructive sleep apnoea (OSA), is the most common sleep breathing disorder. OSA prevalence in adults is estimated to be between 2% and 14%, rising to 20% in elderly patients (Eikermann *et al.* 2007, Young *et al.* 2008, Pombo *et al.* 2017). Primary symptoms of OSA occur at night and include snoring, breathing pauses, excessive sweating, nycturia, dry mouth, and headache (Pelletier-Fleury *et al.* 2004, Peppard *et al.* 2006). Day symptoms include excessive sleepiness, energy loss, irritability, social withdrawal, cognitive dysfunction, increased anxiety, and depressive symptoms (Peppard *et al.* 2006). Many studies have confirmed that obesity, higher age, male sex, snoring, pharyngeal anatomy abnormalities, and cephalometric characteristics present significant risk factors for OSA (Šonka *et al.* 2007, Somers *et al.* 2008, Ong *et al.* 2009). OSA is a real risk factor for hypertension, diabetes, hyperlipidemia, and psychiatric conditions like depression, increased irritability and cognitive impairment (Schroeder *et al.* 2005, Deldin *et al.* 2006, Nasr *et al.* 2010, Hattori *et al.* 2009, De Castro *et al.* 2013). One proposed interlink between OSA and mood disorders is chronic inflammation. In both OSA and mood disorders, its presence has been established in contemporary literature (McNicholas 2009, Monji 2021).

Studies of the comorbidity of OSA and mood disorders have widely diverged, putting the prevalence of depressive symptoms between 15 and 48% of OSA patients, with one study referring up to 63% of patients with OSA having depressive symptoms (Saunamaki & Jehkonen 2007, Harris *et al.* 2009, Ejaz *et al.* 2011). Given the increased prevalence of depressive symptoms in this population, sleep laboratories regularly assess depressive symptoms of their patients using screening tools. However, the similarities between OSA and mood disorder symptoms may lead to misdiagnosing sleep apnoea as a mood disorder and subsequent misuse of pharmacotherapy in traditional clinical settings (Smith *et al.* 2002). Moreover, the frequent side effect of antidepressants is weight gain, which can worsen OSA severity (Stores 2003).

According to guidelines, screening for OSA in psychiatric patients is recommended, especially when they show other risk factors (Braitman 2018). Considering OSA during psychiatric diagnostics is especially important because unrecognized OSA may worsen the symptoms of existing psychiatric disorders and obstruct the treatment process. Despite the guidelines' recommendations, screening for OSA in patients with mood disorders is far from typical. OSA screening in psychiatric care further highlights the considerable incidence of OSA in depressed patients, ranging from around 11-18%. After adjusting for risk factors, it can increase by up to 59% (Hattori *et al.* 2009, Hobzova *et al.* 2017, Vanek *et al.* 2020).

Previous studies on OSA screening in psychiatric patients used either validated questionnaires (e.g., Epworth's sleepiness scale, Berlin questionnaire, etc.) or polysomnography as a diagnostic standard (Hattori *et al.* 2009, Hobzova *et al.* 2017, Vanek *et al.* 2020). However, screening via questionnaires has limitations in its subjectivity when evaluating daytime symptoms, and some patients may not be aware of their night symptoms. Conversely, polysomnography is not readily available to screen all psychiatric patients (Hobzova *et al.* 2017, Vanek *et al.* 2020).

The first aim of our work was to identify the prevalence of OSA among patients with a mood disorder with at least two independent risk factors – an increased BMI and night-time snoring (observed by a nurse). The second aim was to assess known risk factors for OSA and associated disorders (hypertension, chronic inflammatory status) and compare them between OSA and non-OSA groups. The third aim was to assess whether a screening device for OSA routinely used in the outpatient practice is effective and practical for inpatients in the psychiatric department.

Our study is trying to answer whether the grade of depression and the severity of psychopathology may be related to the severity of OSA or other OSA-related risk factors, such as obesity, hypertension, chronic subclinical inflammation or hyperglycemia, or diagnosed hypertension or diabetes mellitus.

Hypotheses were set as follows:

(1) The severity of depression assessed by BDI-II is related to the severity of OSA.

(2) The overall severity of psychopathology assessed by CGI is associated with the severity of OSA.

(3) Depressed patients with OSA have a higher severity of depression than depressed patients without OSA.

(4) Depressed patients with OSA have a higher blood pressure than patients without OSA.

(5) Depressed patients with OSA show a lower decrease in blood pressure than patients without OSA.

(6) Depressed patients with OSA suffer more frequently from hypertension than depressed patients without OSA.

METHODS

Inpatients admitted with mood disorders from 1 February 2019 to 31 August 2019 into a university psychiatric department's inpatient psychiatric department were evaluated for participation in the study. The inclusion criteria were: 1) a diagnosis of an affective disorder (major depressive disorder- MDD/ bipolar affective disorder – BD) according to ICD-10 (Dilling & Dittmann 1990) confirmed by two independent psychiatrists, 2) snoring or apnoeic pauses witnessed during regular night check-ups by nurses, 3) overweight or obesity indicated by BMI > 25. The exclusion criteria were 1) a comorbid psychotic disorder, 2) previously diagnosed OSA, 3) a diagnosis of mental retardation, 4)

an organic mental disorder, 5) acute coronary syndrome, 5) acute or chronic heart failure, 6) acute pulmonary diseases, 7) a history of stroke, 8) neuromuscular disorders, 9) myorelaxant treatment. 10) acute respiratory infection. The selected patients then completed interviews regarding primary demographic data, comorbid diseases (hypertension, diabetes), completed questionnaires regarding sleep, adjacent anxious symptoms, and adherence to therapy, and were assessed for risk factors of OSA.

Assessment instruments

Following instruments validated for the Czech population were used: Beck Depression Inventory (BDI-II), Epworth's sleepiness scale (ESS), and Drug Attitude Inventory (DAI-10).

- BDI-II (Beck Depression Inventory, second edition; consists of 21 items from which patients pick depressive signs and rate their severity) (Beck & Steer 1984). Patients evaluate their state during the preceding two weeks. The internal consistency of the inventory is good ($\alpha = 0.86$ for the psychiatric population) (Storch *et al.* 2015). The Czech standardization was performed with Cronbach's alfa 0.90 (Ocisková *et al.* 2017).
- Epworth's sleepiness scale (ESS) – The ESS is a self-administered questionnaire with eight questions, rating on a 4-point scale (0-3) the likelihood of dozing off or falling asleep while engaging in eight different activities. The ESS score can range from 0 to 24, and higher ESS scores show increased sleep propensity in daily life. The scale has a high level of internal consistency (Cronbach's alpha 0.88) (Johns 1991, Kendzerska *et al.* 2014).
- Drug Attitude Inventory 10 (DAI-10) is a questionnaire created to measure patients' attitudes toward their prescribed medications (Shariati *et al.* 2018). The patient decides whether statements about the drugs are true or false, and the statements focus on drug effectiveness, perceived necessity, and adherence. The scale assesses current attitudes toward the medications, not whether the patients discontinued their medicine in the past (Hogan *et al.* 1976).

A senior psychiatrist completed the clinician version of the Clinical Global Impression (CGI) scale at the beginning and end of inpatient treatment.

- CGI (Clinical Global Impression-Severity of the disorder) is used to global assess the severity of a mental disorder (Guy 1976, Zaider *et al.* 2003).

Blood pressure was measured using a standard protocol recommended by (BHS – British hypertension society) via a standardized manometer with valid certification in all patients upon admittance and discharge from inpatient care, and routine blood tests were conducted. All included patients underwent overnight monitoring by a screening device, SomnoCHECK Micro Cardio, according to AASM (American Academy of Sleep Medicine) guidelines. The SomnoCHECK Micro Cardio is a certified screening device using non-invasive pulse oximetry and air-flow monitoring. Via analysis of pulse oximetry, the device can distinguish between central and obstructive sleep apnoea (Sommermeyer *et al.* 2012). The device provides the following data: apnoea hypopnea index (AHI), oxygen desaturation index (ODI), and percentage of sleep spent under the saturation of 90% (t90%). A certified somnologist evaluated all records. The device shows highly consistent results compared to polysomnography (Ficker *et al.* 2001, Bilgin *et al.* 2016); monitoring was conducted in standard conditions of the psychiatric department. All patients were treated according to guidelines for MDD and bipolar depression. No specific interventions were conducted regarding blood pressure during the inpatient care. Patients with confirmed OSA were referred to the sleep laboratory for further diagnosis and treatment.

Ethics

All patients signed informed consent, and their participation was voluntary and without reward. The research was conducted following the Helsinki declaration and the Guideline for Good Clinical Practice (EMA 2018). The ethical committee of the University Hospital Olomouc accepted the study's design (approval nr. 41/18).

Statistical evaluation

The results were processed with the statistical software Prism 3 (GraphPad Software Inc, La Jolla, CA, USA) and G*Power (Paul *et al.* 2007). Demographic data and mean total scores in the separate scales were calculated using descriptive statistics to find the means, standard deviations, and the type of data distribution. Correlation coefficients estimated associations between different categories. Fisher's test assessed the relationships between frequencies of alternative variables (sex, presence or absence of OSA, hypertension). Unpaired t-test analyzed differences between the subgroups. Paired t-tests calculated the significance of differences between the measurements at one group's start and the treatment's end. Two-way ANOVA for repeated measures was then used to compare groups and time. Effect sizes were interpreted according to Cohen (Cohen 1988). A 5% level of significance was used for all statistical tests.

RESULTS

A total of 32 subjects (23 women and 9 men) met the inclusion criteria and were included in the study. The mean age was 49.8 ± 8.8 years. The mood disorder present was major depressive disorder in 23 patients and depressive phase of bipolar disorder in 9 patients. The mean CGI score upon admittance (CGI-1) was "moderately to markedly ill" and decreased to "mildly

Tab. 1. Demographic data and comparison between non-OSA and OSA groups

Parameter	All patients	Comparison of non-OSA and OSA patients			
	(n=32)	non-OSA (n=16)	OSA group (n=16)	t, df /U	*p*-value
Age	49.8 ± 8.9	47.5 ± 9.4	52.1 ± 7.9	t=1.506 df=30	0.14
BMI	34.7 ± 6.7	34.2 ± 6.1	35.1 ± 7.2	t=0.421 df=30	0.67
AHI	9.6 ± 13.4	2.4 ± 1.6	16.7 ± 16.1	t=3.515 df=30	***p*<0.005**
ODI	9.3 ± 19.8	2.3 ± 2.6	16.3 ± 26.4	t=2.102 df=30	***p*<0.05**
t90%	8.5 ± 17.9	7 ± 14.52	10 ± 21.2	U=99	0.28
ESS	7.6 ± 4.4	7.7 ± 5.4	7.8 ± 3.4	t=0.2753 df=30	0.78
BDI-II	20.9 ± 14.2	20.9 ± 15.4	21 ± 13.2	t=0.025 df=30	0.98
DAI-10	5.8 ± 1.3	5.8 ± 0.9	5.8 ± 1.6	U=113	0.58
Paroxetine index	29.5 + 19.7	32.8 + 22.6	26.3 + 16.3	t=0.9430 df=30	0.35
Risperidone index	1.7 + 1.9	1.9 + 2.1	1.4 + 1.7	t=0.6593 df=30	0.51
CGI-1	4.4 ± 0.8	4.6 ± 0.6	4.1 ± 0.8	t=1.967 df=30	0.058
CGI-2	2.9 ± 0.8	3.1 ± 0.8	2.6 ± 0.7	t=1.852 df=30	0.07
Statistic: paired t-test	t=8.928 df=31; ***p*<0.0001**	t=5.809 df=15; **p<0.0001**	t=6.708 df=15; ***p*<0.0001**		
Two-way ANOVA		F= 0.36; Df=32, n.s.			
Hospital. days	24.5 ± 8,9	25.2 ± 7.6	23.8 ± 10.2	t=0.4502 df=30	0.65
BP systolic – 1	143.4 ± 16.3	144.8 ± 15.7	142 ± 17.1	t=0.4725 df=30	0.04
BP systolic – 2	132.0 ± 12.5	127.6 ± 12.2	136.4 ± 11.6	t=2.093 df=30	***p*<0.05**
Statistic: paired t-test	t=3.456 df=31; ***p*<0.005**	t=5.054 df=15; ***p*<0.0001**	t=1.040 df=15; n.s.		
Two-way ANOVA		F=1.38; Df=32, n.s.			
BP diastolic – 1	92.0 ± 10.2	93 ± 8.3	91 ± 11.9	t=0.5339 df=30	0.59
BP diastolic – 2	85.4 ± 9.1	83.1 ± 7.6	87.81+ 10.0	t=1.514 df=30	0.14
Statistic: paired t-test	t=3.203 df=31; ***p*<0.005**	t=4.167 df=15; ***p*<0.001**	t=1.011 df=15; n.s.		
Two-way ANOVA		F=2.058; Df=32, ***p*<0.05**			

Abbreviations: BMI – body mass index, AHI – Apnoea/hypopnea index, ODI - Oxygen desaturation index, t90% - the percentage of time spent under 90% saturation per night, ESS- Epworth's sleepiness scale, BDI-II - Beck Depression Inventory, DAI-10 - Drug Attitude Inventory 10, CGI- Clinical Global Impression (CGI-1 upon admittance, CGI-2 upon discharge, BP- Blood Pressure (BP-1 Blood pressure upon admittance, BP-2 Blood pressure upon discharge), *p* – statistical value, n.s. – not significant.

ill" at the end of the treatment – CGI-2 (Table 1). All subjects showed clinical improvement throughout the treatment. The thirty-two patients met both risk factors –snoring verified by nurses via their regular check-ups, and the mean BMI was 34.6 ± 6.6 (Table 1).

The average daily dose of antidepressant calculated as an equivalent dose of paroxetine was 29.5 ± 19.7 mg. 29 out of 32 subjects were treated with antidepressant medication. Nineteen subjects were treated with antipsychotics, with the average daily dose calculated as an equivalent dose of risperidone being 1.7 ± 1.9 mg. The depressed patients without OSA did not statistically significantly differ in the average doses of drugs from the OSA group (32.8 ± 22.6 versus 26.3 ± 16.3, unpaired t-test, t=0.9430 df=30; n.s. for antidepressants; 1.9 ± 2.1 versus 1.4 ± 1.7; unpaired t-test, t=0.6593 df=30; n.s. in antipsychotics).

Sixteen patients met the diagnostic criteria for OSA (AHI ≥ 5) according to ICSD-3.[40] In the sixteen patients in OSA, nine patients had AHI ≥10 (mild OSA); four patients had AHI ≥ 20(moderate OSA), and three patients had severe OSA (AHI ≥ 30). ICSD-3 also allows diagnosing OSA specifically in patients with mood disorders with added criteria that in patients with mood disorders with no other symptoms, AHI has to be ≥ 15 (Sateia 2014). This criterion was met in 5 patients. The OSA group included eight women and eight men. Twelve patients in the OSA group had comorbid hypertension. On the other hand, only five patients had hypertension in the non-OSA group. Comorbid

Tab. 2. Correlation analysis

	AHI	ODI	t90%	ESS	BMI	BP systolic	BP diastolic
Age	0.25	**0.35 S***	0.15	-0.14	-0.20	-0.16	0.01
BMI	0.16	0.21	0.23	0.08		-0.18	-0.05
BDI-II	0.09	-0.06	-0.24	0.11	0.26	0.21	0.22
EES	0.20	0.26	0.31		0.08	0.12	0.10
DAI-10	-0.06	-0.15	-0.10	-0.19	**-0.35 P***	-0.09	-0.002
CGI-1	-0.29	-0.22	-0.09	**-0.41 S***	-0.17	-0.28	-0.16
CGI-2	**-0.40 S***	-0.29	-0.15	-0.30	0.07	-0.02	0.21
CGI-difference	0.08	0.06	0.06	-0.08	-0.16	-0.15	-0.31

Abbreviations: BMI – body mass index, AHI – Apnoea/hypopnea index, ODI - Oxygen desaturation index, t90% - the percentage of time spent under 90% saturation per night, ESS- Epworth's sleepiness scale, BDI-II - Beck Depression Inventory, DAI-10 - Drug Attitude Inventory 10, CGI- Clinical Global Impression (CGI-1 upon admittance, CGI-2 upon discharge, BP- Blood Pressure (BP-1 Blood pressure upon admittance, BP-2 Blood pressure upon discharge), * $p<0.05$, S – Spearman's correlation, P – Pearson's correlation,

diabetes mellitus was found in 4 patients in the OSA group and five in the non-OSA group. OSA diagnosis rate in our study in a population with high-risk factors and comorbid mood disorders was 50% (16/32). By sex, OSA was diagnosed in 88% of men (8/9) and 33% of women (8/24).

Correlation analysis

We conducted exploratory correlation analysis with obtained data to further understand our results. We used Spearmen's and Pearson's correlation analysis according to the distribution of the correlated values.

The first part of the results focuses on the correlation analysis of the relationships between the degree of psychopathology and the essential parameters of OSA.

The severity of psychopathology and parameters evaluating the severity of OSA

The severity of depression as assessed by BDI-II and OSA severity parameters such as AHI, ODI, and t90% (Table 1). The results of the correlation analyses are described in Table 2. BDI-II showed only a marginally significant relationship with CRP (C-reactive protein) (Pearson's r = 0.51, $p = 0.052$, large effect size).

A non-significant negative relationship was found between the overall clinical severity assessed by CGI at baseline and AHI (Spearman r = -0.29; $p = 0.09$; small effect size). The initial CGI score significantly negatively correlated with AHI (Spearman r = -0.40; $p < 0.05$; medium effect size). No other assessed parameter was significantly related to CGI.

The severity of depression assessed by BDI-II was not statistically significantly related to the degree of drowsiness assessed by EES (Pearson r = 0.11; n.s.; small effect size). However, the overall severity of psychopathology assessed by the baseline CGI was statistically significantly related to the degree of drowsiness assessed by EES (Spearman r = -0.41, $p < 0.05$; medium effect size). The relationship between mean CGI and EES scores was not statistically significant at the end of treatment, but there is a statistical trend (Spearman r = -0.30; $p = 0,09$; medium effect size).

The severity of depression and BMI

The mean BMI values did not statistically significantly correlate with any measures assessing the degree of psychopathology, neither depression nor the overall clinical impression or the degree of drowsiness (Table 2). Moreover, BMI did not correlate statistically significantly with any of the parameters measuring OSA severity – AHI (Pearson r = 0.16; n.s.; small effect size), ODI (Pearson r = 0.21; n.s.; small effect size) or t90% (Pearson r = 0.23; n.s., small effect size). Interestingly, the mean BMI values were statistically significantly negatively connected with medication adherence evaluated by DAI-10 (Pearson r = -0.35; $p < 0.05$; medium effect size).

Depression and CRP

The severity of depression assessed by BDI-II was positively correlated with the CRP values at the borderline of statistical significance (Pearson r = 0.51 $p = 0.052$; large effect size) (Table 2). The higher the rate of depression, the higher the non-specific markers of inflammation.

Comparison of patients with diagnosed OSA and patients without OSA

The second part of the results compares the severity of psychopathology and the parameters evaluating OSA severity in depressed patients with OSA and depressed patients without OSA. In the total sample of 32 patients with affective disorder, 16 were diagnosed with OSA, while 16 did not meet OSA criteria.

Depression with OSA versus depression without OSA

Depressed patients with OSA did not show statistically significantly higher depression scores in BDI-II

than the non-OSA group (Cohen's d: 0.01; negligible difference). However, their average baseline CGI scores were lower, though this difference only approached the statistical significance threshold (Table 3, Cohen's d: Cohen's d: 0.71; medium difference).

Depression with OSA, blood pressure, and arterial hypertension

Depressed patients with OSA did not significantly differ in their mean systolic or diastolic blood pressure values at the beginning of treatment (Table 1; Cohen's d: 0.17; negligible difference). However, they differed statistically significantly in the systolic blood pressure at the end of hospitalization (unpaired t-test; t = 2.093 df = 30; p <0.05; Cohen's d: 0.74; medium effect size) (Table 1).

Both pressures, systolic and diastolic, significantly decreased during the treatment in depressed patients without OSA, which did not happen in the depressed patients with OSA (Table 1). The two-way ANOVA did not show a statistically significant difference between the groups over time in the systolic blood pressure. Still, the decrease in diastolic blood pressure was significant after treatment, and the blood pressure lowered more in the group without OSA (Table 1).

Considering hypertension, 12 patients in the OSA group and five in the non-OSA group had previously been diagnosed with hypertension. The difference between the frequencies reached statistical significance (Fisher's exact test; p <0.05).

Diabetes mellitus was equally common in both subgroups (in five patients with the comorbid OSA and four without the OSA, Fisher's exact test; n.s.).

DISCUSSION

This pilot study of the OSA screening in high risk patients with affective disorders is the first in the Czech psychiatric clinical environment. In our study of the at-risk population of psychiatric patients, the overall prevalence of OSA was 50% (16/32). A similar study by Hattori *et al.* with a high risk population captured a prevalence of 59.4% (Hattori *et al.* 2009). Kayukawa and Okada reported the OSA prevalence of 11.8% while applying the diagnostic criterion of AHI ≥ 10 in a broader sample of 119 patients. (Kayukawa & Okada 2005). Alam *et al.* found that 69% of depressed patients are also at high risk for comorbid OSA (Alam *et al.* 2012). In a more recent work from 2020, Tanielian *et al.* similarly reported that up to 58.2% (n = 91) of depressed patients were at high risk for OSA (Tanielian *et al.* 2020). The literature generally agrees that patients with depression are at an increased risk of developing OSA, and the prevalence of OSA is higher than in the general population (Eikermann *et al.* 2007, Hobzova *et al.* 2017, Vanek *et al.* 2020). The conclusions of our study confirm this. Of note is also a sex difference in OSA in our results. OSA is generally more prevalent in men than women (Eikermann *et al.* 2007, Young *et al.* 2008). Results of our study support this fact, as 88% of men in our sample met the criteria for OSA, whereas only 33% of women included in our study were diagnosed with OSA.

(1) The severity of depression assessed by BDI-II is related to the severity of OSA.

Our study found no correlation between the severity of depression and the number of apnoeas assessed by AHI, ODI, and t90%. Similarly, Hattori *et al.* did not find a statistically significant difference between patients with OSA and depression and patients with sole depression measured by BDI-II and the HAM-D scale (Hattori *et al.* 2009). One possible explanation for this can be a small sample in both studies (n=32 in our study and Hattori *et al.* 2009). A more extensive clinical sample could lead to statistically significant results, as shown by statistical trends in correlation analysis. The second possible explanation is a factor of subjectivity in BDI-II assessment, as shown by a relatively more significant standard deviation in mean BDI-II values in both OSA and non-OSA patients in our study.

(2) The overall severity of psychopathology assessed by CGI is associated with the severity of OSA.

No significant correlation was found between the overall clinical severity of the disorder and sleep parameters. The only exception was the correlation between CGI and AHI scores with a trend towards statistical significance. There was also a statistically significant correlation between CGI and AHI at the treatment end. This finding may suggest that patients with more sleep apnoea may have difficulty improving their overall clinical condition after the psychopharmacological treatment. OSA may thus prevent reaching remission or the clinical improvement in this population (Knechtle *et al.* 2019). This result is in accordance with Kaufmann *et al.*, who surveyed 265 000 individuals and found that patients with OSA and comorbid psychiatric disorders were substantially more likely to report unmet need for mental health care, despite reporting a more effective mental health service use (Kaufmann *et al.* 2017).

(3) Depressed patients with OSA have a higher severity of depression than depressed patients without OSA.

This hypothesis was not confirmed. Depressed patients without OSA and depressed patients with OSA showed, on average, a similar severity of depression as assessed by BDI-II. This outcome does not agree with the literature as several studies presented that the patients with OSA report higher severity of depression and suffer more commonly from daytime drowsiness (Akashiba *et al.* 2002, Sforza *et al.* 2002, Asghari *et al.* 2012). We attribute this to BDI-II being a subjective evaluation of depressive symptoms and thus prone to a bias. This explanation may be backed by significant differences in the clinician ratings in CGI.

(4) Depressed patients with OSA have a higher blood pressure than patients without OSA.

This hypothesis was not confirmed in our study. The depressed patients without OSA showed comparable systolic and diastolic blood pressure at the beginning of treatment, and the groups did not differ statistically significantly from these parameters. In the contemporary literature, it is widely understood that OSA, especially untreated, is closely linked to hypertension and cardiovascular risk (Williams *et al.* 1985, Silverberg *et al.* 1998, Phillips & O'Driscoll 2013, Imayama *et al.* 2021). The process of measuring blood pressure may explain this difference in findings, as patients in this study were measured shortly after their admittance to the psychiatric ward. The necessity of admittance to a closed ward is a stressful event for many patients, and their increased sympathetic activity caused by the stress could have influenced the measurements (Godoy *et al.* 2018).

(5) Depressed patients with OSA show, on average, a lower decrease in blood pressure than patients without OSA.

Our study confirmed this hypothesis for the diastolic blood pressure and less conclusively for systolic blood pressure. In the group without OSA, there was a statistically significant decrease in the systolic and diastolic blood pressure during the treatment of depression. In contrast, the group with OSA experienced, on average, no significant reduction in their blood pressure during the treatment of depression (Table 1). The difference between the groups was insignificant in the systolic blood pressure using the two-way ANOVA for repeated measures. For the diastolic blood pressure, the difference in the blood pressure drop was statistically significant between the groups. That means that the group without OSA returned to normal pressure after treatment of depression, while the group with OSA did not. According to our literature search and previous review articles, this result is unique and not described by other studies (Hobzova *et al.* 2017, Vanek *et al.* 2020). One proposed explanation for these findings is that mood disorders and OSA present a stressful burden on patients' organisms, resulting in the overactivation of the autonomous nervous system (ANS) (Hattori *et al.* 2009, Vanek *et al.* 2020). As both groups were treated for depression, the activation of ANS in the non-OSA group was resolved, whereas, in the OSA group, comorbid OSA remained a factor causing higher blood pressure and lower decrease on average.

(6) Depressed patients with OSA suffer more frequently from hypertension than depressed patients without OSA.

This hypothesis was confirmed: in the group with OSA; statistically, significantly more patients suffered from hypertension than those without OSA. This finding is in line with other studies that detected arterial hypertension in up to 50% of patients with OSA (Silverberg *et al.* 1998, Nieta *et al.* 2000, Baquet *et al.* 2009).

All patients in our study underwent routine blood tests that included CRP at our clinic. We included one incidental finding from the correlation analysis: a marginally significant correlation between BDI-II and CRP (Pearson's r = 0.51, p = 0.052, large effect size). Studies showed that CRP appears to be a peripheral biomarker that reflects peripheral and central inflammation in patients with MDD (Köhler-Forsberg *et al.* 2017, Felger *et al.* 2020, Osimo *et al.* 2019). Similarly, other studies found elevated interleukin-6 and TNF-alpha levels (Osimo *et al.* 2019).

Limitations

Our work's primary limitation is a small sample of patients. Given some statistical trends and in line with the current literature, it can be assumed that several correlations in the study reported would reach statistical significance in a larger sample. Considering this is the first work in psychiatric care in the Czech Republic, this pilot project will need to be followed by further research with a larger and broader sample of the clinical population.

Inclusion criteria give the second limitation as we choose to include patients with mood disorders and at least one of the symptoms of OSA, which can introduce selection bias in our sample. This selection bias can result in overestimating OSA prevalence and reduce the generalizability of our findings.

Another limitation of the work is using a screening device to assess the presence and severity of OSA. It can be assumed that using the gold standard of diagnostics, full polysomnography, would obtain more accurate data. However, it should be noted that one of the aims of the study was to test whether a given screening test is suitable for use in clinical practice and the detection of untreated OSA in at-risk psychiatric populations.

Another limit of the work presents the self-report measurements. We tried to balance the potential bias with the clinician version of the CGI. The cross-sectional nature of our study is also a possible limitation.

Future research

We suggest a study on a larger sample of psychiatric patients for future research. It would be advisable to divide patients according to their psychiatric disorders in a larger sample, as there is limited literature on OSA and bipolar disorder. Future studies should also employ subjective and objective questionnaires plus CGI to assess psychopathology's severity comprehensively. A study comparing the reliability and usability of screening devices versus polysomnography in a psychiatric setting would also be beneficial in further advancing the OSA problem in the psychiatric field.

CONCLUSION

At-risk patients with affective disorders who snore during the night and have other OSA risk factors (e.g., overweight) should be considered for the OSA screening. Easy-to-use and transfer screening devices can enhance the OSA diagnostics in psychiatric patients.

DISCLOSURE

The authors declare no conflicts of interest in this work.

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