Sleep quality in stable chronic obstructive pulmonary disease patients in Zagazig University Hospitals, Egypt

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Abstract  

Background: Chronic obstructive pulmonary disease (COPD) causes changes in sleep quality with accompanied nocturnal drops in oxygen saturation leading to cardiac dysrhythmias, pulmonary hypertension, and more arousal. However, this sleep disturbance is an aspect of COPD that is still under evaluation. Therefore, this work is designed to detect sleep quality with type and frequency of sleep-related breathing disorders in stable COPD patients. 

Results: This is a cross-sectional study that included 60 stable COPD patients. Full night polysomnographies were performed to all patients with assessment of their sleep quality and presence of sleep-disordered breathing. The studied patients were classified into two groups: group I (COPD with good sleep quality) and group II (COPD with poor sleep quality). The results revealed that the more severe the COPD degree, the worse the sleep quality parameters represented by sleep latency, sleep efficiency, and arousal index with a significant statistical difference ($P < 0.05$). Furthermore, a highly significant statistical difference was noticed regarding time spent in REM sleep among poor sleep quality patients with different grades of severity ($P = 0.003$). Additionally, obstructive sleep apnea hypopnea syndrome was the most frequent sleep-disordered breathing (65%), followed by nocturnal oxygen desaturation (17%) among poor quality sleeper. 

Conclusion: Abnormal sleep quality is more common in moderate and severe COPD patients. Obstructive sleep apnea is the most frequent sleep-related breathing disorders among COPD patients. Increased BMI%, higher Berlin scores, and lower values of PaO₂ and FOSQ-10 scores are strong predictors of poor sleep quality among COPD patients. 

Keywords: Chronic obstructive pulmonary disease (COPD), Obstructive sleep apnea hypopnea syndrome (OSAHS), Sleep quality

Background

Chronic obstructive pulmonary disease (COPD) is a serious global health problem [1]. Different studies proposed that more than 75% of patients with COPD may have nocturnal symptoms and sleep disturbances in the form of prolonged sleep latency, arousals, and frequent insomnias [2], resulting in decrease in patient’s activity during daytime associated with more severe disease and frequent exacerbations [3].

In COPD, the pathogenesis of sleep disorders appears to be a complex and multifactorial process, which may be a consequence of one or more of the following: physiological changes associated with sleep, disturbance in gas exchanges, and/or COPD medications [4]. Obstructive sleep apnea hypopnea syndrome (OSAHS) is considered a serious disorder represented by repeated nocturnal events of partial or complete cessation of breathing [5].

Patients with both OSA and COPD are considered to have the overlap syndrome (OVS) [6]. Both are associated with similar common pathophysiology, such as

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hypoxia and systemic inflammation, that contribute to cardiovascular and other comorbidities, particularly pulmonary hypertension which is highly prevalent in those patients [7]. OVS had more profound nocturnal and daytime hypoxemia and hypercapnia than patients with COPD or OSA alone [8]. Moreover, both were associated with poor outcome, including high mortality [6]. Unfortunately, sleep-disordered breathing symptoms are an aspect of COPD that is considerably neglected by many physicians leading to negative impact on treatment efforts for COPD patients [2].

Therefore, this work is designed to detect sleep quality with type and frequency of sleep-related breathing disorders in stable COPD patients.

Methods

Study design and setting

This cross-sectional study was carried out at a Sleep-Disordered Breathing unit (SDB) of Chest Department and Out-patient Clinic, Zagazig University Hospitals, during the period from February till August 2016.

Patients

This study included 60 stable chronic obstructive pulmonary disease patients with different degrees of severity as assessed by spirometry according to GOLD [9]. They were 42 males and 18 females with mean age of 58.46 ± 7.87 years.

All patients fulfilling the criteria of stable COPD with different degrees of severity were included [9], while the following conditions were excluded: COPD in exacerbations [9], or under mechanical ventilation or receiving non-invasive positive pressure ventilation, obstructive airway diseases rather than COPD, e.g., asthma or bronchiectasis. COPD patients with uncontrolled hypertension, unstable angina, uncontrolled diabetes, neuromuscular disease, cerebrovascular accident (CVA), left-sided heart failure, chronic renal failure, chronic liver cell failure, thyrotoxicosis, malignancy, psychiatric diseases, addiction and alcohol intake, or obesity hypoventilation syndrome were excluded. This research was approved by the Ethics Committee of Zagazig Faculty of Medicine. An informed written consent was collected from all participants.

Methods

All patients were subjected to the following:

1. Full meticulous medical history taking stressing on: associated comorbidities, history of drug intake and special habits (e.g., smoking, alcohol intake), and sleep-disordered breathing symptoms that were assessed by the following: Epworth sleepiness scale (ESS) questionnaire [10], Berlin questionnaire [11], and functional outcome of sleep questionnaire (FOSQ-10 short version) [12].

2. General and local chest examination, stressing on recording neck circumference, waist circumference, and body mass index (BMI) (weight in kg/height m2) [13].

3. Plain chest x-ray (postero-anterior and lateral views).

4. Pulmonary function tests (PFT)

5. Laboratory investigations: complete blood count (CBC), liver function tests (LFTs), kidney function tests (KFTs), random blood sugar (RBS), thyroid function tests (TSH, Free T3, and Free T4), and arterial blood gas analysis (ABGs): after awakening and during day time.

6. Electrocardiography (ECG).

7. COPD assessment test (CAT) [14] and the modified Medical Research Council (mMRC) dyspnea scale for assessment of dyspnea level [9].

8. Full-night polysomnographic sleep study (PSG): It was done using SOMNO screen™ plus (Germany) to all patients.

A. The following respiratory disorders were reviewed:

- Apneas and hypopneas with recording the presence of obstructive, central, or mixed events.

| Table 1 Demographic data of the studied patients |
|-----------------------------------------------|
| **Group I COPD patients with good sleep quality** (n = 20) | **Group II COPD patients with poor sleep quality** (n = 40) | **Test of significance** |
| Age (years) (mean ± SD) | 56.85 ± 7.13 | 59.27 ± 8.18 | t = −1.12 |
| | | | P = 0.264 |
| Sex | Male No. (%) | 11 (55%) | 31 (77.5%) | χ² = 3.21 |
| | Female No. (%) | 9 (45%) | 9 (22.5%) | P = 0.073 |
| Smoking | Smoker No (%) | 8 (40%) | 28 (70%) | χ² = 7.81 |
| | Non-smoker No (%) | 12 (60%) | 12 (30%) | P = 0.005 |

S = P value < 0.05
Apnea hypopnea index (AHI) which is classified as follows: normal (AHI < 5), mild sleep apnea (AHI 5–14), moderate sleep apnea (AHI 15–29), and severe sleep apnea (AHI ≥30) [15].

- Respiratory effort-related arousal (RERA), respiratory disturbance index (RDI), and arousals
- Cheyne-Stokes breathing

B. Detecting oxygen desaturation index (sum of all desaturations, minimal and baseline oxygen saturation)

C. Snoring Index

D. Assessment of sleep quality according to the following parameters: sleep latency, duration, efficiency, fragmentation, and the hypnogram. Accordingly, good sleep quality is defined as: sufficient duration > 7 h/night, high sleep efficiency > 85%, low sleep fragmentation < 25, and normal hypnogram [16].

Statistical analysis

Data were imported into the Statistical Package for the Social Sciences (SPSS version 20.0) software for analysis. According to the type of data, qualitative was presented as number and percentage and quantitative continuous group was presented by mean ± SD; the following tests were used to test differences for significance. Differences between frequencies (qualitative variables) and percentages in groups were compared by Chi-square test. Differences between parametric quantitative independent groups were compared by t test, while differences between non-parametric data were compared by Mann-Whitney test, however multiple parametric groups were compared by ANOVA test, and multiple non-parametric groups were compared by Kruskal-Wallis test. Correlation between groups was carried out by Pearson’s correlation. The results were considered significant if P value < 0.05 was detected.

Results

This study included 60 stable COPD patients with different grades of severity. All patients underwent PSG study. According to their sleep quality, they were classified into two groups: group I (20 COPD patients with good sleep quality) and group II (40 COPD patients with poor sleep quality). Both studied groups were matched regarding age and sex with non-significant statistical difference (P > 0.05). However, there is a highly significant statistical increase in smoking habit in poor sleep quality group II than those of group I (P < 0.01) (Table 1).

Table 2 Comparison between both groups regarding total sleep time, sleep latency, sleep efficiency, and arousal index

| Sleep Parameters                      | Group I COPD patients with good sleep quality (n = 20) | Group II COPD patients with poor sleep quality (n = 40) | Test of significance |
|---------------------------------------|--------------------------------------------------------|--------------------------------------------------------|----------------------|
| Total sleep time (min) (mean ± SD)    | 370.50 ± 39.6                                         | 269.32 ± 26.56                                         | t = 11.75a           |
|                                        |                                                        |                                                        | P = 0.00             |
| Sleep latency (min) (mean ± SD)       | 27.35 ± 2.8                                           | 32.92 ± 16.89                                         | z = 3.02b            |
|                                        |                                                        |                                                        | P = 0.004            |
| Sleep efficiency (%) (mean ± SD)      | 89.55 ± 2.58                                          | 61.6 ± 6.76                                           | t = 17.77a           |
|                                        |                                                        |                                                        | P = 0.00             |
| Arousal index (No/h) (mean ± SD)      | 4.385 ± 2.1                                           | 18.73 ± 6.15                                          | z = −9.7b            |
|                                        |                                                        |                                                        | P = 0.000            |

*a* Student t test

*b* Mann-Whitney test were used

H5 = P value < 0.001, S = P value < 0.05

Table 3 Sleep parameters among poor sleep quality COPD patients with different grades of severity

| Sleep Parameters                      | Poor sleep quality COPD patients (n = 40) |
|---------------------------------------|------------------------------------------|
|                                       | Mild (N = 4) | Moderate (N = 18) | Severe (N = 14) | Very severe (N = 4) | Test of significance |
|---------------------------------------|--------------|-------------------|-----------------|---------------------|----------------------|
| Total sleep time (min) (mean ± SD)    | 287.2 ± 18.8 | 274.8 ± 14.9      | 263.7 ± 32.3    | 246.0 ± 39.2        | F = 2.31             |
|                                        |              |                   |                 |                     | P = 0.09             |
| Sleep latency (min) (mean ± SD)       | 9.2 ± 2.9    | 10.1 ± 6.8        | 10.5 ± 5.3      | 27 ± 4.9            | F = 11.01            |
|                                        |              |                   |                 |                     | P = 0.009            |
| Sleep efficiency (%) (mean ± SD)      | 73 ± 1.6     | 65 ± 3.8          | 56 ± 2.3        | 53 ± 3.3            | F = 46.7             |
|                                        |              |                   |                 |                     | P = 0.007            |
| Arousal index (No/h) (mean ± SD)      | 11.7 ± 4.2   | 15.8 ± 4.3        | 21.8 ± 3.8      | 28 ± 6.1            | F = 15.12            |
|                                        |              |                   |                 |                     | P = 0.02             |

*a* F ANOVA test

S = P value < 0.05
Table 2 shows that COPD patients with poor sleep quality had a lower total sleep time and sleep efficiency ($P = 0.00$), but a more prolonged sleep latency ($P = 0.004$) and a higher arousal index ($P = 0.00$) when compared to COPD patients with good sleep quality.

Table 3 illustrates that among poor sleep quality COPD patients, the more severe the COPD degree, the worse the sleep quality parameters including sleep latency ($P = 0.009$), sleep efficiency ($P = 0.007$), and arousal index ($P = 0.02$), but not the total sleep time ($P = 0.09$).

Table 4 shows that the more the severity of COPD, the less the time spent in REM sleep ($p = 0.003$) among patients with poor sleep quality.

Various types of sleep-disordered breathing were observed among COPD patients with poor sleep quality in which OSAHS is the most frequent sleep-disordered breathing (65%), followed by nocturnal oxygen desaturation (17%) (Table 5).

Our results revealed a significant positive correlation between sleep efficiency and $\text{FEV}_1$, accompanied with highly significant negative correlation between sleep efficiency and BMI and a significant negative correlation between sleep efficiency and mMRC dyspnea scale (Figs. 1, 2, and 3) with $P$ value < 0.01, < 0.001, and < 0.05 respectively, as seen in Table 6.

Table 7 shows multiple regression analysis used to assess predictors of poor sleep quality in COPD patients. It isolates four significant independent predictors: increased BMI and Berlin score, decrease in $\downarrow \text{PaO}_2$ and $\downarrow \text{FOSQ-10}$ score ($P = 0.04$, $P = 0.008$, $P = 0.01$, $P = 0.000$) respectively. Values were estimated in logistic regression by ROC curve as $PH < 7.39$, Epworth SS > 9.8, FOSQ-10 Q < 13.2, Berlin Q > 1, BMI > 26.5, $\text{PaO}_2$ < 86.2, $\text{PaCO}_2$ > 45.5 and $\text{FEV}_1$ < 80% according to GOLD [9].

**Discussion**

Chronic obstructive pulmonary disease (COPD) patients are at risk of insomnia and difficulty in initiating and maintaining sleep. This sleep fragmentation and poor sleep quality had an important impact on their quality of life and health outcome measures [17]. Therefore, this work was designed to detect the frequency of sleep-related breathing disorders in COPD patients and its impact on sleep quality in such patients. In this study, stable COPD patients were included and classified into: group I (20 COPD patients with good sleep quality) and group II (40 COPD patients with poor sleep quality) patient.

In the current study, both groups were matched regarding age and sex (Table 1). In contrast to these results, Akerstedt et al. [18] reported that sleep quality was affected by gender difference and age. This may be due to their study being carried out on patients with extremes of age while our study was conducted on elderly patients: their mean age was 58.46 ± 7.87 years only. Besides, a significant statistical difference between group I

### Table 4 Sleep stages among poor sleep quality COPD patients with different grades of severity

| Sleep stages | Poor sleep quality COPD patients ($n = 40$) |
|--------------|------------------------------------------|
|              | Mild ($n = 4$)                           | Moderate ($n = 18$) | Severe ($n = 14$) | Very severe ($n = 4$) | Test of significance |
| N1 Min (mean ± SD) | 78.7 ± 25.5                             | 87.3 ± 23.8        | 94.8 ± 29.01     | 70.0 ± 46.9          | $F = 0.88$           |
| % of TIB (mean ± SD) | 16.50 ± 6.24                            | 18.11 ± 5.83       | 19.92 ± 6.00     | 14.75 ± 10.66        | $P = 0.41$           |
| N2 Min (mean ± SD) | 137.5 ± 17.09                           | 118.6 ± 38.5       | 133.3 ± 21.2     | 147.0 ± 62.8         | $F = 1.42$           |
| % of TIB (mean ± SD) | 28.75 ± 3.77                            | 24.83 ± 8.48       | 27.64 ± 4.60     | 30.75 ± 16.85        | $P = 0.78$           |
| N3 Min (mean ± SD) | 43.0 ± 15.6                             | 43.6 ± 17.9        | 28.2 ± 19.8      | 21.0 ± 42.6          | $F = 1.19$           |
| % of TIB (mean ± SD) | 9.00 ± 4.24                             | 8.50 ± 4.68        | 5.71 ± 4.77      | 4.50 ± 9.00          | $P = 0.31$           |
| REM Min (mean ± SD) | 28.0 ± 9.2                              | 25.21 ± 20.4       | 7.3 ± 6.9        | 7.2 ± 15.0           | $F = 3.92$           |
| % of TIB (mean ± SD) | 5.75 ± 1.70                             | 5.16 ± 5.57        | 1.46 ± 1.57      | 1.50 ± 3.00          | $P = 0.04$           |

TIB time in bed, REM Rapid eye movement, F ANOVA test

S=P value < 0.05

### Table 5 Sleep-related breathing disorders in COPD patients with poor sleep quality

| COPD patients with poor sleep quality ($n = 40$) | No. | % |
|-----------------------------------------------|-----|---|
| Obstructive sleep apnea hypopnea syndrome (OSAHS) | 26  | 65% |
| Central sleep apnea hypopnea syndrome (CSAHS) | 2   | 5%  |
| Nocturnal hypventilation                      | 5   | 13% |
| Nocturnal oxygen desaturation                 | 7   | 17% |
and group II was observed as regard smoking habit. These results signified that there were other factors that add further burden on sleep quality like smoking as a special habit independent from the pathophysiology of the COPD itself. Cohrs et al. [19] and Liao et al. [20] strongly supported that smoking negatively affect sleep. Preliminary data suggested that cigarette smoking was associated with poor sleep quality in the form of impairment in subjective sleep quality, sleep latency, sleep duration, and habitual sleep efficiency, with frequent sleep disturbances. Moreover the physiological desire for additional nicotine during sleep may cause smokers to awaken, leading to insomnia. Furthermore, nicotine causes the release of nitric oxide which relaxes the pharyngeal smooth muscles and reduces muscular tone and activity [21] with increasing the risk for snoring and obstructive sleep [20].

Regarding sleep parameters (Table 2), our results were in harmony with Mcsharry et al. [17] who concluded an impaired sleep quality among COPD patients with total sleep time = 272.5 ± 86.6 min, sleep latency = 36.8 ± 33.9 min, sleep efficiency = 66 ± 17%, and arousal index = 16.2 ± 14.1/h with REM stage 12.7 ± 8.3 min. Also, Krachman et al. [22] and Azuma et al. [23] reported lower values of sleep efficiency and total sleep time among such patients after performing polysomnographic sleep studies.

Concordantly, Adetiloye et al. [24], who assessed sleep quality in COPD patients after using the Pittsburgh Sleep Quality Index (PSQI), had noticed that the overall
Sleep quality is poor with the median sleep latency was 30 min, 33% having median total sleep time of less than 5 h; 90% of the studied patients had < 85% sleep efficiency, 41.7% reporting sleep efficiency of < 65%.

This sleep fragmentation is attributed to the aggravating common nocturnal symptoms of cough, dyspnea, chest tightness, or wheezing that are related to COPD. However, lying flat per se impairs pulmonary mechanics and gas exchange resulting in more hypoxemia, which is likely a stronger stimulus for arousals, as it induces neural injury that elicits cortical activation with neurotransmitter-modulated reflex for arousal aiming to correct the situation [24].

In the current study, it was obvious that the more COPD severity, the more worsening of sleep quality (Tables 3 and 4). Similar results were noticed by Chang et al. [25] and Vogelmeier et al. [26], who concluded that COPD patients with more symptoms (group B or group D; mMRC ≥ 2 or CAT ≥ 10) had a higher percentage of sleep disturbance; its causes are multifactorial, including increase in respiratory symptoms, oxygen desaturation, presence of OSA, psychiatric disorders, and medication-related insomnia like oral steroids, inhaled steroids, beta agonists, and theophylline.

This signified how COPD and its consequences, comorbidities, and complications affect sleep pattern of these patients. The combination of alveolar hypoventilation and ventilation-perfusion mismatch occurs mostly in the form of severe episodes during REM sleep. Besides, during REM sleep, the diaphragm and intercostal muscle activities were decreased, that proceed to reduced tidal volume and reduced minute ventilation. This progressed into frequent arousals, disturbed REM sleep with decreased sleep time, and more changes in sleep stages [27]. Additionally, Sanders et al. [28] reported that if poor sleep quality was detected with mild COPD patients which is less to occur, there would be an underlying cause for this sleep disturbance rather than COPD itself.

It was obvious from the current results that OSAHS is the most frequent sleep-disordered breathing (65%), followed by nocturnal oxygen desaturation (17%) (Table 5). These results were close to that of Abdel Maboud et al. [29] who stated that OSAHS and nocturnal oxygen desaturation were common in COPD patients with prevalence 76% and 24% respectively. Also, Povitz et al. [30] and Copur et al. [31] found that the prevalence of OSA in COPD patients in their studies were 69% and 77% respectively.

This certify how sleep in chronic obstructive pulmonary disease (COPD) is associated with oxygen desaturation, and the coexistence of COPD and obstructive sleep apnea (overlap syndrome) is very common. Overlap patients have worse sleep-related hypoxemia and hypercapnia than patients with COPD or OSA alone, as oxygen desaturation during sleep is more pronounced when the two conditions coexist. This causes more subjectively and objectively impaired sleep quality [32].

### Table 6

|                | r      | P value |
|----------------|--------|---------|
| BMI %          | −0.612 | <0.001  |
| FEV₁           | 0.660  | <0.01   |
| mMRC dyspnea scale | −0.540 | <0.05   |

BMI body mass index, FEV₁ forced expiratory volume at first second, mMRC modified Medical Research Council
S = P value < 0.05, HS = P value < 0.001

**Fig. 3** Correlation between sleep efficiency and mMRC dyspnea scale among studied COPD patients
Increased BMI and Berlin score, decreased \( \text{PaO}_2 \) and FOSQ-10 score were identified as independent predictors of poor sleep quality in COPD patients. It is also important to consider other factors such as smoking status, \( \text{PaO}_2 \) and \( \text{FEV}_1\% \) and sleep quality (PSQI). This controversy may be due to a limited group of studied patients, small number of studied patients, and different stages of stable COPD patients. Furthermore, future studies are suggested to find out the impact of depression, anxiety, and comorbidities on sleep quality in COPD patients and their effect on quality of life.

**Table 7** Logistic regression analysis for predictors of poor sleep quality in COPD patients

| Independent factors | Wald | Std. error | Standardized coefficient betas | OR (CI 95%) | \( P \) value |
|---------------------|------|------------|-------------------------------|-------------|--------------|
| BMI % > 26.5        | 4.521| 0.212      | −0.196                        | 2.14 (1.2–8.52) | 0.044**     |
| Low \( \text{FEV}_1\% < 80\% \) | 1.256| 0.073      | 0.103                         | 1.87 (0.85–12.5) | 0.282       |
| \( \text{PH} < 7.39 \) | 2.014| 0.276      |                               | 1.75 (0.62–5.62) | 0.053       |
| Smoking             | 2.142| 14.25      | 0.054                         | 2.85 (0.79–11.23) | 0.125       |
| \( \text{PaO}_2 < 86.2 \) (mmHg) | 5.682| 0.105      | 0.388                         | 3.85 (1.85–13.2) | 0.011*      |
| \( \text{PaCO}_2 > 45.5 \) (mmHg) | 0.954| 0.185      | 0.037                         | 2.11 (0.92–8.32) | 0.711       |
| ESS score > 9.8     | 0.854| 0.256      | −0.014                        | 3.12 (0.64–9.36) | 0.868       |
| FOSQ-10 score < 13.2| 6.254| 0.452      | 0.510                         | 4.25 (2.1–7.58)  | 0.00**      |
| Berlin score > 1    | 5.897| 0.195      | −0.394                        | 3.01 (1.85–13.28) | 0.008*      |

**Abbreviations**

BMI: Body mass index; \( \text{FEV}_1\): Forced expiratory volume at first second; \( \text{PaO}_2\): Partial arterial oxygen pressure; \( \text{PaCO}_2\): Partial arterial carbon dioxide pressure; ESS: Epworth sleepiness scale; FOSQ: Functional outcome of sleep questionnaire

\( S = P \) value < 0.05, \( HS = P \) value < 0.001

Besides, a significant positive correlation between sleep efficiency and \( \text{FEV}_1\) was observed, accompanied with highly significant negative correlation between sleep efficiency and BMI and a significant negative correlation between sleep efficiency and mMRC dyspnea scale (Table 6). This signifies that \( \text{FEV}_1\) and BMI highly contributed in determining sleep quality in COPD patient. Similar results were obtained by Chen et al. [33]. Additionally, In et al. [34] and Abd El-Fattah et al. [35] found negative correlation between PSQI and \( \text{FEV}_1\)% and FVC. However, Scharf et al. [36] found unremarkable difference regarding the relationship between \( \text{FEV}_1\)% and sleep quality (PSQI); this controversy may be due to a limited group of their studied patients. On the other side, a group of studies had more or less different results; Chang et al. [25] proposed that CAT score was an independent factor for poor sleep quality in such patients; additionally, Ulfathinah et al. [37] demonstrated that patients with moderate to severe dyspnea were 2.28 times more likely to suffer from poor sleep quality.

Abd El-Fattah et al. [35] confirmed that there is strong association between poor sleep quality and physical limitation, COPD exacerbation, anxiety, depression, and poor health-related quality of life after using a group of subjective questionnaires for its assessment, and lastly, Shah et al. [38] noticed that presence of restless legs and obstructive sleep apnea symptoms, waist circumference, predicted diffusion capacity, and HADS anxiety and depression scores were identified as independent predictors of poor sleep quality.

This study had the following limitations. First, not all precipitating factors involved in disturbed sleep quality were evaluated (e.g., depression and anxiety). Second, small number of studied patients. Third, Pittsburgh Sleep Quality Index (PSQI) was not used in this study which is more sensitive in evaluating sleep quality.

Finally, we recommend involving sleep quality assessment in the risk categorization and management of different stages of stable COPD patients. Also, further studies are suggested to find out the impact of depression, anxiety, and comorbidities on sleep quality in COPD patients and their effect on quality of life.

**Conclusions**

Abnormal sleep quality is common in COPD patients with different grades of severity mostly in moderate and severe cases. Obstructive sleep apnea is the most frequent sleep-related breathing disorders among COPD patients followed by nocturnal \( \text{O}_2 \) desaturation, nocturnal hypoventilation, and central sleep apnea. Increased BMI %, lower values of \( \text{PaO}_2\), higher Berlin scores, and lower FOSQ-10 scores are strong predictors of poor sleep quality among COPD patients.
expiratory volume at first second; PaO$_2$: Partial arterial oxygen pressure; PaCO$_2$: Partial arterial carbon dioxide pressure.

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**Authors’ contributions**

AG and ME participated in performing study design and coordination. DG and YA contributed in patient selection and acquisition, analysis, and interpretation of data. Preparation of the draft was carried out by DG. The manuscript was substantially revised by AG, ME, and DG. All contributing authors have read and approved the final manuscript file.

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**Availability of data and materials**

Data used are available from the corresponding author upon reasonable request.

**Ethics approval and consent to participate**

This study was approved by the Ethics Committee of Zagazig Faculty of Medicine. The committee’s reference number is not applicable. An informed written consent was collected from all participants.

**Consent for publication**

Consent for publication was obtained from all contributors.

**Competing interests**

Not applicable.

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