



Assessing Sleep and Mental Health Disorders in COPD Patients During Severe Exacerbations

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Abstract:

Introduction: Acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD) often lead to short-term hospitalization of patients. The sleep health of hospitalized COPD patients is an overlooked element within the inpatient setting. The objective of this study is to assess and define the prevalence of sleep and mental health complaints in patients admitted to a hospital with acute exacerbation of COPD.

Methods: In this prospective study, patients admitted with a COPD exacerbation at a local community hospital were administered a variety of questionnaires pertaining to sleep and mental health, including the Beck Depression Inventory (BDI-II), the Functional Outcomes of Sleep Questionnaire (FOSQ), the Epworth Sleepiness Scale (ESS), and the Pittsburgh Sleep Quality Index (PSQI). Questionnaires were administered in a stable steady state on discharge.

Results: 53 patients filled out the questionnaires. 50.9% of patients reported poor sleep quality with scores indicative of chronic insomnia, and 41.2% of patients reported excessive daytime sleepiness on the PSQI. 64% of patients indicated abnormal total scores (<18) on the FOSQ, and 84.3% reported severe impairment in social outcomes. Clinical depression BDI scores >9) was observed in 73% of patients.

Conclusion: Our results indicate a significant prevalence of sleep and mental health comorbidities in patients hospitalized for acute COPD exacerbations and highlight the need for screening tools and clinical interventions to reduce the burden of these comorbidities.

Keywords: Sleep, insomnia, depression, COPD.

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1. INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a major global health challenge, affecting approximately 200 million people worldwide and contributing to nearly 10% of the global disease burden [1, 2]. COPD is characterized by chronic airflow limitation, progressive dyspnea, and a

high prevalence of systemic comorbidities, including sleep and mood disorders such as depression and anxiety [3, 4]. These comorbidities worsen the clinical course of COPD, leading to reduced Quality of Life (QOL), impaired physical functioning, and increased healthcare utilization [5, 6]. The pathophysiological mechanisms linking COPD

with sleep and mood disturbances are multifactorial, including chronic hypoxemia, hypercapnia, sleep-related hypoventilation, nicotine withdrawal, and adverse effects of medications like bronchodilators and corticosteroids (Table 1) [6, 7].

Acute exacerbations, defined as episodes of worsening respiratory symptoms requiring medical intervention, affect more than 50% of COPD patients annually and remain a leading cause of morbidity, hospitalization, and mortality [8, 9]. These exacerbations not only accelerate lung function decline but also add significant psychological and physical burdens to patients. Evidence suggests that poor sleep quality is associated with increased dyspnea, reduced exercise tolerance, and a higher risk of subsequent exacerbations [10]. However, while the impact of sleep and mood disorders in stable COPD is well-documented, their prevalence and clinical significance during acute exacerbations of COPD (AECOPD), particularly in inpatient settings, remain underexplored.

Our study aims to address this critical gap in knowledge by quantifying the prevalence of sleep disturbances and mood disorders in patients hospitalized for AECOPD. Using validated tools, we assess sleep quality, daytime sleepiness, and depression to characterize the burden of these comorbidities. Understanding these trends may help develop better strategies to manage COPD by addressing both respiratory symptoms and mental health concerns.

2. MATERIALS AND METHODS

This prospective, single-center study was conducted at a community hospital and included adult patients admitted for moderate acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD). The patient admitted had the prior diagnosis of COPD as an outpatient based on spirometry and clinical vignette. Participants were

identified based on clinical presentation and a confirmed diagnosis of COPD exacerbation, defined as a sudden worsening of respiratory symptoms requiring medical intervention. AECOPD was diagnosed by the treating physician based on a combination of clinical history (increased dyspnea, sputum volume, or purulence), physical examination findings, and radiologic and laboratory evaluations. Patients with primary diagnoses of pneumonia (as defined by radiographic consolidation), congestive heart failure (based on echocardiographic findings or elevated BNP with compatible clinical presentation), or asthma (based on prior diagnosis and bronchodilator reversibility) were excluded from the study to minimize diagnostic confounding. There were patients with COPD and Asthma overlap in the study. Inclusion criteria comprised adult patients aged 18 years or older who were hospitalized for an acute exacerbation. Exclusion criteria included evidence of pulmonary conditions other than COPD (*e.g.*, interstitial lung disease, pulmonary embolism, or lung cancer), a history of radiographically suspicious pulmonary nodules, or a Body Mass Index (BMI) greater than 34, as obesity can independently influence sleep and mood parameters, history of insomnia, obstructive sleep apnea, depression, psychosis and post-traumatic stress disorder. Following eligibility screening and application of exclusion criteria, 53 patients were included in the study.

Approval for this study was obtained from the Institutional Review Board (IRB), and written informed consent was obtained from all participants. Data collection occurred on the day of discharge, as patients were considered to be in a clinically stable state, thereby minimizing the acute impact of exacerbations on subjective assessments. Participants were administered a series of validated self-reported questionnaires to evaluate sleep quality, daytime sleepiness, functional impairments, and depressive symptoms.

Table 1. Pathogenesis of sleep disorders in COPD.

Sleep Disorder	Etiologic Factors	Increased Risk of
Insomnia	-Respiratory symptoms -Nicotine use/withdrawal -Increased WOB, -hypoxia -Increased sympathetic activity -Comorbid anxiety and depression -Primary sleep disorders -Medications: theophylline, beta agonists, steroids	-Hypertension -Diabetes -Atherosclerosis -Myocardial infarction -Mortality
Sleep related hypoxemia	-Increased airway resistance -reduced TV, FRC, minute ventilation	Hypertension Pulmonary hypertension
Sleep hypoventilation	-hyperinflation and flattening diaphragm -Diminished tone of accessory respiratory muscles	Right ventricular dysfunction Cardiac arrhythmias Nocturnal death
OSA Central sleep apnea	-Chronic Steroid use -Increased airway edema from cor pulmonale -Decreased exercise capacity > obesity -Muscle weakness > increased airway collapsibility -presence of GERD, allergies, obesity, smoking	-cardiac dysrhythmias -severe pulmonary hypertension -right heart failure.
Restless leg syndrome	hypoxemia- hypercapnia induced dopamine imbalance Co-existing iron deficiency, renal failure	depression, anxiety, and panic disorder

The Beck Depression Inventory-II (BDI-II) was utilized to assess depressive symptoms over the preceding two weeks. This 21-item instrument employs a 4-point Likert scale (0–3) and provides total scores categorized as minimal/no depression (0–9), mild to moderate (10–18), moderate to severe (19–29), and severe (30–63). Items specific to sleep disturbances, such as difficulty initiating or maintaining sleep, are particularly relevant in COPD [11]. To evaluate the impact of sleep disturbances on daily functioning, the Functional Outcomes of Sleep Questionnaire (FOSQ) was administered. This 30-item instrument examines five domains: activity level, vigilance, intimacy and sexual relationships, general productivity, and social outcomes. Responses are rated from “extreme difficulty” to “no difficulty,” with lower scores indicating greater functional impairment caused by excessive sleepiness [12]. The Pittsburgh Sleep Quality Index (PSQI) was used to assess subjective sleep quality across seven domains: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. Each domain is scored on a scale from 0 to 3, with the global PSQI score (range: 0–21) reflecting overall sleep quality—higher scores indicate poorer sleep quality [13]. Excessive daytime sleepiness (EDS) was assessed using the Epworth Sleepiness Scale (ESS). This 8-item questionnaire evaluates the likelihood of dozing off during various sedentary activities, such as watching television or sitting quietly. Responses range from 0 (“would never doze”) to 3 (“high chance of dozing”), with total scores ranging from 0 to 24. A score of 11 or higher was considered indicative of significant EDS [14].

Descriptive statistics were calculated to summarize demographic and clinical data. Continuous variables, such as age, BMI, and questionnaire scores, were expressed as means with standard deviations, while categorical variables were presented as frequencies and percentages. Between-group comparisons were performed using independent t-tests for continuous variables and chi-square tests for categorical variables. A p-value of less than 0.05 was considered statistically significant. All statistical analyses were conducted using Microsoft Excel and SPSS version 25 for Windows (IBM Corp., Armonk, NY).

3. RESULTS

This study included 53 patients admitted for acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD). The majority of the study population (92.5%) had a diagnosis of COPD alone, while a small proportion presented with features of COPD and asthma overlap syndrome. The mean age of participants was 65 ± 14 years, and the cohort consisted of 52.8% females (n=28) and 47.2% males (n=25), reflecting a near-equal gender distribution.

3.1. Beck Depression Inventory-II (BDI-II)

The BDI-II was utilized to assess the severity of depressive symptoms within the study population. Clinical depression, defined as a BDI-II score >9 , was observed in

73% of the participants. Notably, 39.6% (n=21) exhibited moderate to severe depression (BDI-II score 19–29), and 13.2% (n=7) reported scores indicative of severe depression (BDI-II score ≥ 30). Depression prevalence did not differ significantly between genders, with 43.4% of males and 39.6% of females reporting clinically significant depressive symptoms. The analysis of specific BDI-II items revealed notable trends. Anhedonia, reported by 70.5% (n=39) of participants during exacerbation, underscores the emotional burden in this acute phase. While causality cannot be established and mood symptoms may be transiently influenced by steroid use, these findings highlight a need for psychological support during exacerbations. Additionally, 29.4% of patients indicated waking up 1–2 hours earlier than usual, with difficulty returning to sleep, which reflects underlying sleep disruption associated with depressive symptoms. Functional impairments (question 15), particularly difficulty in maintaining daily routines, were reported in 29.4% of participants, underscoring the overlap between physical debilitation and depressive symptomatology in this population.

3.2. Functional Outcomes of Sleep Questionnaire (FOSQ)

The Functional Outcomes of Sleep Questionnaire (FOSQ) revealed significant sleep-related functional impairments in the cohort. The mean FOSQ score for the group was 15.4 ± 6.12 , indicating substantial difficulty across various functional domains. A total of 64.2% (n=34) of participants had abnormal FOSQ scores (<18), reflecting clinically relevant impairments in daytime functioning. Among those with abnormal scores, 59.4% (n=19) were males and 40.6% (n=13) were females. Domain-specific analysis of FOSQ revealed that social outcomes were the most severely affected, with 84.3% of patients reporting significant impairment (scores ≤ 1). This suggests a profound disruption in social engagement, possibly exacerbated by sleep disturbances, daytime sleepiness, and mood disorders. Additionally, general productivity was severely reduced in 27.4% of participants, while 41.1% reported an absence of or significantly impaired intimate relationships, highlighting the far-reaching impact of poor sleep quality on emotional and interpersonal functioning. Vigilance, an essential component of cognitive functioning, was another domain significantly affected, with 33.3% of patients reporting extreme difficulty (scores <1) maintaining alertness during daily activities. Notably, there was a significant overlap between depression and poor functional outcomes: 78.1% of patients with abnormal FOSQ scores also exhibited at least mild-to-moderate depression, reflecting the interconnected nature of sleep disturbance, functional impairment, and mood disorders [Figure 1].

3.3. Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI) was administered to evaluate subjective sleep quality. Poor sleep quality, defined as a PSQI global score ≥ 5 , was reported by 50.9% of participants, indicating a high

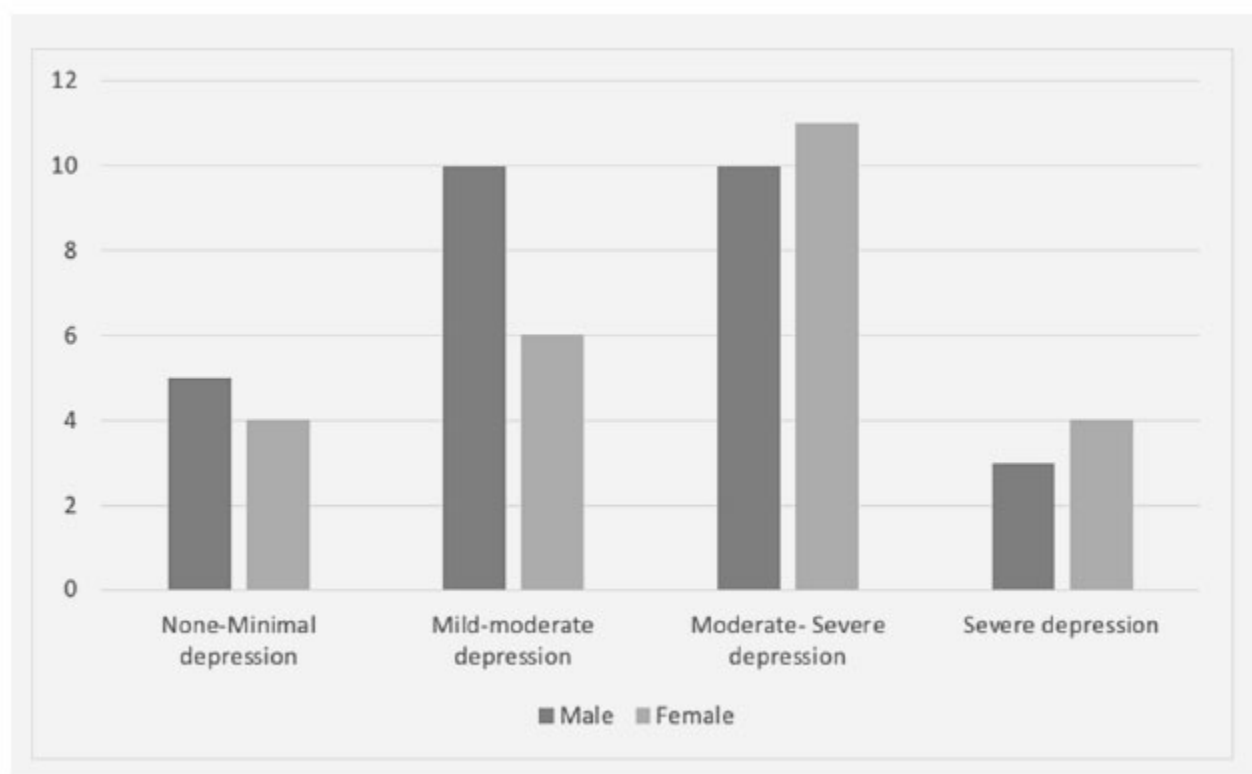


Fig. (1). Overview of FOSQ and PSQI in COPD patients with acute exacerbation ((n) indicates prevalence of abnormal results).

prevalence of sleep disruption. Sleep latency, the time taken to initiate sleep, was markedly prolonged, with an average latency of 65 minutes. Specifically, 74.5% of participants reported taking 30 minutes or longer to fall asleep, with 25.4% experiencing severe delays (>60 minutes). Despite the prolonged sleep latency, the majority of patients (60.8%) reported good sleep efficiency, defined as >85%. Sleep duration was notably variable, with 23.5% reporting ≤6 hours of total sleep time, while only 39.2% achieved >7 hours of sleep. Importantly, 68.6% of patients indicated that they had not used any sedatives or hypnotics, reflecting either patient reluctance to use pharmacologic interventions or clinician hesitancy to prescribe them due to concerns regarding respiratory depression. Mild to moderate daytime disturbances related to sleep, such as waking up early, temperature-related discomfort, dreams, and breathing difficulties, were reported by 96.1% of participants. Daytime dysfunction, including trouble staying awake during activities such as driving, eating meals, or social interactions, was noted in all patients. However, the majority (96.1%) rated these disturbances as mild to moderate on the scale (Fig. 1).

3.4. Epworth Sleepiness Scale (ESS)

Excessive Daytime Sleepiness (EDS), as assessed using the Epworth Sleepiness Scale (ESS), was prevalent in this cohort. A total of 41.2% of participants had ESS scores

>10, consistent with clinically significant EDS. Severe daytime sleepiness, defined as an ESS score >16, was observed in only 5.7% (n=3) of patients. While severe EDS was relatively uncommon, the high prevalence of moderate EDS underscores its impact on daily functioning and highlights the need for comprehensive sleep assessments in patients with acute COPD exacerbations.

4. DISCUSSION

4.1. Sleep and COPD - Sleep Quality

The relationship between sleep disturbances and COPD is multifactorial, with contributions from disease physiology, comorbidities, and environmental factors. Sleep quality is particularly impacted by chronic hypoxemia, hypercapnia, systemic inflammation, medications such as bronchodilators or corticosteroids, and habitual nicotine use, which can interfere with normal sleep architecture [11-18].

Our study found that 50.9% of patients reported poor sleep quality, with Pittsburgh Sleep Quality Index (PSQI) scores indicating moderate to severe chronic insomnia. Interestingly, although the majority (84.4%) reported preserved sleep duration (>5 hours), sleep latency was abnormal in 74.5%, with 25.4% experiencing severe delays. This highlights a unique feature of sleep dysfunction in COPD, where prolonged sleep onset is prevalent despite overall sleep preservation. Similar

findings were reported by Budhiraja *et al.*, who found that reduced sleep efficiency (<85%) affected 44% of COPD patients [15]. Comparatively, our data indicated slightly worse sleep efficiency, with 39.2% of participants experiencing reduced quality. This discrepancy may be related to differences in study settings or population characteristics.

The mechanisms driving insomnia in COPD patients are likely rooted in the interplay of nocturnal symptoms, such as cough, dyspnea, and hypoxia, which disrupt the ability to initiate and maintain sleep. Physiologically, these disruptions may lead to fragmented sleep, reduced slow-wave sleep, and an increased time spent in lighter, non-REM stages [7]. These abnormalities have downstream effects on daytime function, emotional well-being, and quality of life, underscoring the importance of evaluating sleep health in COPD patients.

4.2. Daytime Sleepiness and COPD

Excessive Daytime Sleepiness (EDS) is a frequently reported symptom in COPD and can significantly impair daily functioning, productivity, and overall quality of life (7). In our study, 41.2% of patients reported abnormal daytime sleepiness based on the Epworth Sleepiness Scale (ESS). This result aligns with previous literature highlighting EDS as a common but often underrecognized symptom in COPD, particularly during acute exacerbations when nocturnal symptoms may worsen [19]. A significant confounding factor here may be the presence of co-morbid sleep apnea in patients with COPD. Although formal screening for Obstructive Sleep Apnea (OSA) was not performed, prior studies have reported that OSA is present in up to 66% of COPD patients, suggesting it may have been a contributing factor in our cohort [19]. While the coexistence of COPD and OSA (often termed overlap syndrome) exacerbates both daytime and nocturnal symptoms, this was beyond the scope of our study.

The mechanisms underlying EDS in COPD are multifactorial. Nocturnal hypoxemia, hypercapnia, dyspnea, and frequent awakenings due to cough or discomfort disrupt sleep continuity and reduce overall sleep quality [7]. Additionally, systemic inflammation during acute exacerbations and altered respiratory mechanics likely exacerbate sleep fragmentation, further contributing to daytime sleepiness [15].

4.3. Mood Disorders, Functional Outcomes, and COPD

Mental health comorbidities are among the most pervasive yet underrecognized issues in COPD patients. In our study, clinical depression (Beck Depression Inventory-II score >9) was reported in 73% of participants, indicating a significant psychological burden. This prevalence aligns with findings from other studies, where rates of depression in COPD patients have ranged from 40% to 75%, depending on disease severity and study setting [20, 21]. However, the majority of those studies are in outpatient settings. In a prior study in an inpatient setting using the Hospital Anxiety and Depression Scale

(HADS) in Spain, the prevalence of probable depression was estimated to be 67.7% and this appears consistent with our data [22].

Our study also demonstrated a significant impact of poor sleep and depression on functional outcomes, as measured by the Functional Outcomes of Sleep Questionnaire (FOSQ). A total of 64% of patients reported abnormal FOSQ scores, suggesting severe impairment in key functional domains. Notably, 84.3% reported deficits in social functioning, likely reflecting the combined effects of sleep disruption, daytime fatigue, and mood disorders. These deficits exacerbate social isolation and physical inactivity, which are common in COPD patients and further reduce overall quality of life.

The interplay between COPD, sleep, depression, and poor functional outcomes is well-documented. Depression is associated with higher healthcare utilization, increased readmission rates, and worse adherence to treatment regimens [21]. Furthermore, sleep disturbances and depression are bidirectionally related, each exacerbating the other [23]. Addressing these issues in parallel through targeted interventions may help mitigate their combined impact on patient outcomes.

4.4. Study Limitations

While our findings provide valuable insights, several limitations must be acknowledged. First, our study relied on self-reported questionnaires, which, while validated, remain subjective. Objective assessments such as polysomnography or actigraphy could provide a more robust measure of sleep quality. Second, our sample size was relatively small (n=53) and drawn from a single community hospital, which may limit the generalizability of our findings. The high proportion of female participants (52.8%) is atypical for COPD cohorts, which are generally male-predominant. This may reflect selection bias related to our recruitment site or underlying regional smoking patterns. The limited sample size also precluded meaningful subgroup analyses, such as by sex, and we were unable to perform sex-adjusted analyses. These factors should be considered when interpreting our results. Although our findings are consistent with previous studies, larger multicenter studies are necessary to confirm the generalizability of our results. Assessments were conducted at discharge to ensure clinical stability; however, we acknowledge that this timing may reflect nonspecific effects of hospitalization (*e.g.*, sleep disruption, stress) rather than exacerbation-related changes alone. Additionally, the absence of a control group (hospitalized patients without COPD) limits our ability to attribute these findings solely to COPD rather than the general effects of hospitalization. Inclusion of clinical variables such as hypoxia, ICU admission, or use of non-invasive ventilation or intubation could have avoided further confounding association between disease severity and sleep or mental health outcomes. Additionally, while the PSQI assesses sleep quality over the past month, it may not accurately capture chronic insomnia, which requires symptoms for at least three months. Future

studies should incorporate these clinical variables and validated tools for chronic insomnia. Lastly, while we focused on sleep quality and mental health during hospitalization, it would be valuable to assess the longitudinal impact of these comorbidities following discharge. Identifying whether interventions targeting sleep or mental health improve long-term COPD outcomes could help refine management strategies.

4.5. Clinical Implications and Future Directions

Our study identifies a significant gap in the routine assessment of sleep quality and mental health among hospitalized COPD patients. Simple and validated screening tools, such as the PSQI, ESS, and BDI-II, can be readily incorporated into clinical practice to identify patients at risk. Early recognition of sleep disturbances and mood disorders enables timely interventions, including non-pharmacological treatments like cognitive-behavioral therapy for insomnia (CBT-I) and targeted therapies for depression and anxiety.

The high prevalence of daytime sleepiness observed in our cohort underscores the need to screen for comorbid Obstructive Sleep Apnea (OSA). Formal sleep studies for high-risk patients, combined with early initiation of CPAP therapy when indicated, may offer significant benefits for respiratory function, symptom management, and overall sleep quality, particularly in cases of overlap syndrome. Future research should evaluate the impact of integrated interventions targeting sleep quality, mental health, and functional impairments on COPD-related morbidity and mortality. A multidisciplinary care model that includes pulmonologists, sleep specialists, and mental health providers may enhance patient outcomes by addressing these interconnected issues comprehensively.

CONCLUSION

Exacerbations of Chronic Obstructive Pulmonary Disease (COPD) are a major cause of hospital admissions, and research suggests that COPD patients are at high risk of experiencing sleep disturbances and mental health issues. While treating COPD exacerbations is crucial, hospitalization can also provide an opportunity to identify and address these underlying health problems, which can worsen COPD and its comorbidities. Increased awareness and access to screening tools can help healthcare providers recognize subtle or masked symptoms of sleep and mental health disturbances in this patient population. By implementing effective interventions, healthcare providers can improve the quality of life for COPD patients and potentially reduce the burden of COPD exacerbations. By prioritizing sleep and mental health care, clinicians can take meaningful steps toward improving COPD management and enhancing patient well-being.

DECLARATION

An abstract of this study was presented at the American College of Chest Physicians (CHEST) annual meeting and published as a supplement in the *CHEST* journal [24].

Conceptualization was carried out by SS and SSU, while methodology was developed by T. Dighe, SS, SR, MS, and SSU. Validation was performed by TD, SS, and SSU. Formal analysis was conducted by TD, SS, SR. Investigation was done by TD, SS, and SR. Resources were provided by SS and SSU. Data curation was managed by TD, SS, SR, and SSU. The original draft was prepared by TD, SS, SR, MS, and SSU, who also contributed to writing, review, and editing. Visualization was handled by TD, SS, SR, MS, and SSU. Supervision was provided by SS and SSU, with project administration managed by SSU.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of Corpus Christi Medical Center (4/12/2018).

HUMAN AND ANIMAL RIGHTS

No animals were used in this research. All procedures performed in studies involving human participants were in accordance with the ethical standards of institutional and/or research committee and with the 1975 Declaration of Helsinki, as revised in 2013.

CONSENT FOR PUBLICATION

Informed consent was obtained from all subjects involved in the study.

AVAILABILITY OF DATA AND MATERIALS

The data and supportive information are available within the article.

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None.

CONFLICT OF INTEREST

Dr. Salim Surani is the Co-EIC of the journal TORMJ.

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