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Short Review

SIGNIFICANCE OF EARLY DIAGNOSIS OF PSYCHIATRIC DISORDERS IN SLEEP APNEA PATIENTS

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ABSTRACT

Obstructive Sleep Apnea (OSA) is a debilitating condition stemming from disruption to the respiratory system during sleep. At present, the nature of the relationship between OSA and mood, specifically depression and anxiety, is still unclear. The purpose of this paper is to shed some light on this relationship. There is an urgent need to better understand the roles of psychological illness in OSA. Well-designed longitudinal studies are needed to examine temporal relationships between the OSA, and psychological diseases and further research is needed to establish the role of confounders, and effect modifiers such as gender, in any apparent relationship. Sleep clinicians are advised to consider psychological diseases a likely cause of sleepiness and fatigue. Several possible causal mechanisms linking OSA, and depression have been proposed but not established. Patients who have depression as well as OSA appear worse off than those with OSA only, and depressive symptoms persist in at least some patients in short term studies of treatment for OSA. Direct treatment of psychological diseases in OSA might improve acceptance of therapy, reduce sleepiness, and fatigue and improve quality of life, but intervention trials are required to answer this question.

Keywords: Depression, Continuous Positive Airway Pressure, Psychological Illness, Sleep Apnea.

1. INTRODUCTION

Obstructive Sleep Apnea (OSA) is a potentially serious sleep related breathing disorder characterized by repeated episodes of upper airway obstruction during sleep [1]. According to a US-based study, Sleep Apnea was diagnosed by polysomnography (PSG) and the prevalence of Sleep Apnea was defined by an apnea-hypopnea index (AHI) ≥ 5 without includingthe daytime sleepiness criteria, and 24% for men and 9% for women under the age of 65 years were reported to be affected [2]. Obstruction of the upper airway may present as apnea, hypopneas, or respiratory effort-related arousals (RERAs) which often results in oxygen desaturation, repeated arousals and fragmentation of sleep. Sleep apnea is also associated with metabolic syndrome like comorbid obesity, hypertension, and diabetes. In recent times, there has been tremendous increase in cases of comorbidity of Sleep Apnea with psychological/psychiatric symptoms. Psychiatric comorbidity in Sleep Apnea and adherence to Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BiPAP) therapy has been reported to adversely affect the quality of life of patients [3]. Anxiety and Depression are reported commonly in adults with Sleep Apnea. Various prevalence studies and reviews have suggested evidence of elevated rates of psychological symptoms in individuals with sleep apnea [4-6]. Sleep symptoms may also artificially elevate patient scores on psychiatric assessment scales such as the Beck Depression Inventory (BDI), Profile of Mood States (POMS), and Minnesota Multiphasic Personality Inventory (MMPI) since these scales have questions relating to sleep symptoms such as insomnia and fatigue that are common to both OSA and psychiatric conditions [7, 8].

The lack of diagnosis of Sleep Apnea in people with mental illnesses has various negative consequences. The symptoms of sleep apnea might imitate the symptoms of multiple mental illnesses such as negative symptoms of schizophrenia and symptoms of depression; they might as well aggravate the cognitive impairment [9, 10]. Several drugs used in mental disorders have been found to aggravate symptoms of Sleep Apnea [11].

2. PATHOGENESIS OF SLEEP APNOEA

Respiratory ways within the nasal cavity and the lower respiratory tract are rigidly supported by cartilage and bone tissue, which prevents the loss of their patency but on the contrary the patency of the lower part of the upper respiratory tract depends upon the stabilizing tension of muscles of the pharynx, soft palate, and tongue. During inhalation when the negative pressure in the airway becomes higher than stabilizing muscle tension, the Upper Respiratory Tract gets narrowed or completely closed. It may occur along the entire length, but it mainly takes place within the central part of the pharynx, at the level of the soft palate and the tongue. This phenomenon causes episodes of apnea [12, 13].

3. RISK FACTORS

The most common risk factors for Sleep Apnea include Obesity, Male gender, Old age (although the most severe forms of apnea occur in younger age groups), Hypothyroidism, Acromegaly, Alcoholism, and Tobacco consumption.

The patency of the Upper Respiratory Tract is also affected by sleeping in a supine position, elongated soft palate, enlarged tongue and tonsils, changes in the anatomical construction of facial and pharyngeal wall - excess of body fat and lymphoid tissue, and diseases that deteriorate nasal patency.

Breathing disorders during sleep with obstruction of Upper Respiratory Tract worsen during treatment with numerous drugs like sedatives, anesthetics, narcotic analgesics, neuroleptics, beta-adrenolytics etc. which have inhibitory effect on breathing or myorelaxant effect [14, 15].

4. OBSTRUCTIVE SLEEP APNOEA IN SCHIZOPHRENIA

It is commonly known that sleep disorders accompany schizophrenia. Studies conducted on small groups of patients revealed evidence as high as 15 to 48%.Obesity is a major risk factor for sleep apnea and in schizophrenia it is twice as frequent as in the general population therefore it is assumed that the incidence of Sleep Apnea in patients with schizophrenia is higher than in the general population. In addition, there is evidence, that smoking and drinking alcohol increases the risk of Sleep Apnea and both behaviors are very common in people with schizophrenia [16].

The literature has also reported cases where in patients with residual schizophrenia and receiving the treatment of sleep apnea with CPAP caused a re-occurrence of acute psychosis with agitation, aggression and positive symptoms that required discontinuation of CPAP and intensification of antipsychotic treatment [17]. This is explained by increased dopaminergic activity or stimulation of GABAergic pathways that modulate cortical activity, and as a result of which there is increase of REM sleep and slow wave sleep after initiation of treatment with CPAP. Sleep Apnea in schizophrenia is a subject that requires attention because its effective treatment reduces cardiovascular risk, improves cognitive function, and hence, the quality of life of the patients.

5. SLEEP APNOEA IN DEPRESSION

Various studies have been undertaken regarding the links between sleep apnea and depression. The incidence of Sleep Apnea in patients with depression is higher than in the general population and reaches about 11-18% and the frequency of depression in patients with Sleep Apnea is estimated from 7 to as much as 63% [18]. Qhayon et al. conducted a study on 18,980 people from five countries with a telephone query and concluded that the relationship between major depression and Sleep Apnea remains significant even after excluding the impact of obesity and hypertension. People with diagnosed depression in a telephone interview were at five times higher risk of sleep apnea. Both sleep apnea and major depression were found in 0.8% of the surveyed people. 17.6% of people having one of these diagnoses also turned out to suffer from the other. Studies consider gender differences in the frequency of co-occurrence of depression in patients with Sleep Apnea, emphasizing higher women predilection [19].

There is important evidence that depressive symptoms in patients with Sleep Apnea should not be considered only as a consequence of respiratory disorder. A multicenter study on 300 patients with depression and OSA treated with CPAP found that CPAP therapy in many patients did not result in resolution of symptoms of depression.

6. PHARMACOLOGICAL AND NEUROPHYSIOLOGICAL ASPECTS OF SLEEP APNOEA IN DEPRESSION

According to the theory of neuroplasticity, chronic stress such as nocturnal hypoxia increases the levels of corticosteroids, which leads to atrophy of the hippocampal neurons and decreases the expression of brain-derived neurotrophic factor (BDNF), which is responsible for neurogenesis and long-term memory. This leads to a decreased mood and cognitive disorders

[20]. Hypoxia also produces hyperintense subcortical lesions. It also induces inflammation and the production of inflammatory cytokines which are an important component of the pathogenesis of depression. The inverse relationship that is depression as a risk factor of Sleep Apnoea can be explained by serotonergic neurotransmission. Low serotonin level is a major cause of disturbed sleep in depression and also a cause for reduction in muscle tone of the Upper Respiratory Tract. But no significant efficacy of antidepressants that increase serotonin level has been proven to be efficacious in the treatment of Sleep Apnea. This lack of response of Sleep Apnea to serotonergic drugs is explained by reduced neuronal excitability as a consequence of oxidative stress induced by hypoxia [21]. There are studies which suggest that in patients with sleep apnea who require sleep-promoting medication, sedative antidepressants are an important alternative to hypnotic drugs. A good example is trazodone, which significantly improves the quality of sleep, without worsening the Apnea-Hypopnea Index (AHI) factor.

7. BIPOLAR DISORDER

Very few studies have been undertaken on the coexistence of Sleep Apnea and bipolar disorder. There are studies which suggest that the presence of sleep apnea in patients with bipolar disorder may be as high as 21-47(5%) [22]. Benzodiazepines and atypical antipsychotics that are commonly used in bipolar disorder, increase sleep related breathing disorders and thus the co-existence of Sleep Apnea should be excluded before choosing such treatment.

8. CONCLUSION

Sleep-related breathing disorders often go undiagnosed in patients with psychiatric disorders. This increases their risk for cardiovascular diseases, worsens cognitive functions, increases the symptoms of the mental disorders, and can cause adverse health effects on patients taking psychiatric drugs by increasing severity of sleep apnea. Psychiatric hospitals often prove to be an important place for recognition and diagnosis of sleep apnea in patients with severe mental illnesses.

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