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FATIGUE AND SLEEPINESS IN PATIENTS WITH DEPRESSION – A SIGN TO LOOK FOR OBSTRUCTIVE SLEEP APNEA

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INTRODUCTION

There is a bidirectional relationship between psychiatric disorders and disturbances of sleep and circadian rhythms. For example, insomnia appears prior to (around 40%) or simultaneously (around 22%) with the symptoms in mood disorders and hypersomnia appeared in 46.5% in subjects with mental illness. Approximately two-thirds of depressed patients complain of insomnia, while 15% complain of hypersomnia (Abad & Guilleminault 2005). Depression according to WHO is the leading cause of disability worldwide, with around 300 million people affected and is a major contributor to the overall global burden of disease with women more often affected than men (WHO 2021). Among main complaints besides depressed mood are loss of energy, reduced activity and sleep difficulties (WHO 1993). Some patients complain about sleepiness and fatigue during the day, loss of energy and poor concentration that can mislead the psychiatrist to treat them as severe depressive episode, often with combined treatment due to lack of apparent treatment efficacy and worsening of symptoms.

In the clinical practice there is often an overlap between depression and other co-morbidities such as obstructive sleep apnea (OSA) among others, associated with several medical conditions, increased risk of accidents and overall healthcare expenditure. Although highly prevalent OSA is under diagnosed disease (Ejaz et al. 2011). It is a sleep and breathing disorder with excessive daytime sleepiness (EDS), snoring and witnessed apneas, cognitive problems, poor memory or depressive symptoms (Jones & Morel 2008). Prevalence studies in community and clinical populations have shown high rates of depression among people with OSA with wide variation in reported rates with range of 7-63% (Harris et al. 2009, Ejaz et al. 2011).

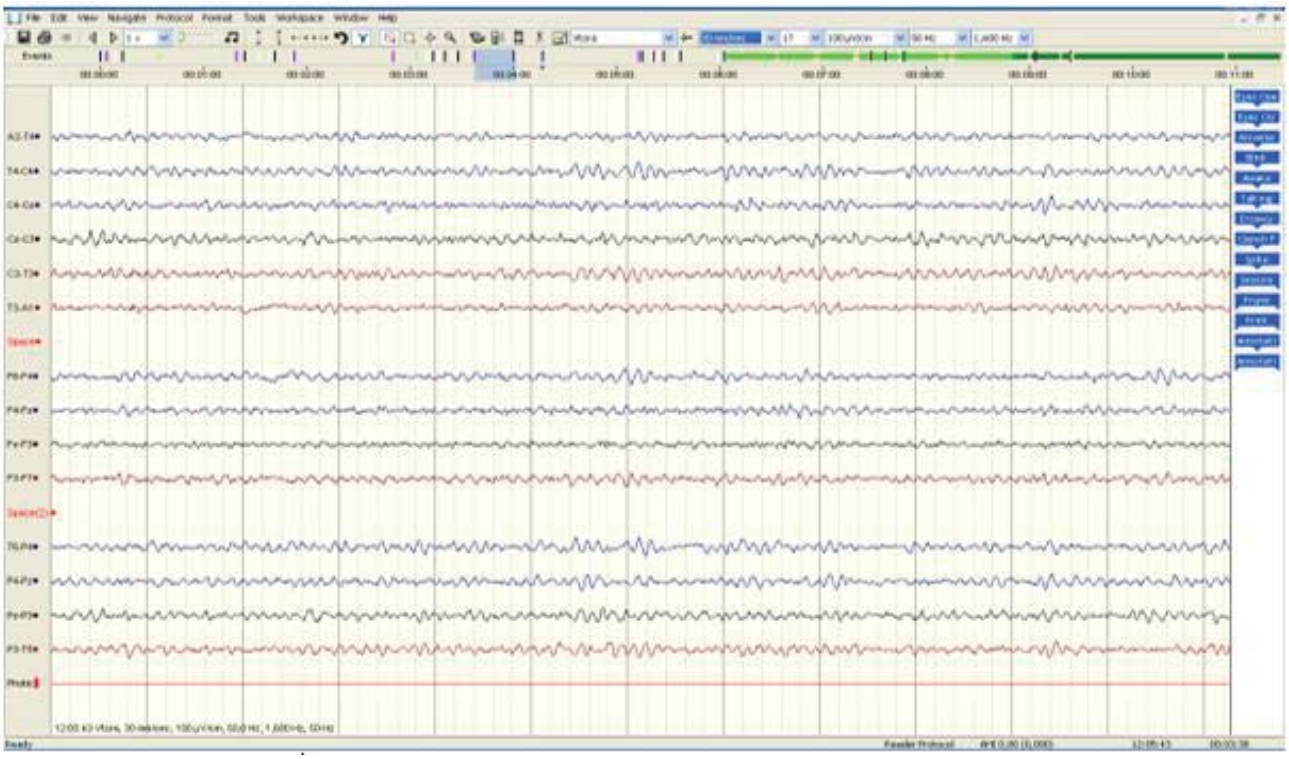

Figure 1. First EEg

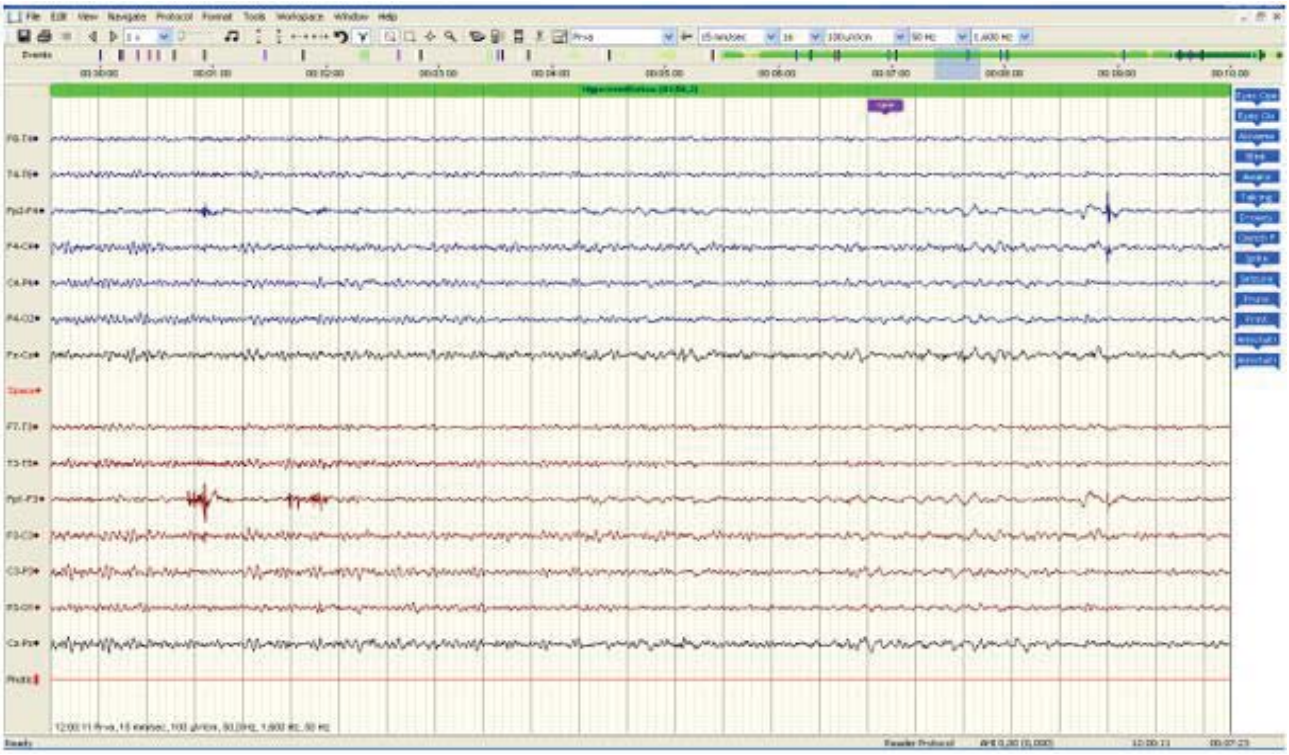

Figure 2. Second EEG

CASE VIGNETTE

Case report of 58 years old obese male patient, treated as chronic obstructive pulmonary disease. In the last three years because of anxiety, insomnia, worries about his health and headache, he was treated as depressive disorder with SSRI, SGA, anticholinergics and sedative neuroleptics. Brain neuroimaging (CT, MRI) revealed global cortical atrophy and micro-vascular changes. Actual psychiatric evaluation was because of hypersomnolence and dysarthria. Heteroanamnesis gave data for snoring. Performed Epworth Sleepiness Scale (ESS) was 20/24. EEG revealed theta/slow alpha activity (7-8Hz) and slow waves (Figure 1) and psychopharmacotherapy was ceased. After a month his mental health improved but sudden excessive daytime sleepiness was still present and ESS score was 14/24. Second EEG was with background activity around 8 Hz and elements of video recorded slow wave sleep patterns (Figure 2), pulse-oximetry was 92% (oxygen hyposaturation). Polysomnography results: severe snoring, without abnormal leg movements, sleep onset at 6.5 minutes without REM phase, sleep efficiency was 68.5% and total arousal index was 89.7/hour. Registered hypopneas/apneas were associated with average oxygen saturation of 91% and maximal reduction at 63% and respiratory disturbance index was 75.9/hour. Continuous positive airway pressure (CPAP) was introduced in the treatment.

DISCUSSION

OSA is chronic disorder caused by upper-airway collapse repeatedly occurring during sleep with recurrent asphyxia/hypoxia, fragmented night sleep and also fluctuations in blood pressure. This condition left untreated bare high risk for hypertension and other cardiovascular disease, cerebrovascular insults, car accidents and depression (Heinzer et al. 2015). In such cases there is a dilemma which disorder is first, because depressive patients also present symptoms of sleeping problems resulting in fatigue and sleepiness during the day. Patients with OSA because of the poor sleep have EDS but also can present anxiety and depressed mood, irritability or psychomotor retardation (Ejaz et al. 2011). The question is: should depressive symptoms associated with OSA be viewed as secondary to a primary medical condition or as an adjustment disorder with depressed mood (Baran & Richert 2003). Other investigators were interested whether depressive symptoms improve with treatment of OSA. There is a lack of longitudinal studies that will examine the temporal relationship between the two conditions and the role of specific confounders like obesity, gender etc. (Harris et al. 2009). Recently published studies that assess depression and anxiety in untreated OSA found that the symptoms overlap leads to reduction of anxiety scores and inflation of depressive scores in patients with OSA (Nanthakumar et al. 2017). Some authors think that there is co-morbidity between these two entities and this is important not only for the diagnosis but also for the adequate treatment. When OSA is primary disorder the necessary and efficacious treatment is continuous positive airway pressure (CPAP) (Jones & Morell 2008).

In our case report depression should be considered as secondary in OSA where severity correlates more with EDS than with hypoxemia (Saunamäki & Jehkonen 2007). This could lead to inadequate treatment and

worsening of the disease with psychopharmacotherapy because CPAP treatment is necessary and markedly improves depressive symptoms (Edwards et al. 2015). Because the main symptom during the day may be constant fatigue with day time sleepiness or depressive mood often the patients are first examined by psychiatrist and treated for their affective state and misperception that fatigue is a result of depressive episode. Often this is followed by treatment with psychopharmacotherapy possibly with selective serotonin reuptake inhibitors that could improve depressed mood but fatigue would persist because it is consequence of the OSA syndrome. As psychiatrists we are thinking that this is partial improvement of the depressive episode and so we try for another treatment, often with combination of anti-depressant and antipsychotic. That is why patients are often mistreated which could lead to worsening of symptomatology but in fact proper treatment with CPAP could rapidly improve the mental state of the patient.

CONCLUSION

Because OSA and depression have similar symptoms such as fatigue, daytime sleepiness, poor concentration, irritability and weight gain, often OSA is misdiagnosed if we don't ask the patients about snoring although this is recommended from sleep centers. In depressive patients with atypical clinical presentation we always need to exclude this co-morbidity. So there is a need for close cooperation between pulmonologists and psychiatrists, and proper diagnostics in sleep centers. Also education of medical students and doctors about sleep and breathing disorders is needed so they can recognize this condition promptly and treat the disorder effectively.

Contribution of individual authors:

Nensi Manusheva contributed to the creation of this paper with data collection, interpretation of data, literature search, study design, manuscript writing, first draft.

Dimitar Karkinski contributed to the creation of this paper with data collection, interpretation of data.

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