

CASE REPORT

Obstructive Sleep Apnoea (OSA) With Psychological Intervention in Geriatrics: A Complicated Case Study

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ABSTRACT

Background: Obstructive sleep apnoea (OSA) is a fragmented sleep disorder, characterized by snoring, frequent episodes of partial or complete cessation of breathing. It often results in a wide range of co-morbidities, predominantly the cardiovascular/respiratory, endocrine, and neuropsychiatric symptoms. In view of the ambiguity of literature regarding the association between OSA and depression, we conducted this study to show any association between the two disorders as it may co-exist. Thus, the quality of life of an individual's health can be improved by the early detection of the overlapping symptoms of OSA and depression. So, by addressing these issues early, the associated healthcare costs and burden can be reduced simultaneously. Case: A 68-year-old woman with a past medical history of psychotic depression, Type 2 DM and mild hypertension was admitted with persistent symptoms of depression accompanied by excessive daytime sleepiness. Type 2 DM was being treated with Metformin 500 mg and was given lifestyle interventions for the latter. She was referred to the sleep department due to the presenting symptoms as reported by the caretaker. We performed polysomnography (PSG) study to assess the condition. Psychiatric diagnosis was done using mini international neuropsychiatric interview plus scale. The aim of the study was to see the association between OSA and depression through a case report. Results: The depressive symptoms of the patient was improved through antidepressant & CBT while CPAP and anti-hypertensive therapy, ameliorated OSA along with hypertension. Conclusion: This study demonstrates the significant overlap between sleep apnoea and depression which often goes unnoticed resulting in delayed diagnosis and diminished quality of life. Health specialists need more information about screening for patients with OSA to ensure proper diagnosis and treatment of those with the condition. Thus, it is important to appropriately identify and treat OSA symptoms in depressive patients.

Keywords: Obstructive Sleep Apnoea, Psychological, Geriatrics, Depression, Diabetes, Arousal

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INTRODUCTION

The Obstructive Sleep Apnoea (OSA) is the most common sleep disorder worldwide, affecting 3- 7% of the adult population, increasing up to 18%–32% in those aged 70–100 years old.[1,2] OSA is more prevalent in men than in women and the risk increases with age, those with DM, depression and obesity. It is also observed in postmenopausal women. [1] It is a de-structured sleep disorder related to breathing, characterized by snoring, severe fragmentation of sleep and partial (hypopnoea) or complete (apnoea) cessation of breathing during sleep which decreases arterial oxygen saturation.[3] OSA can be defined by Apnoea Hypopnea Index (AHI) (the AHI is the combined average number of apnoeas and hypopnoeas per hour of sleep)[4]. An AHI of <5 is considered normal, 5 - 14 is mild, 15 - 29 is moderate, 30 and above is severe OSA.[5] Both apnoea and hypopnoea can cause sleep arousals and these arousals can simply cause the sleeper to shift into a lighter stage of sleep reducing the quality of sleep.[6] OSA also increases the risk for poor neuro-cognitive performance when intermittent hypoxemia and/or repeated arousals during sleep is persistent over months to years.[7] If the AHI is greater than 30 events per hour of sleep, there is an additional risks of mortality in patients with consequences of cardiovascular complications such as

hypertension, heart disease, stroke and road traffic accidents.[8] Depression and Obstructive sleep apnoea (OSA) are the major associated co-morbidities.[9] There is a higher prevalence of OSA in depression. Indeed, up to 63% of patients with depression report OSA. [10] But this association is underappreciated in medical practice and this can sometimes cause undesirable medical disorders that could otherwise be treated. It is highly recommended that primary care and mental health clinicians should be aware of this connection since sleepiness is one of the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) criteria of depressive disorder and it overlaps with the criterion of fatigue or loss of energy. Both of these problems can disguise each other because of their similar symptoms. [9] These diagnostic complexities along with the non-recognition of sleep disorder symptoms may lead to under-diagnosis of OSA and other sleep disorders in patients with depression and thus influence its treatment.[9]The most common and effective first line of treatment for OSA is Continuous Positive Airway Pressure (CPAP) therapy, which splints the pharyngeal airway space leading to improved oxygenation and decreased sleep fragmentation.[11]Other treatments includes weight loss, dental devices (which advance the tongue or mandible to increase posterior airway space) or upper airway surgery (e.g. combined tonsillectomy/ adenoidectomy, nasal reconstruction, and uvulopalatopharyngoplasty).[11]This case report is to increase the awareness of the possibility of OSA in patients with depression and to illustrate that the non-identification of OSA in a depressive patient can exert an influence on the process of treatment and/ or contributes to the anti- depressant therapy failure, thus highlighting the importance of assessment of OSA using a sleep study with polysomnography in a more regular basis.[12]

CASE REPORT

A 68-year-old woman was admitted in the department of geriatric psychiatry because of persistent symptoms of psychotic depression accompanied by sleepiness. The latter have been evaluated multiple times in the general practice over three years, each time it was considered to be a symptom of depression. The patient is from a middle-class socio- economic background and was the eldest of 2 children. She worked as an elementary school teacher and has no personal history of smoking, use of alcohol or other toxic substance abuse. She was diagnosed with type 2 diabetes at the age of 60 and is being medically treated with Metformin 500 mg/day. Also, the patient was monitored and lifestyle interventions were provided for mild hypertension by her General Practitioner. She had a family history of Type 2 DM and but no known history of psychiatric problems or neurodegenerative diseases like Parkinson's disease, Alzheimer's or Huntington's disease. Regarding the psychiatric antecedents she had two previous serious depressive episodes related to important life events. First episode happened at the age of 61 after her brother's suicide and the second depressive episode occurred 5 years later when her daughter died due to leukaemia. The patient was admitted to the psychiatry department with the complaints of depressed mood, feeling of worthlessness, helplessness & hopelessness, difficulty falling asleep at night, auditory hallucinations, increased need of sleep, loss of energy, loud snoring, morning headache, unintentional sleep episodes even while praying as reported by her husband. The symptoms fluctuated over years and she had gradually withdrawn from social activities after retiring from her job. The initial episode was alleviated following a brief course of anti-depressants for about 2 years (Amitriptyline 20 mg/day). Recently, after her daughter's death, she started to show psychotic symptoms like extensive auditory hallucinations, delusions, fatigue and subjective experience of forgetfulness and was diagnosed as psychotic depression. The treatment with Amitriptyline 20 mg/day was started again along with Quetiapine 25 mg/day and Risperidone 2 mg/day. Her depressive symptoms only improved partially with treatment, but was still not participative. When the patient was seen in the outpatient mental health department, she continued to have depressive symptoms. So, Amitriptyline was withdrawn and a new treatment with Dosulepin 75 mg/ day was started.

Investigation

In her next visit to our hospital, the depressive symptoms of the patient have been reduced. The scores on the Mini-Mental State Exam (MMSE) were not indicative of cognitive problems (MMSE: 30/30). She scored 35 points on the 30-item Inventory of Depressive Symptomatology (IDS).

But her husband complained of excessive daytime sleepiness and loud snoring. Based on this information, she was admitted in the hospital and additionally referred to a sleep specialist of our hospital.

Table no: 1. The Values For Vitals

| Parameters | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 |
|-----------------|---------|--------|---------|---------|--------|
| Temp (°F) | 98.6 | 98.6 | 98.6 | 98.6 | 98.6 |
| BP (mm Hg) | 150/100 | 150/94 | 162/100 | 150/100 | 140/90 |
| RR (per min) | 20 | 22 | 22 | 20 | 22 |
| Pulse (per min) | 76 | 72 | 78 | 75 | 76 |

Temp – Temperature, BP – Blood Pressure, RR -Respiratory Rate.

Table no: 2. Values for Lab Investigations

| PARAMETERS | NORMAL VALUE | Day 1 |
|----------------|------------------------------------|--------|
| Hb | 12- 16g/DL | 14.8 |
| DLC: P | 40-80% | 71 |
| L | 20-40% | 28 |
| E | 1-6% | 1 |
| Platelet Count | 1.5 – 4.5 L cells /mm ³ | 1.8 |
| TC | 4,000-11,000 cells/mm ³ | 12,600 |
| TSH | 0.4 – 4 uU/ L | 1.93 |

Hb – Hemoglobin, DLC -Differential Leucocyte Count, P – Polymorphs, L – Leucocytes, E – Eosinophils, TC – Total Count , TSH – Thyroid Stimulating Hormone.

Table No : 3 . Values for Blood Sugar

| Parameters | Day1 | Day 2 | Day 3 | Day 4 |
|------------|----------|----------|----------|----------|
| FBS | 153 mg % | 135 mg % | 128 mg % | 130 mg % |
| HbA1C | - | 9.6 % | - | - |

FBS – Fasting Blood Sugar, HbA1C – Glycated Hemoglobin

Table No: 4. Values for Liver Function

| Parameters | Day 1 |
|------------------------|-----------|
| SGOT | 101 U/L |
| SGPT | 128U/L |
| Serum Bilirubin Total | 1.5 mg/dL |
| Serum Bilirubin Direct | 0.2 mg/dL |
| Alkaline Phosphatase | 140 IU/L |
| Serum Protein | 7 g/dL |
| Serum Albumin | 3.5 mg/Dl |
| Serum Globulin | 2.5 mg/dL |

SGOT – Serum Glutamic Oxaloacetic Transaminase, SGPT – Serum GlutamicPyruvic Transaminase

Table No: 5. Values for Renal Function

| Parameters | Day 1 |
|------------------|-----------|
| Sodium (Na+) | 140mEq/L |
| Potassium (K+) | 3.0Eq/L |
| Serum Creatinine | 0.8 mg/dL |
| Blood Urea | 015 mg/dL |

TREATMENT

Dosulepin slightly improved her depressive and psychotic symptoms over the course of 6-7 weeks. But the OSA symptoms were still existing. The treatment goals for OSA includes reducing the number of apnoeic episodes, improvement of OSA symptoms and reduced oxygen saturation. Usually no pharmacotherapy is consistently effective in normalizing sleep in OSA patients. The patient was started with cognitive-behavioural therapy (CBT), to improve coping with her family situation and to increase her self-esteem which continued for 6 weeks. This was followed by nasal (CPAP) therapy at night to treat her OSA. Education about normal sleep and habits of good sleep hygiene was also provided.

CPAP therapy greatly improved the patient's functional status, by providing efficient oxygenation and restorative sleep, improving cognitive deficits and by raising the quality of life. Sleep apnoea can also be improved with some antidepressants due to their REM-suppressant activity, the sleep stage in which most apnoeas occur. Evidence shows that TCAs, SSRIs, Protriptyline and Imipramine has improved OSA with depressive symptoms causing a fewer adverse effects.

Outcome and Follow-up

Since starting Dosulepin, the depressive symptoms of the patient improved. With CBT, she became more interactive with family members and her self-image became more positive. Over the course of 2 months, prior to the start of CPAP, her depressive symptoms improved further. After 2 weeks of CPAP therapy, OSA symptoms of the patient reduced and she felt more energetic and started rebuilding her life. CPAP therapy along with antihypertensive also improved the nocturnal and daytime BP levels. Full recovery was achieved after 4 months of therapy.

DISCUSSION

Although the symptoms may point to depression, OSA should be assessed by AHI using polysomnography which is the gold standard for proper diagnosis. An AHI of 5–14 per hour indicates a mild, 15–30 a moderate and ≥ 30 a severe OSA.[10] Apnoea can cause sleep arousals and a fragmented sleeping pattern. Night-time symptoms include snoring, breathing pauses, a feeling of choking, excessive salivation, excessive sweating, gastro-oesophageal reflux, nocturia, waking up with a dry mouth and/or even a headache.[13,14] Patients wake up feeling unrefreshed and during the day, the patient experiences excessive sleepiness, loss of energy, irritability[15] withdrawal of social activities, difficulties in concentrating [16] cognitive dysfunction[17] loss of interest in daily activities[18] temper issues, [16] psychomotor changes[18] anxiety and depressed mood.[19] These symptoms bear a striking resemblance to the symptoms of depression.[14] OSA can sometimes induce oxidative stress and inflammation to the major organs which in turn increases the levels of corticosteroids in the body and hence worsen the sleep and decreases the quality of a sound sleep.[20,21] OSA, depression, and cardiovascular diseases are associated with an increased levels of various pro-inflammatory mediators and cytokines.[16,22] Some of these markers play a major role in between depression and coronary artery disease.[16] Interleukin 6 (IL6) and tumour necrosis factor (TNF) are thought to be responsible for increased daytime sleepiness.[23] TNF is found to be increased in patients with depression.[24] Different patients respond differently to the same dose of a drug and sometimes even treatment resistance may also be observed in those with phenotypic variations in the metabolising liver enzymes.[25] As we know, liver is a major drug metabolising organ certain changes may alter the proper functioning of the enzymes within it as in case of antidepressant therapy, CYP2D6 is responsible for metabolism. Phenotypic variation of CYP2D6 may result in rapidly metabolising enzymes which causes decreased plasma concentration of drug resulting in therapeutic failure. Most sleep clinics should therefore regularly evaluate OSA symptoms of their patients. The similarity in phenotypic expression of OSA and depressive disorders results in misdiagnosis of sleep apnoea for a depressive disorder and wrongfully treats only with antidepressants. In our case, the OSA symptoms accompanying depression have been wrongfully attributed to a depression and have been only treated with antidepressants. When her family problems due to OSA triggered a full-blown depressive disorder, she was referred for specialised mental healthcare. With reference to this case the depressive disorders and sleep disorders co-occur more often and the association is considered to be bidirectional as depression can evoke sleep disturbances.[26,10] Studies have shown that 90% of patients with major depression, experience some kind of sleep problem.[27] Several authors have advised screening for OSA in psychiatric patients, as this will accelerate appropriate diagnosis. Especially in patients with treatment resistant depression, OSA must be considered. Our patient noticed some improvement after starting on Dosulepin and starting CBT, but the most significant improvement was seen following CPAP therapy. Aside from the improving psychiatric symptoms, the treatment of OSA may reduce the risks of cardiovascular diseases. The hypertension our patient presented with might be related to her OSA. Attributing symptoms of OSA to a psychiatric disorder may result in inappropriate prescriptions of antidepressants, as was the case in our patient. Adjunct treatment to antidepressant medication such as BZDs and hypnotics can exacerbate OSA by decreasing the muscle tone in the already functionally impaired upper airway dilator muscles, blunting the arousal response to hypoxia and hypercapnia as well as increasing the arousal threshold for the apnoeic event, therefore increases the number and duration of apnoeas. These effects might differ depending on the patient population and the severity of OSA. Older depressive subjects are of primary concern: both, frequency of OSA and depressive symptoms increase with age, as do prescription and

consumption of sedative psychotropic medication.[14] But with the right treatment and self-help strategies, however, we can control OSA symptoms during depression.

With this case report, we emphasize the importance of diagnosing and treating OSA in patients with a depressive disorder. As illustrated by our patient, proper diagnosis and increased awareness of the relationship between depression and OSA may improve quality of life by alleviating the co-morbid diseases and prevent misdiagnosing affective symptoms as a treatment-resistant depression.

CONCLUSION

Both OSA and depression are common co-existing diseases with a serious impact on health. The presence of OSA is greater in those with depression as compared to the normal population. The most common coinciding symptoms of both depression and OSA includes snoring, breathing pauses, a feeling of choking, increased sweating and salivation, GERD, increased urination at night, waking up with a dry mouth and/or a frequent debilitating headache. Moreover, OSA places the patient at a greater risk of other disorders like cardiovascular disease. In addition, other contributing factors, like diabetes, hypertension, and cardiovascular disease, may worsen the relationship between depression and OSA. It is very commonly seen in those with diabetes but more often it remains undiagnosed. OSA can cause hypertension, but hypertension is not required for prediction of sleep apnoea. To an extent insulin resistance play a role in the pathogenesis of major depressive disorder and has been predicted to possess association between depression and other cardiovascular disease related to that with OSA. Treating sleep apnoea with CPAP therapy can improve both blood pressure as well as glycaemic index. So, a proper diagnosis is required to prevent further complications of other systems in the body since delayed diagnosis of OSA will definitely diminish the patient's quality of life.

Treatment with antidepressants along with good sleeping techniques can improve OSA in depression. Despite the fact that antidepressant drugs improve overall mood, it can deteriorate sleeping pattern and also poor sleep acts as a symptom and a cause of depression. No single antidepressant is proved to be good for sleep, but each has features that can help or harm the patient's sleep disorder. Choosing the perfect drug can improve daytime performance and lead to a complete recovery.

ABBREVIATIONS

AHI : APNOEA – HYPOPNOEA INDEX ; BP : BLOOD PRESSURE; BZD : BENZODIAZEPINES ; CBT : COGNITIVE BEHAVIOURAL THERAPY; CPAP : CONTINUOUS POSITIVE AIRWAY PRESSURE ; CYP2D6 : CYTOCHROME P450 2D6; DM : DIABETES MELLITUS; DSM : DIAGNOSTIC AND STATISTICAL MANUAL GERD : GASTROESOPHAGEAL REFLUX DISEASE ; IDS : INVENTORY OF DEPRESSIVE SYMPTOMATOLOGY IL- 6 : INTERLEUKIN 6 ; MMSE : MINI- MENTAL STATE EXAM ; OSA : OBSTRUCTIVE SLEEP APNOEA; PSG : POLYSOMNOGRAPHY ; REM : RAPID EYE MOVEMENT ; SSRI : SELECTIVE SEROTONIN REUPTAKE INHIBITOR; TCA : TRICYCLIC ANTI- DEPRESSANT; TNF : TUMOUR NECROSIS FACTOR

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CONFLICT OF INTEREST

The authors found that there is no conflict of interest to declare.

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