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High Risk of Obstructive Sleep Apnea in Hemorrhagic Stroke Patient

Rossy Triana[1], Desak Ketut Indrasari Utami[2]

[1]Department of Neurology, [2]Department of Neurology,
Udayana University/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

Corresponding Author: Rossy Triana

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ABSTRACT

Background: Stroke is one of the biggest causes of mortality and morbidity in the world, about 10% - 20% are caused by hemorrhagic strokes. Obstructive Sleep Apnea (OSA) is a sleep disorder that is often found in 5-15% of the general population and has a high prevalence in patients with cerebrovascular disease.

Case summary: A 51 year old female with a sudden loss of consciousness since 1 day ago, while working. The patient has a history of hypertension since 2014. On computerized tomography scan (CT scan) of the head without contrast shows bleeding. During hospitalization, it appears that the patient snoring, easily falls asleep during the day and there are periods of apnea when the patient sleeps. The blood gas analysis was normal. The patient's sleep score shows high risk of OSA.

Discussion: Patient with a hemorrhagic stroke and high risk OSA. There are several risk factors that influence the emergence of OSA in this case, obesity and hypertension. The therapy is to prohibit positioning, sleep hygiene, and losing weight.

Conclusion: We have reported a case of hemorrhagic stroke with a high risk of OSA that was experienced long before. The high risk of OSA in these patients can be a risk factor for hemorrhagic stroke. Polysomnography examination needs to be done in patients with a high risk of OSA, as the gold standard. Management of these patients apart from giving pharmacological therapy combined with behavioral and positional therapy to treat OSA symptoms. Mandibular enhancement devices and continuous positive airway pressure (CPAP) have not been given to this patient because polysomnography has not been performed and cost constraints.

Keywords: hypertension; hemorrhagic stroke; obesity; OSA

INTRODUCTION

Sleep is a physiological and recurring form of reversible loss of consciousness in which there is a global decline in cognitive function so that the brain does not fully respond to surrounding stimuli.[1]

Obstructive Sleep Apnea Syndrome (OSAS) is one of the most common sleep disorders. OSAS can be characterized by obstruction of the upper airway, which can cause excessive daytime sleepiness, oxygen desaturation and nighttime awakenings. OSAS is often associated with an increased risk of morbidity and mortality from cardiovascular, cerebrovascular, diabetes and cognitive impairment.[2]

CASE REPORT

Female, 51 years old, Balinese, with decreased consciousness, occurred suddenly the day before entering Prof Ngoerah General Hospital, when the patient was working. It was said that at that time, the patient vomited more than 1 time accompanied by severe headaches. While in the emergency room, it was seen that parts of the patient's left body were more inactive and spoke sluggishly. The patient has a history of hypertension and has not taken medication since 2014.

During treatment, it appears that the patient sleeps snoring and easily falls asleep during the day. According to the family, the patient has been snoring for the last 10 years. In addition, there was a period of stopping breathing in this patient which was witnessed and recorded by the patient's family.

On physical examination, blood pressure was 180/100 mmHg. From nutritional status, the upper arm circumference was 35 cm, height 160 cm, approximate body weight 81.75 kg, with a body mass index (BMI) of 31.91 kg/m (the impression of being obese class I). During examination, Glasgow Coma Scale E2V2M5, supranuclear left facial nerve paresis, supranuclear left hypoglossal nerve paresis, flaccid left lateralization grade less than 3 and Babinski reflex dextra et sinistra. The patient's sleep scores included PSQI 6 (poor sleep quality), STOP Bang 4 (High risk of OSA), Berlin 3 positive categories (most likely sleep apnea), Snoring score 5 (high risk of obstructive sleep apnea), and Epworth 11 (light drowsy). From the results of blood gas analysis, the impression was normal (pH 7.4, pO2 119 mmHg, pCO2 40 mmHg, HCO3- 24.8 mmol/L, BEecf 0 mmol/L). Electrocardiography revealed left ventricular hypertrophy. The results of the chest X-ray examination suggest cardiomegaly. On CT scan of the head without contrast, it suggests bleeding in the thalamus, intraventricular hemorrhage, and non-communicating hydrocephalus.

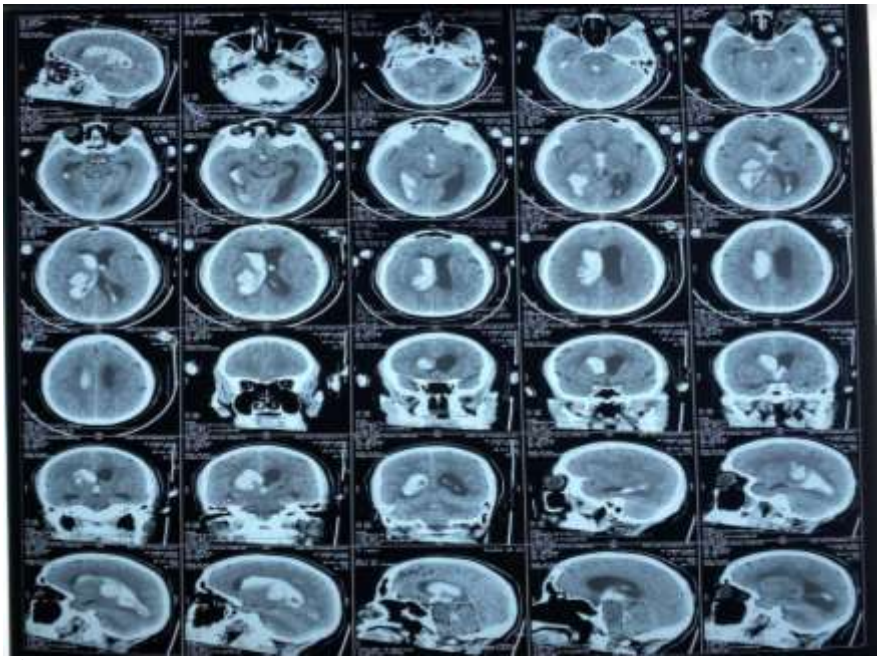

Figure 1. CT scan of the head without contrast. There are hyperdense lesions in the thalamus and intraventricular and non-communicating hydrocephalus.

Furthermore, the patient is given Kocher S ventriculoperitoneal shunt by the neurosurgery colleague, supportive management, nutrition and medical rehabilitation while in the hospital. As for high risk OSA symptoms in these patients, from the sleep division they were taught positioning (sleeping on their side, avoid sleeping on their backs), sleep hygiene, and losing weight.

DISCUSSION

Obstructive Sleep Apnea Syndrome (OSAS) is a sleep disorder that is quite common, but is not widely known in Indonesia. This disease can occur in 3-7% of men and 2-5% of women in the general population.[3]

Obstructive sleep apnea (OSA) is characterized by recurring episodes of cessation (apnea) or reduction (hypopnea) in airflow during sleep caused by obstruction of the upper airway. About 25% of patients

with OSA report daytime sleepiness; a greater proportion report unrefreshing sleep or fatigue. Other symptoms include frequent nocturnal waking due to choking or gasping, nocturia, morning headaches, poor concentration, irritability and erectile dysfunction. Bed partners may report snoring or witnessed apneas. Atypical symptoms, which are more frequently reported by women, include insomnia, impaired memory, mood disturbance, reflux and nocturnal enuresis.[4]

During sleep, muscle tone throughout the body decreases and pharyngeal dilator muscles relax. In OSA sufferers, there is severe upper airway narrowing during inspiration which results in turbulent currents and vibrations that cause snoring. Arousal restores upper airway dilator muscle tone and the patient takes a sudden gasping breath, takes several deep breaths, and then drifts back to sleep. At that time there is relaxation of the dilator muscles again and the above cycle is repeated. This can occur hundreds of times a night, causing disturbances during the day which is a clinical symptom of this disease.[1]

Several risk factors are associated with OSA and include the following:[1,5]

1. Older age. Loss of muscle mass due to the aging process
2. Male sex. OSA is approximately two to three times more common in males than females, although the risk appears to be similar once females are peri- and postmenopausal.
3. Obesity. The risk of OSA correlates well with the body mass index (BMI). In one study, a 10 percent increase in weight was associated with a six-fold increase in risk of OSA. Fat accumulation in the upper airway will cause narrowing and tends to close when the muscles are loose
4. Craniofacial and upper airway abnormalities. Examples of abnormalities include an abnormal maxillary or short mandibular size, a wide craniofacial base, and tonsillar and adenoid hypertrophy, the latter being common in children.
5. Smoking. Current smokers were nearly three times more likely to have OSA than past or never smokers. Smoking causes inflammation, swelling and narrowing of the airways
6. Family history of snoring or OSA. It has been suggested that about 40 percent of the variance of the AHI has a genetic basis.
7. Alcohol and sedatives cause the muscles of the upper airway to relax

Polysomnography is the gold standard in diagnosing OSA. Polysomnography includes recording airflow, breathing movements, EEG, EMG, EOG EKG, oxygen saturation and body position. Ideally Polysomnography is done in a sleep laboratory for a full night and monitored by a doctor/nurse. The results that appear are the number of respiratory stops per hour, the apnea-hypoapnea index (AHI).[1]

Table 1. Diagnostic criteria for obstructive sleep apnoea, adult (adapted from ICSD-3) [6]

(A and B) or C satisfy the criteria
A. The presence of one or more of the following:
1. The patient complains of sleepiness, non-restorative sleep, fatigue or insomnia symptoms.
2. The patient wakes with breath holding, gasping or choking.
3. The bed partner or other observer reports habitual snoring, breathing interruptions or both during the patient's sleep
4. The patient has been diagnosed with hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation or type 2 diabetes mellitus
B. Polysomnography (PSG) or out-of-centre sleep testing (OCST) demonstrates:
1. Five or more predominantly obstructive respiratory events [obstructive and mixed apnoeas, hypopnoeas or respiratory effort-related arousals (RERAs)] per hour of sleep during a PSG or per hour of monitoring (OCST)
or
C. PSG or OCST demonstrates:
1. Fifteen or more predominantly obstructive respiratory events (apnoeas, hypopnoeas or RERAs) per hour of sleep during a PSG or per hour of monitoring (OCST)

Classification of OSA[7]

1) Mild OSA: (apnea–hypopnea index [AHI] of 5–15) – Involuntary sleepiness

at times of activities that demand little attention, such as watching TV or reading.

2) Moderate OSA: (AHI of 15–30) – Involuntary sleepiness during activities that need some attention, such as meetings or presentations.
3) Severe OSA: (AHI of >30) – Involuntary sleepiness during activities that need more attention such as talking or driving.

Treatment options for OSA include medical, behavioral and surgical therapies. There are several recommendations were obtained for the non-surgical management of OSA:[8]

1. Recommendation 1: overweight and obese patients diagnosed with OSA should be encouraged to lose weight. (Grade: strong recommendation; low-quality evidence)
2. Recommendation 2: continuous positive airway pressure treatment as initial therapy for patients diagnosed with OSA. (Grade: strong recommendation; moderate-quality evidence)
3. Recommendation 3: mandibular advancement devices as an alternative therapy to continuous positive airway pressure treatment for patients diagnosed with OSA who prefer mandibular advancement devices or for those with adverse effects associated with continuous positive airway pressure treatment. (Grade: weak recommendation; low-quality evidence).

In this case, there are several risk factors that influence the emergence of OSA, namely obesity and hypertension. Based on the ICSD-3 diagnostic criteria, the patient met 3 symptoms of OSA, that is light sleepiness during the day, snoring, and diagnosed with hypertension. In this patient, polysomnography was not performed because the facilities for polysomnography were not yet available. According to several assessments of sleep disorders examined, Pittsburgh Sleep Quality Index (PSQI) 6 (poor sleep quality), STOP Bang 4 (high risk of OSA), Berlin Questionnaire (BQ) 3 positive categories (most likely sleep apnea), Snoring score 5 (high risk of obstructive sleep apnea), and Epworth Sleepiness Scale 11 (mild sleepiness). The management of OSA given to this patient is behavior therapy by losing weight and positional therapy (sleeping on the right and left sides, avoid sleeping on your back). Other treatments such as mandibular advancement devices and CPAP have not been given to these patients because polysomnography has not been performed and cost constraints.

OSA is an independent risk factor for intracerebral hemorrhage (ICH). Possible roles of OSA in the pathogenesis of stroke include sympathetic activation, inflammation and endothelial dysfunction that lead to atherosclerosis. Due to the high incidence of OSA and its potential effect on morbidity and mortality, examinations to diagnose and treat OSA are recommended for stroke patients.[9]

Untreated patients with comorbid OSA may have worse functional outcomes and higher mortality after acute stroke. Several observational studies suggest that OSA is a predictor of poor functional outcome after stroke, increasing the likelihood of dependency and poststroke mortality. Potential mechanisms of OSA contributing to poor neurologic recovery include direct effects of reduced cerebral blood flow and modulation of blood pressure and oxygen saturation associated with apneic episodes, resulting in further neurologic injury due to a compromise in perfusion to the ischemic penumbra.[10]

CONCLUSION

In the case of a woman, 51 years old, Balinese, right with a high risk of OSA in hemorrhagic stroke. In this case a CT scan of the head was performed without contrast suggesting bleeding in the thalamus, intraventricular hemorrhage, and non-communicating hydrocephalus. There are several risk factors that influence the emergence of OSA in these patients, namely obesity and hypertension. Several

assessments of sleep disorders have been carried out and obtained PSQI 6 (poor sleep quality), STOP Bang 4 (High risk of OSA), Berlin 3 positive categories (most likely sleep apnea), Snoring score 5 (high risk of obstructive sleep apnea), and ESS 11 (mild drowsiness). The patient underwent a Kocher S ventriculoperitoneal shunt by neurosurgery colleagues and pharmacological therapy for anti-hypertension. As for the management of OSA in this patient, namely behavioral therapy by losing weight and positional therapy (sleeping on the right and left sides, avoid sleeping on your back). In the follow-up of this patient, an improvement was found in the clinical condition but snoring was said to still exist.

Based on the cases raised in this case report, the important thing that can be used as learning is that in patients with high risk OSA, it is necessary to carry out further examinations, namely polysomnography (PSG) to establish a diagnosis of OSA and comprehensive management to prevent recurrent strokes.

Declaration by Authors
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