Asia-Pacific Journal of Science and Technology: Volume: 21. Issue: 04. Article ID.: APST-21-04-01. Research Article

APST

Asia-Pacific Journal of Science and Technology

https://www.tci-thaijo.org/index.php/APST/index

Published by the Faculty of Engineering, Khon Kaen University, Thailand

CPAP therapy in a young hypertension patient

Vichai Senthong[1,*], Chalongchai Phitsanuwong[2]

[1] Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand
[2] Colleges of Medicine, University of Chicago, Chicago, USA
*Corresponding author: vichaisenthong@gmail.com

Received June 2016
Accepted July 2016

Abstract

Obstructive sleep apnea (OSA) is a common cause of hypertension in adults. However, data regarding OSA are limited in hypertensive patients aged less than 40 years. Here, we reported clinical features and outcomes of OSA induced hypertension in a young patient. A 24-year-old man presented with epistaxis from high blood pressure for one day. He was at risk for OSA by having high body mass index of 29 kg/m2, retrognathia, and Mallampati class 3. There was no evidence of other causes of hypertension. The diagnosis of OSA was confirmed by polysomnography with an apnea-hypopnea index of 10 times/hour. A continuous positive airway pressure (CPAP) machine was successfully introduced. The apnea-hypopnea index was lower to 2.8 times/hour with CPAP therapy. He was complied with CPAP resulting in normal blood pressure (120/70 mmHg). No antihypertensive medication was required. In conclusion, OSA can be the cause of hypertension in young patients. CPAP therapy alone may be the effective therapy to control hypertension without any antihypertensive medication..

Keywords: hypertension patient; obstructive sleep apnea; continuous positive airway pressure

1. Introduction

Obstructive sleep apnea (OSA) has been defined as a cause of hypertension by the JNC 7 since 2003 [1]. The prevalence of OSA in hypertension cases is approximately 50% and may be up to 71% in resistant hypertension studies [2],[3]. Persons with an apnea-hypopnea index (AHI) of more than 5 times/hour increase risk of hypertension by three times and hypertension may develop in four years or more after having OSA [4] & [5].

Hypertension in the young is defined as the occurrence of hypertension in persons of 40 years or less in age [**Error! Bookmark not defined.**]. The prevalence of OSA in young patients with hypertension is unknown. Here, we report a young patient with hypertension with regards to cause for treatment, treatment, and result of treatment.

2. A case history

A 24-year-old man presented with hypertension and epistaxis for one day. The patient had previous history of high blood pressure on clinic visits but received no medication. Physical examination revealed a body mass index of 29.3 kg/m^2, blood pressure of 150/90 mm Hg, no thyroid gland enlargement, retrognathia, Mallampatic class 3, normal heart, no renal bruit, negative Bimanual palpitation, and all normal and equal peripheral pulses. On nasal endoscopy, there was blood clot at right nasal vestibule without active bleeding. Enalapril 5 mg and metoprolol (100 mg) were prescribed.

The patient was sent to the sleep/hypertension clinic for further workups due to the onset of hypertension at a youthful age. Polysomnography was performed and showed an AHI of 10 times/hour with lowest desaturation of 75%. A Continuous Positive Airway Pressure (CPAP) machine was introduced.

The patient was able to maintain the auto-CPAP with a compliance rate of 80%; average hourly rate used was 5 hrs and 44 mins; and 90% pressure of 8.4 cm H_2O. The residual AHI was 2.8 times/hour and the CPAP pressure pattern was compatible with OSA (Figure 1). Echocardiogram revealed a normal size of the left ventricle but moderate concentric LV hypertrophy was noted. No albuminuria was detected. After CPAP treatment, the blood pressure was normal (120/70 mmHg) and no antihypertension medication was required.

3. Discussion

The two main conclusions derived from this patient are 1) OSA is a cause of hypertension in young patients and 2) CPAP therapy may successfully control blood pressure in young hypertension patients with OSA.

OSA induced hypertension can be explained by several mechanisms including stimulations of a sympathetic nervous system and a renin-angiotensin-aldosterone system [6],[7]. These abnormalities occur from intermittent hypoxemia during apneic events. The blood pressure pattern observed in the OSA patient while sleeping was a non-dipping pattern [8]. Even though there is limited data of these mechanisms in young patients with hypertension, I believe that similar mechanisms contribute to hypertension in young patients with OSA.

A previous case-control study showed that OSA is associated with hypertension in young patients whose ages range between 18 years to 40 years [9]. Risk factors of hypertension in these young patients depend on severity of OSA. Mild and severe OSA increase risk of hypertension by 2.76 and 7.94 times, respectively. Our patient is diagnosed as secondary hypertension from mild OSA (AHI of 10 times/hour). Note that other causes of hypertension should be excluded in all patients with hypertension regardless of onset of hypertension.

There are three to four main risk factors for OSA; obesity, age/gender, craniofacial abnormalities, and co-morbid diseases [10]. Even though obesity is the main risk factor of OSA, only 36.6% of OSA patients are obese in the Thai population [11]. Additionally, older adult patients with OSA seem to have lower body mass indexes than younger adult patients with OSA [12]. The patient in this study may have OSA from retrognathia and obesity. It is important to remind clinicians that OSA induced hypertension in young patients may occur in non-obese patients.

A meta-analysis showed that CPAP therapy significantly reduced both systolic and diastolic blood pressure by 2.32 and 1.98 mm Hg, respectively [13]. Additionally, blood pressure patterns may change from non-dippers to dippers. The non-dipper pattern is associated with cardiovascular consequences including acute coronary syndrome, stroke, heart failure or arrhythmia and sudden death [14]. In this type of young hypertension patient, CPAP therapy can successfully control the blood pressure and no medication is required. Note that the compliant rate of CPAP should be 100%. I advised the patient to stop both anti-hypertension medications after CPAP therapy due to normal left ventricular size and no albuminuria. Another lesson learned from the CPAP graph of the patient is that the auto-CPAP pattern is compatible with OSA. As mentioned by the 2014 stroke guideline, auto-CPAP titration may be used as a diagnostic test for OSA [15]. In a resource-limited setting, auto-CPAP titration is therefore feasible to diagnose OSA in both stroke and young hypertension patients. Further studies are needed to confirm these claims.

In this patient, long term follow-up is needed to ensure that the blood pressure is well controlled. Repeat echocardiography and albuminuria are needed to monitor the long term effects from both OSA and hypertension. However, CPAP therapy is shown to improve left ventricular function and reduce left ventricular/right ventricular volume [16] & [17].

In conclusion, OSA can be the cause of hypertension in young patients and CPAP therapy may be the applicable therapy for successful treatment.

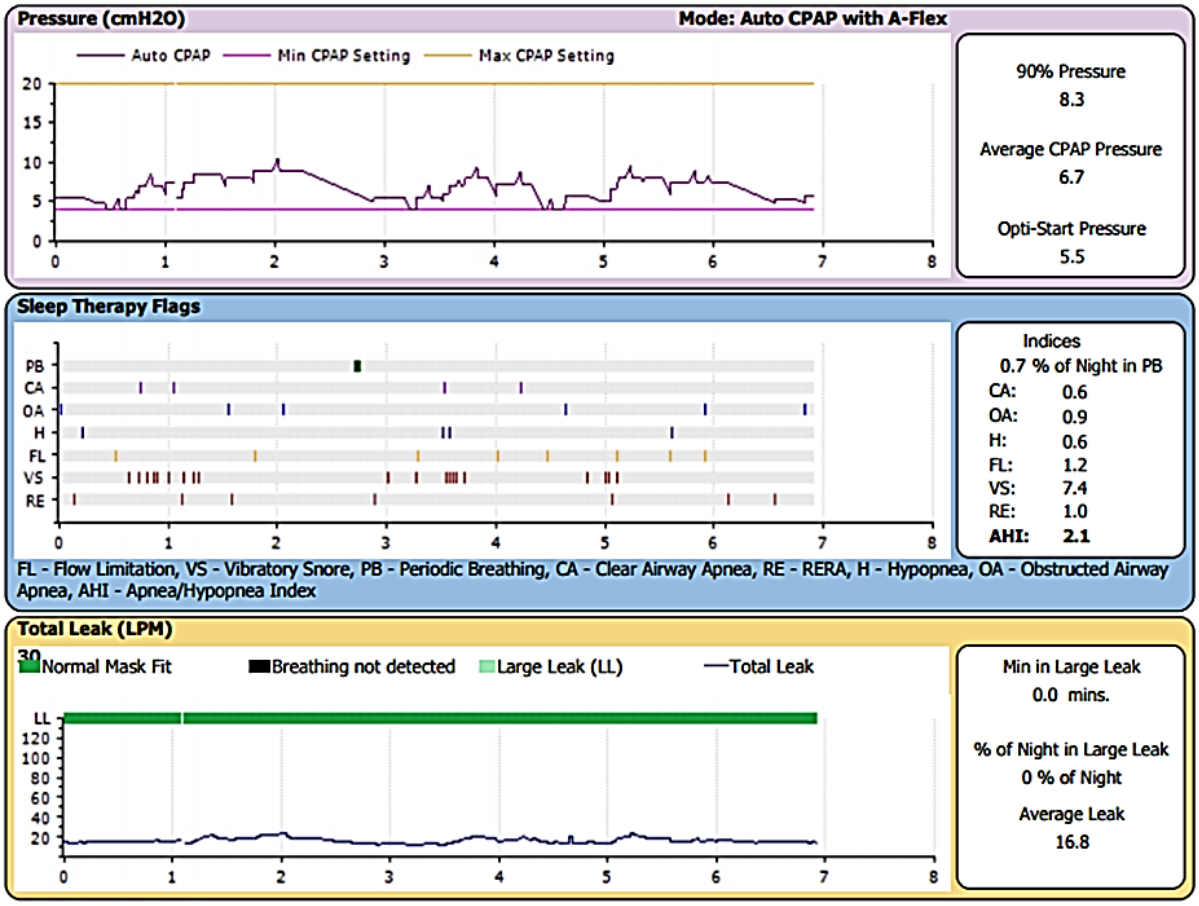

Figure 1 The auto-CPAP graph shows patterns of obstructive sleep apnea.

4. References

[1] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7report. JAMA. 2003;289(19):2560-72.

[2] Silverberg DS, Oksenberg A. Are sleep-related breathing disorders important contributing factors to the production of essential hypertension? Curr Hypertens Rep. 2001;3(3):209-15.

[3] Gonçalves SC, Martinez D, Gus M, de Abreu-Silva EO, Bertoluci C, Dutra I, et al. Obstructive sleep apnea and resistant hypertension: a case-control study. Chest. 2007;132(6):1858-62.

[4] Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med. 2000;342(19):1378-84.

[5] Chen BY, He QY. [A multi-center study on the association between sleep apnea and prevalence of hypertension]. Zhonghua Jie He He Hu Xi Za Zhi. 2007;30(12):894-7.

[6] Dopp JM, Reichmuth KJ, Morgan BJ. Obstructive sleep apnea and hypertension: mechanisms, evaluation, and management. Curr Hypertens Rep. 2007;9(6):529-34.

[7] Wolk R, Shamsuzzaman AS, Somers VK. Obesity, sleep apnea, and hypertension. Hypertension. 2003;42(6):1067-74.

[8] Wolf J, Hering D, Narkiewicz K. Non-dipping pattern of hypertension and obstructive sleep apnea syndrome. Hypertens Res. 2010;33(9):867-71.

[9] Asha'ari ZA, Hasmoni N Rahman J, Yusof RA, Ahmad RA. The association between sleep apnea and young adults with hypertension. Laryngoscope. 2012;122(10):2337-42.

[10] Lurie A. Obstructive sleep apnea in adults: epidemiology, clinical presentation, and treatment options. Adv Cardiol. 2011;46:1-42.

[11] Chirakalwasan N, Teerapraipruk B, Simon R, Hirunwiwatkul P, Jaimchariyatam N, Desudchit T, et al. Comparison of polysomnographic and clinical presentations and predictors for cardiovascular-related diseases between non-obese and obese obstructive sleep apnea among Asians. J Clin Sleep Med. 2013;9(6):553-7.

[12] Sawanyawisuth K, Chindaprasirt J, Senthong V, Makarawate P, Limpawattana P, Domthong A, et al. Lower BMI is a predictor of obstructive sleep apnea in elderly Thai hypertensive patients. Sleep Breath. 2013;17(4):1215-9.

[13] Hu X, Fan J, Chen S, Yin Y, Zrenner B. The role of Continuous Positive Airway Pressure in blood pressure control for patients with

obstructive sleep apnea and hypertension: a meta-analysis of randomized controlled trials. J Clin Hypertens (Greenwich). 2015;17(3):215-22.

[14] Sousa F, Neves J, Ferreira R, Polonia J, Bastos JM. 1B.05: In hypertension the change from a non-dipper to a dipper pattern is associated with a better cardiovascular prognosis than the persistence within the non-dipper pattern. J Hypertens. 2015;33(S1):e6.

[15] Kernan WN, Ovbiagele B, Black HR, Bravata DM, Chimowitz MI, Ezekowitz MD, et al. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45(7):2160-236.

[16] Sun H, Shi J, Li M, Chen X. Impact of continuous positive airway pressure treatment on left ventricular ejection fraction in patients with obstructive sleep apnea: a meta-analysis of randomized controlled trials. PLoS One. 2013;8(5):e62298.

[17] Mehta S, Liu PP, Fitzgerald FS, Allidina YK, Douglas Bradley T. Effects of Continuous Positive Airway Pressure on cardiac volumes in patients with ischemic and dilated cardiomyopathy. Am J Respir Crit Care Med. 2000;161(1):128-34.