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White coat hypertension and obstructive sleep apnea

Li Li Li-zhu Guo*

Cardiovascular Center, Beijing Tong Ren Hospital, Capital University of Medical Sciences, 100730 Beijing, China

Mini Review

White coat hypertension (WCH) was presented first time by Pickering in1988 and is characterized by elevated office BP but normal reading when measured outside the office with either ABPM or HBPM [1]. The prevalence of WCH is from 13% to 35% in different populations [2,3]. Although most studies have shown scarcely difference of all-cause mortality or CVD risks between WCH and normotensive people [4], WCH patients have a 1.5–2.0-fold higher risk of developing sustained hypertension than normotensive controls after 10 years [5], especially for those people who are older, obesity, or black race [3,6]. Obstructive sleep apnea (OSA) is characterized by recurrent collapse of the upper airways during sleep, inducing intermittent episodes of apnea/hypopnea, hypoxemia, and sleep disruption [7], and is one of the risk factors for cardiovascular diseases, such as hypertension, coronary heart disease and cerebrovascular diseases, heart failure and atrial fibrillation [8]. Studies demonstrated that hypertension highly prevalent in OSA, and the presence of OSA is associated with an increased incident of resistant hypertension [9,10].

However, fewer studies evaluated BP characteristics of WCH patients with OSA and the relationship between the two. In our study, WCH patients with OSA presented significantly increased daytime and nighttime BP and decreased diurnal difference in SBP, which means higher rate of "non-dipper" BP. It is the no-dipper status predicted the sustain hypertension happened, and eventually, 42.8 % of the WCH patients with OSA developed sustained hypertension after 26 months [11].

WCH may be associate with nervous, psychological and metabolism. Patients with WCH respond to hospital environments and doctors, and are easily excessive nervous and active sympathetic nervous, and presented with a high level adrenaline, renin, aldosterone and cortisol [12]. Hosaka also revealed psychology factor because WCT patients have bar to fit the environment and often depress their emotion [13]. Insulin resistance, metabolic syndrome, abnormal glucose tolerance is suggested concern to WCH. A large Meta-analysis has shown that WCH have highly CVDs incidents, but no difference in all-cause mortality [14].

OSA is chronic intermittent hypoxemia and the consequent is sympathetic nervous activity, RASS activity, oxidative stress injury, systemic inflammatory response etc. WCH patients with OSA have a higher WASO and lower percent of III–IV sleep stages, which suggests that OSA augments sympathetic neural drive to the heart and to the peripheral circulation [11]. Garcia-Rio et al. examined OSA and WCH and reported that WCH was a frequent phenomenon in patients with OSA, and 73% of patients with OSA and WCH have non-dipper BP during the nighttime, and particularly the non-dipper DBP, became the main predictive factor for sustained hypertension in our study [11,15]. Non-dipper DBP status is due to increased peripheral arterial resistance from the sympathetic vasoconstriction effect of apnea and by excessive vascular sensitivity to sympathetic stimulation from changes to the endothelial response [16]. So, WCH may represent a prehypertension status, which could develop into sustained hypertension with OSA.

BPM is one of important detecting methods to distinguish the WCH from sustained hypertension in adults [1,17]. When ABPM resources are not readily available, HBPM (home BP monitoring) provides a reasonable but less desirable alternative to screen for WCH, although the overlap with ABPM is only 60% to 70% for detection of WCH [18,19]. We should monitor ABPM for OSA patients, and WCH patients should detect OSA by polysomnography, especially for those with risk factors of OSA.

Modified lifestyle is an adaptive for patients with OAS and WCH, and ARB and CCB are the best choice of medicine. Some studies found that improving OSA by CPAP can decrease blood pressure. But other studies demonstrated that there are no benefits in decreasing CVDs by CPAP to moderate–severe OSA patients [20]. Further studies are expected to explore the mechanism between WCH and OSA, and provide effective treatment method.

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Correspondence to: Li Li Li-zhu Guo, Cardiovascular Center, Beijing Tong Ren Hospital, Capital University of Medical Sciences, 100730 Beijing, China, E-mail: ellentmc2010@hotmail.com

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