

Research Article

White coat effect in hypertensive patients: the role of hospital environment or physician presence

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Abstract

This study was to evaluate the role of hospital environment or physician presence for white coat effect (WCE) in hypertensive patients. At first, 54 hypertensive outpatients diagnosed on office blood pressure (OBP) were included for 2-week placebo run in. During the second week of the run in period, home BP was measured using electronic BP monitors for 5–7 days. Finally, 26 sustained hypertensive patients with home systolic BP/diastolic BP over 135/85 (but <180/110) mm Hg were enrolled for 8-week treatment of nifedipine controlled-release tablet. In the visit day, BP was measured by patient-self (OBP-p) or by doctor (OBP-d) according to order determined with randomization method. The self-BP measurement was performed in a reception room of hospital. The differences between home BP and OBP-d or OBP-p were calculated as WCE calculated on doctor-measurement (WCE-d) or WCE calculated on patient-measurement (WCE-p), respectively. The home and OBP were measured with the same BP device for each patient during the study period. In the total 54 outpatients received placebo, the WCE-d was similar to the WCE-p (for systolic BP 6.6 ± 14.4 vs. 6.8 ± 15.8 mm Hg, NS; for diastolic BP 3.3 ± 8.8 vs. 2.9 ± 9.2 mm Hg, NS). Meanwhile, the 26 sustained hypertensive patients had similar systolic WCE-d and WCE-p (4.8 ± 10.3 vs. 5.0 ± 12.2 mm Hg, NS) at placebo stage. Similarly, these values were comparable (3.0 ± 14.0 vs. 2.2 ± 14.4 mm Hg, NS) in treatment stage. Hospital environment plays a main role for the WCE in hypertensive patients. *J Am Soc Hypertens* 2017; ■(■):1–5. © 2017 Published by Elsevier Inc. on behalf of American Society of Hypertension.

Keywords: Blood pressure; heart rate; hypertension; nifedipine.

White coat effect (WCE) of blood pressure (BP) indicates a phenomenon that the BP measured by doctor in hospital is higher than that measured in home or by 24-hour BP monitoring. For a patient with normal home BP (HBP) or mean 24-hour BP (<135/85 mm Hg), if his or her office BP (OBP) is $\geq 140/90$ mm Hg white coat hypertension (WCH) could be diagnosed. However, WCE often exists in sustained hypertensive patients who have high OBP and HBP.

Recently, a study pointed out that WCE is an independent predictor of myocardial ischemia in resistant

hypertensive patients.¹ Many studies have suggested that WCE still exists in the treated hypertensive patients.^{2–4}

Although WCE is known for a long time, the contribution of the hospital environment and the physician's presence for the development of WCE is uncertain. Previously, the physician's presence was considered as the main reason for the WCE⁵; therefore, a new type of BP measurement device, automated office blood pressure, was instructed into clinical practice to attenuating the physician effect for the WCE.⁶ Recently, Adiyaman et al. indicated that environment of hospital is the main cause for the WCE in 65 hypertensive patients (most were treated patients)⁷ because the enrolled hypertensive patients were diagnosed on OBP, but not on HBP, the studied patients contained both sustained hypertension and WCH. At present, HBP is useful for excluding WCH. There are some differences on clinical characters and underlying mechanism between WCH and sustained hypertension. It is very possible that the cause of WCE in the hypertensive patients diagnosed on HBP may be

No any potential conflict of interest exists.

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different from that on OBP. As no research about the cause of WCE in the hypertensive patients diagnosed on HBP is available, this study was to determine the separate contribution of the physician's presence and the hospital environment to WCE in the sustained hypertensive patients.

Patients and Methods

The proposal and consent procedures of this study were approved by the Ethic Committee of the Second Affiliated Hospital of Nanchang University (N0 201502), and all patients provided written informed consent.

The Inclusion Criteria

During August in 2015 to March in 2016, 54 essential hypertensive outpatients (29 males, 25 females, aged 18–70 years) with moderately elevated systolic BP (SBP) of 140–180 mm Hg or diastolic BP (DBP) of 90–110 mm Hg on OBP measurement were included.

The information about demographic data, smoking, past histories of diseases, family histories for diabetes, hypertension, stroke, coronary heart disease, and cancer was obtained. The history of smoking was defined as the period of smoking over 6 months. All participants received physical examination, heart rate (HR), height and weight of body (for BMI). Laboratory testing included fasting glucose, serum lipids, and hepatic and renal function before and after treatment.

The patients with heart failure, coronary heart disease, stroke, heart failure, cancer, arrhythmia, and autonomic insufficiency were excluded.

Study Protocol

At first, the 54 screened patients received a 2-week single-blind placebo run in. Meanwhile, all patients were trained for using an electronic BP monitor with transmission function (Omron HEM 7080-IC).

During the second week of the run in period, HBP was measured by the patients in the morning using that BP device for 5–7 days. If the mean values of 5- to 7-day home SBP/DBP \geq 135/85 mm Hg and $<$ 180/110 mm Hg, the patients were enrolled into treatment period.

Finally, 26 patients (12 males, 14 females, 52.0 ± 8.9 years) having both systolic and diastolic sustained hypertension were enrolled into the 8-week treatment. At the first 4 weeks, the dosage of nifedipine-co was 30 mg/d. This dosage was continued if the BP $<$ 135/85 mm Hg at the end of the 4-week treatment. Otherwise, the dosage increased to 30 mg/d, bid for another 4 weeks. No other antihypertensive drugs were permitted for all patients. In this study, two patients received treatment with nifedipine-co 30 mg/d, bid, and two patients were received statins for hyperlipidemia.

BP Measurement

After a rest for 10-minute, bi-lateral arm BP was measured for three times in 1-minute interval in sitting position, and their average was recorded as final value. The arm with higher final SBP was used for BP measurement during the whole study period. In this study, each patient used the same electronic BP device for HBP and OBP measurement. HBP was measured in the morning (7:30 AM–8:30 AM) before breakfast and administration of nifedipine-co. The BP measured at the end of the run-in period was recorded as the baseline BP and that at the end of 8-week treatment period as the posttreated BP.

In the visit day, HBP was measured at 7:00 AM–7:30 AM, but OBP was measured about 1 hour later (8:00 AM–8:30 AM) in the condition without drug use. The patients were randomized to have their OBP measured by themselves (BP-p) first or by doctors (BP-d) first to eliminate the possible bias due to the physician being present or not because BP would be expected to decline from the first to second measurement in the clinic. The self-BP measurement was performed in the same reception room of hospital without physician's presentation.

The differences of the HBP with the OBP-d or OBP-p were used as WCE. Similarly, pulse rate (PR or HR) in three situations was also recorded as HPR, PR-p, and PR-d to calculate WCE on HR.

Statistics

All data were presented as the means \pm standard deviation. The SPSS19.0 statistical package was used. Independent

Table 1

The baseline characteristics of the participants in screening stage and treatment stage

	Preruin in (54)	Pretreatment (26)
General information		
Age (y)	52.9 \pm 10.8	52.0 \pm 8.9
Male (%)	26 (48.1%)	12 (46.2%)
BMI (kg/m ²)	24.7 \pm 2.8	26.1 \pm 2.4
Current smokers (%)	12 (22.2%)	4 (15.4%)
Current drinkers (%)	14 (25.9%)	8 (30.8%)
Comorbidity		
Hyperlipidemia (%)	12 (22.2%)	7 (26.9%)
Diabetes (%)	2 (3.7%)	2 (7.7%)
Hyperuricemia (%)	2 (3.7%)	2 (7.7%)
Treatment drugs		
CCB (%)	23 (42.6%)	/
ACEI (%)	4 (7.4%)	/
ARB (%)	3 (5.6%)	/
BB (%)	3 (5.6%)	/
Atorvastatin (%)	2 (3.7%)	1 (3.8%)

ACEI, angiotensin-converting enzyme Inhibitors; ARB, angiotensin receptor blocker; BB, β -receptor blocker; BMI, body mass index; CCB, calcium channel blockers.

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