

Isolated Office Hypertension: Association with Target Organ Damage and Cardiovascular Risk Indices

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Background: Isolated office hypertension (IOH) has been accepted as a benign condition by some researchers, whereas others believe that it is associated with cardiovascular abnormalities and increased cardiovascular risk. The aim of this present study was to evaluate the effects of IOH on target organ damage and cardiovascular risk indices.

Methods: Arterial blood pressure (BP) measured in the office and by 24-hour ambulatory blood pressure measurement (ABPM), carotid intima-media thickness (CIMT), left ventricular mass index (LVMI), cardiothoracic index (CTI), duration of QTc, 24-hour microalbuminuria, fibrinogen, C-reactive protein (CRP), total cholesterol, low-density-lipoprotein (LDL) cholesterol, high-density-lipoprotein (HDL) cholesterol and triglyceride levels were evaluated. Thirty-three subjects with IOH (office BP $\geq 140/90$ mmHg and daytime ambulatory BP $< 135/85$ mmHg), 17 patients with sustained hypertension (office BP $\geq 140/90$ mmHg and daytime ambulatory BP $\geq 135/85$ mmHg), and 17 normotensive control subjects were recruited in the study. The three groups were matched for age, sex and body mass index.

Results: CIMT was greater in patients with IOH than in normotensive subjects, and it was significantly lower than that of sustained hypertension patients. Significantly higher LVMI was determined in subjects with IOH compared to normotensive subjects. CTI, QTc, microalbuminuria, fibrinogen, CRP, total cholesterol, LDL cholesterol, HDL cholesterol and triglyceride levels did not differ significantly among the three groups.

Conclusions: IOH causes significant target organ damage and should not be regarded as a benign condition.

Key words: hypertension ■ cardiovascular ■ C-reactive protein ■ lipids

INTRODUCTION

Isolated office hypertension (IOH) is also known as white-coat hypertension. The most popular definition of IOH requires blood pressure (BP), measured by conventional techniques in the office, to be $\geq 140/90$ mmHg, while daytime ambulatory blood pressure measurements (ABPM) remain normal ($< 135/85$ mmHg).^{1,2} IOH is a frequently encountered clinical problem in the outpatient clinic settings. Incidence rates for the disease range between 12–50%, depending on the definition.^{3,4} Clinical implications of IOH are not fully understood and are still being debated. Different results have been obtained from the previous studies aimed at determining whether IOH is accompanied by metabolic abnormalities and target organ damage.⁵⁻¹⁵ Some studies concluded that IOH has no influence on target organs, favoring the view that considers IOH as a benign condition.⁵⁻⁹ Others stated that IOH is related to hypertensive complications up to a certain degree.¹⁰⁻¹⁵ The aim of the present study was to compare the risk conferred by IOH and untreated hypertensive patients with respect to metabolic abnormalities and the target organ damage.

MATERIAL AND METHODS

Fifty untreated research subjects with hypertension according to clinical BP ($\geq 140/90$ mmHg) and 17 normotensive control subjects in our outpatient clinic were recruited consecutively in the study. All participants underwent ABPM in order to evaluate daytime, nighttime and 24-hour systolic (SBP) and diastolic BP (DBP). The local ethics committee approved the study, and informed consent was obtained from each subject before enrollment into the study.

Patients were excluded from the study if they had any history of current or recent use of antihypertensive medications; concurrent medical conditions such as acute illness, asthma, primary hyperparathyroidism, thyroid function abnormalities or medications that might have a possible impact on BP (such as

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tricyclic antidepressants, selective serotonin reuptake inhibitors, corticosteroids); and if they had any concomitant diseases such as diabetes mellitus, impaired glucose tolerance, renal failure, secondary hypertension, cerebrovascular disease, heart failure, ischemic heart disease and peripheral vascular disease. Subjects were asked to arrive at the outpatient clinic on the following week in order to confirm and perform BP measurements. Subjects refrained from smoking, drinking coffee or tea, or using any other stimulants 30 minutes prior to their arrival at the outpatient clinic. Subjects were seated with their backs resting against the chair, and their arms bared and supported at the heart level in a quiet, warm office in the clinic. The same physician performed the clinical SBP and DBP measurements on all three occasions. Appropriate-sized arm cuff and a mercury sphygmomanometer were applied to the patient in supine position after 5–10 minutes of rest. SBP and DBP values were recorded using the first and fifth phases of the Korotkoff's sounds, respectively, and were rounded to the nearest 2 mmHg.¹⁶ All subjects underwent 24-hour ABPM following the blood pressure measurements.

ABPM was performed using a portable noninvasive recorder (SpaceLabs 90207). The device was programmed to measure the blood pressure at 20-minute intervals throughout the day and night. The cuff was wrapped around the nondominant arm at 8:00 AM and removed the next day. The patient was allowed to perform normal daily activities, except for strenuous exercise, during the measurements. Subjects were instructed to keep a diary recording their asleep and awake times. The data were transferred to a computer and loaded into ABPM report management system software. Mean SBP and DBP values were calculated for daytime and nighttime periods. Daytime and nighttime were defined as the

hours between 6:00 AM and 6:00 PM, and 6:00 PM and 6:00 AM, respectively.¹⁷

Subjects were divided into three groups. Subjects with normal clinical BP (<140/90 mmHg) and normal daytime ambulatory BP (<135/85 mmHg) were defined as normal (control subjects), an elevated clinical BP (\geq 140/90 mmHg) but normal daytime ambulatory BP were defined as IOH, and the elevation of both clinical and daytime ambulatory BP was defined as sustained hypertension.^{1,2,18}

Age, sex distribution, body mass index, smoking habits and family history of hypertension did not differ among the three groups. Thirty-three patients with IOH (18 men, 15 women) aged 46 ± 7 years, 17 patients (eight men, nine women) with sustained hypertension aged 51 ± 10 years, and 17 normotensive control subjects (nine men, eight women) aged 45 ± 6 years were included in the study.

All subjects underwent physical examination, anthropometric measurements, blood chemistry and urine analyses, electrocardiogram (ECG), chest radiography, Doppler echocardiography and carotid ultrasonography. Doppler echocardiography and carotid ultrasonography were performed by the same trained cardiologist and sonographer who were blinded to the subjects' identities. Body mass index was evaluated as an index of adiposity (weight / height²).

Echocardiography

All echocardiography examinations were performed by a single operator, using Hewlett Packard Sonos 5500 (Andover, MA). Correct alignment of the cursor was performed on parasternal long-axis view under two-dimensional echocardiography guidance, then the probe was rotated to obtain short-axis view and M-mode traces. Interventricular septal thickness, posterior wall thickness and left atrial dimension were

Table 1. Characteristics and blood pressure values of study the population

Parameter	Sustained Hypertension ¹	Isolated Office Hypertension ²	Normotension ³
N	17	33	17
Males/females	8/9	18/15	9/8
Age (years)	51 ± 10	46 ± 7	45 ± 6
BMI (kg/m ²)	30.2 ± 5.2	29.6 ± 7.2	30.3 ± 4.4
Waist-to-hip ratio	0.91 ± 0.03	0.92 ± 0.03	0.93 ± 0.03
Smoking (%)	37	39	41
Office SBP (mmHg)	$156.4 \pm 21.1^*$	$141.3 \pm 9.1^\dagger$	$114.4 \pm 10.9^{**}$
Office DBP (mmHg)	$100.7 \pm 7.6^*$	$92.8 \pm 7.2^\dagger$	$72.3 \pm 7.5^{**}$
Mean 24-hour SBP (mmHg)	$136.8 \pm 11.1^*$	117.7 ± 6.9	$112.3 \pm 7.6^{**}$
Mean 24-hour DBP (mmHg)	$88.0 \pm 5.3^*$	74.0 ± 6.4	$71.4 \pm 6.6^{**}$
Mean daytime SBP (mmHg)	$140.5 \pm 11.6^*$	120.7 ± 6.8	$115.2 \pm 8.7^{**}$
Mean daytime DBP (mmHg)	$91.7 \pm 6.3^*$	76.7 ± 6.5	$74.1 \pm 7.0^{**}$
Mean night-time SBP (mmHg)	$133.0 \pm 11.7^*$	$114.6 \pm 8.0^\dagger$	$109.5 \pm 7.8^{**}$
Mean night-time DBP (mmHg)	$84.3 \pm 5.5^*$	70.8 ± 6.7	$69.1 \pm 7.2^{**}$

* P1-2<0.05; ** P1-3<0.01; † P2-3<0.05; HT: hypertension, IOH: isolated office hypertension, BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure

measured using two-dimensional guided M-mode echocardiography in compliance with the American Society of Echocardiography guidelines.¹⁹ If two-dimensional guided M-mode beam could not be optimally oriented, two-dimensional long-axis views were used to obtain linear measurements of LV cavity and walls. Left ventricular mass index (LVMI) was calculated using the equation proposed by Devereux and associates. $LVMI = \{0.8 \times 1.04 [(LVID + PWT + IVST) \div 3 - LVID] + 0.6\} / \text{body surface area (g/m}^2\text{)}$, where LVID is the left ventricular internal diameter, PWT is the posterior wall thickness and IVST is the interventricular septal thickness.²⁰

Carotid Ultrasonography

Both carotid arteries were monitored in terms of carotid intima-media thickness (CIMT) in all patients using the high-resolution ultrasound system equipped with a mechanical sector probe with a 7.5-MHz annular imaging transducer. Patients were laid in supine position with mild hyperextension of the neck to allow optimal visualization of the common carotid artery. The mid and distal portions of the common carotid artery, carotid bulb, and the proximal portions of the internal and external carotid arteries were systematically examined manually in short-axis and long-axis views.²¹ We measured the thickness of the intima-media on the far wall of the bilateral common carotid artery about 10-mm proximal to the bifurcation of the carotid arteries on the B-mode monitor and used the mean values for the study.

QTc

Standard 12-lead ECG recorded at a speed of 50 mm/sec was performed on each patient. QT intervals were measured in 12-lead surface ECGs. The

end of the T wave was defined as the point at which the line of maximal downslope of the T wave crossed the baseline. When U wave was present, the QT interval was measured to the nadir of the curve between the T and U waves. QT values were then corrected by heart rate (QTc) according to Bazett's formula.²² The arithmetic mean of the two measured values was used for the analysis.

Cardiothoracic Index

The cardiothoracic index (CTI) was measured from chest radiography as the transverse diameter of the heart in relation to the diameter of the thoracic cavity.

Serum Analysis

Blood samples were drawn after a minimum fasting period of 12 hours in all patients. Glucose, creatinine (DMA Inc., East Arlington, TX), total cholesterol (Equal Diagnostics, Exton, PA), triglycerides (Thermo DMA, East Arlington, TX) and high-density-lipoprotein (HDL) cholesterol (Sigma Diagnostics, Dorset, United Kingdom) were determined. Low-density-lipoprotein (LDL) cholesterol was calculated using Friedewald's formula ($LDL \text{ cholesterol} = \text{total cholesterol} - (\text{HDL cholesterol} + \text{triglyceride} / 5)$). C-reactive protein levels were determined using an immunonephelometric system in compliance with the manufacturer's instructions (Dade Behring, Marburg, Germany). Fibrinogen was measured using a modified Clauss technique. Twenty-four-hour urine samples of the subjects were collected for the evaluation of microalbuminuria with radioimmunoassay (Immunotech, Prague, Czech Republic).

Data Analysis

All statistical analyses were performed using the software program SPSS (Statistical Package for the

Table 2. Results of cardiovascular and metabolic parameters related to target organ damage

Parameter	Sustained Hypertension ¹	Isolated Office Hypertension ²	Normotension ³
CIMT (mm)	0.78 ± 0.19*	0.69 ± 0.09 [†]	0.56 ± 0.11**
CTI	0.49 ± 0.07	0.46 ± 0.04	0.43 ± 0.03
QTc (sec)	0.42 ± 0.03	0.41 ± 0.02	0.4 ± 0.03
LVMI (g/m ²)	93 ± 22	90 ± 17 [†]	78 ± 16**
IVST (mm)	10.8 ± 1.45	10.0 ± 1.41 [†]	9.2 ± 1.15**
PWT (mm)	10.5 ± 1.15*	9.5 ± 1.28 [†]	8.8 ± 1.16**
LVID (mm)	46.5 ± 6.3	47.8 ± 4.2	47.7 ± 3.4
CRP (mg/dL)	4.08 ± 3.16	3.07 ± 2.55	2.26 ± 1.81
Fibrinogen (g/L)	4.11 ± 0.92	3.35 ± 0.72	3.15 ± 0.74
Microalbuminuria (mg/day)	15.88 ± 18.73	8.95 ± 14.35	6.67 ± 6.65
Glucose (mg/dL)	95 ± 8.9	96 ± 9.7	94 ± 6.8
Total cholesterol (mg/dL)	211 ± 26	194 ± 34	188 ± 39
LDL cholesterol (mg/dL)	129 ± 23	120 ± 27	115 ± 32
Triglyceride (mg/dL)	149 ± 57	136 ± 51	133 ± 62
HDL cholesterol (mg/dL)	46 ± 11	49 ± 13	51 ± 12

* P1-2<0.05; ** P1-3<0.01; † P2-3<0.05; CIMT: carotid intima-media thickness; CTI: cardiothoracic index; LVMI: left ventricular mass index; IVST: interventricular septum thickness; PWT: posterior wall thickness; LVID: left ventricular internal diameter; CRP: C-reactive protein

Social Sciences) for Windows® version 10.0 (SPSS Inc. Chicago, IL). Continuous, normally distributed variables, expressed as the mean (\pm SD) were compared between the three patient groups using one-way ANOVA followed by post hoc testing with the Bonferroni test for multiple comparisons regarding continuous variables. Comparisons were also made using the Kruskal-Wallis test followed by the Mann-Whitney U test for multiple comparisons regarding non-continuous variables. Spearman test was used for correlation analyses. Statistical comparisons for nominal variables were assessed by Chi-squared analysis. Statistical significance was defined as $P < 0.05$.

RESULTS

Blood Pressures

The main characteristics and BPs of the study population are presented in Table 1. Mean clinical SBP and DBP were significantly higher in sustained hypertensives and in IOH patients than in normotensives. The values were higher in sustained hypertensives when compared to IOH patients. Ambulatory 24-hour, daytime and nighttime SBP values were significantly higher in sustained hypertensives than in IOH patients and normotensives. Ambulatory 24-hour, daytime and nighttime DBP values were significantly higher in sustained hypertensives than in IOH patients and normotensives. No difference could be determined between IOH patients and normotensives regarding the 24-hour ambulatory, daytime and nighttime SBP and DBP values.

Carotid Intima-Media Thickness, Cardiothoracic Index and QTc

Data are presented in Table 2 for CIMT, CTI, and QTc measurements. The CIMT was greater in patients with IOH than in normotensive subjects (0.69 ± 0.09 mm vs. 0.56 ± 0.11 mm, $P < 0.05$). CIMT was also significantly higher in subjects with sustained hypertension when compared to IOH patients (0.78 ± 0.19 mm vs. 0.69 ± 0.09 mm, $P < 0.05$). Although an increasing trend was documented in the IOH and sustained hypertension groups, CTI and QTc intervals did not differ significantly among the three groups.

Echocardiography Values

LVMI was higher in patients with IOH than in normotensive subjects (90 ± 17 g/m² vs. 78 ± 16 g/m², $P < 0.05$). In contrast, LVMI in IOH patients was indistinguishable from those in patients with sustained hypertension. IVST was significantly higher in patients with IOH than in normotensives (10 ± 1.41 mm vs. 9.2 ± 1.15 mm, $P < 0.05$). PWT was also found to be increased in patients with IOH

than in normotensives (9.5 ± 1.28 mm vs. 8.8 ± 1.16 mm, $P < 0.05$). On the other hand, there were no significant differences regarding LVID.

Serum Analysis and Microalbuminuria

Blood chemistry, C-reactive protein, fibrinogen and urine analyses results are summarized in Table 2. Plasma glucose, total cholesterol, LDL cholesterol, HDL cholesterol and triglyceride values did not differ significantly among the groups, although an increasing trend was noticed in the IOH and sustained hypertension groups, with respect to total cholesterol, LDL cholesterol and triglyceride values. When compared to the normotensive subjects, those subjects with either sustained hypertension or IOH had higher mean CRP, fibrinogen and microalbuminuria levels, but these results were not statistically significant.

In correlation analyses, CIMT was significantly correlated with office SBP ($r=0.68$, $P < 0.001$) and DBP ($r=0.55$, $P < 0.001$), mean 24-hour ambulatory SBP ($r=0.57$, $P < 0.001$) and DBP ($r=0.36$, $P < 0.05$). LVMI was correlated with office SBP ($r=0.31$, $P < 0.05$) and DBP ($r=0.32$, $P < 0.05$), 24-hour ambulatory SBP ($r=0.24$, $P < 0.05$) and DBP ($r=0.28$, $P < 0.05$). There were significant correlations between microalbuminuria and office SBP ($r=0.27$, $P < 0.05$) and 24-hour ambulatory SBP ($r=0.29$, $P < 0.05$). CRP levels correlated with office SBP ($r=0.38$, $P < 0.05$) and 24-hour ambulatory SBP ($r=0.32$, $P < 0.05$).

DISCUSSION

White-coat hypertension, or IOH, has been defined as the persistent elevation of BP at the clinic or office only. It usually implies that daytime ABPM is normal. White-coat hypertension should not be confused with the white-coat effect. The white-coat effect signifies the difference in BP between the office and daytime ABPM and occurs in patients with white-coat hypertension as well as in patients with sustained hypertension that is treated or untreated.⁴ Controversy remains on whether IOH is a benign clinical condition or a clinical condition, which implies an increased risk for target organ damage. IOH has been evaluated as an innocent clinical phenomenon by some investigators.⁵⁻⁹ However, contradictory conclusions have been reached in other longer-term longitudinal studies, indicating that IOH tends to coexist with metabolic and cardiovascular risk factors, and that it is associated with increased target organ damage. These studies suggest that IOH is not an innocent phenomenon and predicts total and cardiovascular mortality during long-term follow-up.¹⁰⁻¹⁵

Some investigators showed that CIMT and plaque index in subjects with IOH were greater than those of normotensive subjects and equal to that of sustained hypertensives.²¹ However, some studies

concluded that CIMT in IOH was apparently associated with low risk of vascular lesions, practically to the same level as normotensive subjects.⁸ An important finding of the present study is that CIMT in subjects with IOH is on average less than that of sustained hypertensives, but greater than the CIMT of subjects with normal BP values both in the office and under ambulatory conditions. The mean CIMT also significantly correlated with office systolic and diastolic pressures. Our findings add to the body of evidence, suggesting that IOH is associated with end organ damage and is not simply a benign disease.

Another finding of our study is that LVMI is higher in IOH patients than in normal subjects. More importantly, LVMI was found to be similar in patients with IOH and sustained hypertension. IVST and PVT were also higher in patients with IOH than in normotensives. These findings are concordant with the results of some of the previous studies, and they imply that whenever BP is elevated, cardiac structure will face a greater risk of being altered when compared to the normotensive state.^{23,24} It is also possible for transient increases in blood pressure to have an independent effect on left ventricular mass.

Our patients with isolated office hypertension had higher mean nighttime ambulatory systolic blood pressures than normotensive subjects. The difference between the groups was very small (114 mmHg vs. 109 mmHg), but it was sufficient to trigger cardiovascular damage. It seems that even a slightly higher blood pressure may result in a chronic increase in the hemodynamic load of the heart and the vascular system.

Hypertensive patients are at significant risk of cardiovascular complications, possibly related to endothelial damage, cardiac dysfunctions or abnormalities in metabolic parameters. Our findings of a stepwise increase in the cholesterol, C-reactive protein and fibrinogen levels, microalbuminuria, and a trend towards longer QTc and cardiothoracic index measurements among patients with IOH and sustained hypertension in comparison to normotensive subjects suggest that some degree of metabolic and additional cardiac plus vascular deterioration is present. Findings of positive correlations between microalbuminuria and both office and ambulatory systolic blood pressures, as well as also correlations between CRP levels and office and ambulatory systolic blood pressures, may support this hypothesis.

Although the present study supports the belief that patients with IOH represent a high-risk group, it is difficult to draw a definite judgment from a small number of patients. This conclusion drawn from cross-sectional studies like ours should be considered as tentative until long-term prospective studies determine whether IOH is associated with an

increased rate of cardiovascular events.

The results of our study suggest that IOH is not an innocent phenomenon and should be regarded as a prehypertensive state. IOH constitutes a state of risk intermediate between normotension and sustained hypertension, which demands profound evaluation and active monitoring. There seems to be a continuum between the level of ambulatory blood pressure and target organ damage.

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