White coat hypertension is a term used for persons not receiving antihypertensive medication who have a persistently high office blood pressure (≥140/90 mmHg) together with a normal; ambulatory blood pressure (<135/85 mmHg) or home blood pressure [1-4]. Muscle sympathetic nerve traffic inhibition coupled with a skin sympathetic nerve traffic excitation induced by measurement of blood pressure by a doctor has been reported to cause the increase in blood pressure [5]. The prevalence of white coat hypertension in older persons is higher than in younger persons and has prevalence rates between 15% and 25% in the elderly [6,7]. The magnitude of the white coat effect is increased by older age, female gender, and smoking [8]. Ambulatory blood pressure monitoring is recommended to confirm the diagnosis of white coat hypertension in patients with office hypertension but no target organ damage [1,2,4].

The 2013 European Society of Hypertension/European Society of Cardiology hypertension guidelines recommend that patients with white coat hypertension who have no additional cardiovascular risk factors should be treated with lifestyle changes and have a close follow-up [4]. These guidelines also recommend that patients with white coat hypertension who are at increased cardiovascular risk because of metabolic abnormalities or asymptomatic target organ damage should be considered for antihypertensive drug treatment in addition to lifestyle changes [4].

The prognosis of white coat hypertension is controversial. In a longitudinal population-based study of 602 men aged 50 years, at 20-year follow-up, both patients with white coat hypertension and sustained hypertension had impaired insulin sensitivity, increased blood glucose, and increased serum insulin and heart rate compared with normotensive persons [9]. However, left ventricular mass and urinary albumin excretion were increased only in patients with sustained hypertension [9]. A meta-analysis of 7,961 persons from 8 studies showed no difference in cardiovascular outcomes between persons with white coat hypertension and normotensive persons [10]. However, at follow-up antihypertensive drug therapy use was more frequent in persons with white coat hypertension than in normotensive persons, and this influenced the incidence of cardiovascular outcomes [10].

At a median follow-up of 8.3 years of 2,984 persons with a normal blood pressure, 695 persons with white coat hypertension, 404 persons with masked hypertension, and 924 persons with sustained hypertension not treated with antihypertensive drug therapy, compared with normotensive persons, the incidence of cardiovascular events was increased 42%, p=0.02, in persons with white coat hypertension, 55%, p<0.01, in persons with masked hypertension, and 213%, p<0.0001, in persons with sustained hypertension [11]. At a median follow-up of 8.3 years of 328 persons with a normal blood pressure, 230 persons with white coat hypertension, 232 persons with masked hypertension, and 661 persons with sustained hypertension treated with antihypertensive drug therapy, compared with normotensive persons, the incidence of cardiovascular events was insignificantly increased 16% in persons with white coat hypertension, increased 76%, p=0.002, in persons with masked hypertension, and increased 40%, p=0.04, with sustained hypertension [11].

Of 1,589 persons, 825 (52%), mean age 44 years, were normotensive, 391 persons (25%), mean age 55 years, had white coat hypertension, and 373 (24%), mean age 60 years, had hypertension [12]. Mean follow-up was 16 years. Cardiovascular mortality was 1.0% in normotensive persons, 5.4% in persons with white coat hypertension, and 12.9% in persons with hypertension. Compared to persons who were normotensive, the adjusted hazard ratios for cardiovascular mortality were 6.03,
p<0.0001, for persons with white coat hypertension and 15.57, p<0.0001, for persons with hypertension [12]. All-cause mortality was 6.4% in normotensive persons, 19.7% in persons with white coat hypertension, and 30.0% in persons with hypertension. Compared to persons who were normotensive, the adjusted hazard ratios for all-cause mortality were 3.33, p<0.0001, for persons with white coat hypertension and 5.44, p<0.0001, for persons with hypertension [12].

The Dallas Heart Study included 1,804 persons, mean age 40 years (35% whites, 42% blacks, 21% Hispanics, and 56% women), with a normal blood pressure, 123 persons, mean age 49 years (24% whites, 63% blacks, 12% Hispanics, and 58% women), with white coat hypertension, 582 persons, mean age 47 years (31% whites, 56% blacks, 12% Hispanics, and 53% women), with masked hypertension, and 518 persons, mean age 50 years (21% whites, 69% blacks, 8% Hispanics, 54% women), with sustained hypertension [13]. Median follow-up was 9.4 years. Compared to persons with a normal blood pressure, both persons with white coat hypertension and persons with masked hypertension had a significant independent increase in aortic pulsed wave velocity, cystatin C, and urinary albumin-to-creatinine ratio [13]. Compared to persons with a normal blood pressure, the incidence of cardiovascular events was increased in persons with white coat hypertension (adjusted hazard ratio=2.09, p=0.035), in persons with masked hypertension (adjusted hazard ratio=2.03, p=0.0005), and in persons with sustained hypertension (adjusted hazard ratio=3.12, p<0.0001) [13].

On the basis of the available data, I agree with the 2013 European Society of Hypertension/European Society of Cardiology hypertension guidelines which recommend that patients with white coat hypertension who have no additional cardiovascular risk factors should be treated with lifestyle changes and have a close follow-up [4]. I also agree with their guidelines which recommend that patients with white coat hypertension who are at increased cardiovascular risk because of metabolic abnormalities or asymptomatic target organ damage should be considered for antihypertensive drug treatment in addition to lifestyle changes [4].
References


