

Left ventricular filling profiles in young white coat hypertensive patients without hypertrophy

張念中;王子哲;賴志洋;陳保羅

Chang Nen-Chung;Lai Zhi-Yang;Wang Tze-Che

摘要

Abstract

Abstract This study was to assess left ventricular diastolic function in young white-coat hypertensive subjects <50 years of age without hypertrophy. Hypertensive patients (systolic or diastolic blood pressure 140 or 90 mm Hg on all three visits) were defined as white coat if their average 24-hour blood pressure was <127/81 mm Hg and at least 18/16 mm Hg lower than their average office values. We chose three groups balanced for sex, age, and body mass index: 50 sustained hypertensives, 25 white-coat hypertensives, and 25 normotensives. Office blood pressure was similar in white-coat and sustained hypertensives. Ambulatory blood pressure was comparable in white-coat hypertensives and normotensives. Compared with normotensives, white-coat hypertensives had more impaired diastolic function: increased ratio of late to early filling velocities, raised ratio of late to early time-velocity integral, prolonged deceleration time, and lengthened isovolumic relaxation time ($P < .001$, $P < .001$, $P = .002$, and $P < .001$, respectively). No difference was noticed between white-coat and sustained hypertensives. Compared with normotensives, white-coat hypertensives had higher values of plasma and urine norepinephrine ($P < .001$ and $P < .001$, respectively), plasma and urine aldosterone ($P < .001$ and $P = .002$, respectively), plasma renin activity ($P = .04$), total cholesterol ($P = .001$), and LDL cholesterol ($P < .001$). No difference was observed between white-coat and sustained hypertensives. Within white-coat hypertensives, 24-hour urinary aldosterone closely correlated with the ratio of late to early filling velocities ($P = .008$), and plasma and 24-hour urinary norepinephrine correlated well with total cholesterol ($P = .037$ and $P = .006$, respectively). No correlation was detected within the sustained hypertensives and normotensives. Heightened neurohumoral activity clearly supported the progression of diastolic dysfunction and metabolic abnormality in white-coat hypertensives