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VENTRICULAR STIFFNESS INCREASES IN ACCORDANCE WITH THE LEVEL OF MICROALBUMINURIA IN NEVER TREATED HYPERTENSIVE PATIENTS

ACC Poster Contributions

Ernest N. Morial Convention Center, Hall F

Monday, April 04, 2011, 9:30 a.m.-10:45 a.m.

Session Title: Arterial Stiffness in Hypertension

Abstract Category: 16. Hypertension

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Background: Left ventricular (LV) diastolic dysfunction is prerequisite condition of heart failure in hypertensive patients with or without LV hypertrophy (LVH). Microalbuminuria (MAU) has been known as one of the cardiovascular risk factors and is related with cardiovascular mortality. This study was aimed to evaluate the relation between MAU and diastolic function in never treated hypertensive patients.

Methods: Total 123 patients (54±12 years, 54 male) with hypertension who never treated or discontinued medication at least 6 months were evaluated. LV diastolic function was assessed with transthoracic echocardiography and arterial stiffness was evaluated by measuring ankle-brachial index (ABI) and brachial-ankle pulse wave velocity (baPWV). These cardiovascular stiffness indices were compared with urine albumin level.

Results: MAU (spot urine albumin/creatinine between 20-200 mg/g for male and 30-300mg/g for female) was observed in 90 (73.2%) patients. Spot urine albumin/creatinine was correlated with LV filling pressure ($r=0.45$, $P=0.001$) and calculated pulmonary capillary wedge pressure ($r=0.43$, $P=0.001$). The mean value of LV mass index was 102.4±28.9g/m² in men and 99.4±23.8g/m² in women. Even though LVH (LV mass index>90g/m²) was observed in only 74 (60.2%) patients, LV mass index was significantly correlated with spot urine albumin/creatinine ($r=0.22$, $P=0.027$). There was no evidence of relation between spot urine albumin/creatinine and ABI or baPWV.

Conclusions: Our data shows MAU is associated with ventricular stiffness in never treated hypertension. MAU is not only the risk factor of cardiovascular disease but also associated with the LV function, hence, treatment targeting decrease of albuminuria might be effective to prevent LV diastolic dysfunction.