

## OBSERVATIONS

## One-Year Reductions in Body Weight and Blood Pressure, but Not in Visceral Fat Accumulation and Adiponectin, Improve Urinary Albumin-to-Creatinine Ratio in Middle-Aged Japanese Men

Microalbuminuria has been recognized recently as a risk factor for cardiovascular diseases as well as renal failure and is often found in subjects with metabolic syndrome. The relationship between visceral fat accumulation and microalbuminuria, urinary albumin-to-creatinine ratio (UACR), has not been fully clarified. Our cross-sectional study demonstrated that visceral fat accumulation is associated with increases in UACR. However, the accompanying obesity-related risk factors, especially hemoglobin A1c (HbA1c) and elevated blood pressure, strongly increased the risk of UACR (1). We have reported that reductions in both body weight and estimated visceral fat area (eVFA) measured by the bioelectrical impedance analysis method (2) were accompanied by reductions in the number of obesity-related cardiovascular risk factors (3) and increases in serum levels of adiponectin (4) in our longitudinal Amagasaki Visceral Fat Study, in which intensive risk factor-oriented health promotion programs were provided. The present study investigated the relationship between 1-year  $\Delta$  changes in UACR ( $\Delta$ UACR) and the estimated visceral fat area ( $\Delta$ eVFA), and other parameters in these subjects.

The study subjects were 1,539 Japanese males (mean  $\pm$  SD age 45.8  $\pm$  10.4 years [range 20–68 years]) who were employees of the Amagasaki City Office, had undergone an annual health checkup in both 2006 and 2007, and were not taking any medications for diabetes, hypertension, and dyslipidemia. The study was approved by the human

ethics committee of Osaka University, and a signed informed consent form was obtained from each participant. The registration number of the trial at the UMIN is 000002391 (the Amagasaki Visceral Fat Study). Height, weight, and waist circumference (WC) at the umbilical level were measured in standing position. Serum concentrations of adiponectin were measured by the latex particle-enhanced turbidimetric assay. UACR was calculated from a single spot urine specimen.  $\Delta$ UACR correlated with  $\Delta$ BMI ( $r = 0.077$ ,  $P = 0.0036$ ) and  $\Delta$ systolic blood pressure (SBP,  $r = 0.063$ ,  $P = 0.0127$ ), but not  $\Delta$ HbA1c ( $P = 0.0705$ ),  $\Delta$ triglyceride ( $P = 0.0524$ ),  $\Delta$ HDL cholesterol ( $P = 0.9906$ ),  $\Delta$ LDL cholesterol ( $P = 0.5761$ ),  $\Delta$ estimated glomerular filtration rate (eGFR, Modification of Diet in Renal Disease [MDRD],  $P = 0.1724$ ),  $\Delta$ eVFA ( $P = 0.3026$ ), and  $\Delta$ adiponectin ( $P = 0.9860$ ). Stepwise multiple regression analysis identified  $\Delta$ BMI ( $F = 6.341$ ) and  $\Delta$ SBP ( $F = 4.053$ ) as significant determinants of  $\Delta$ UACR.

The present study demonstrated that, unlike  $\Delta$ BMI and  $\Delta$ SBP, 1-year reduction of eVFA only was not sufficient to improve UACR. This meant a significant reduction in blood pressure, which is downstream of visceral fat accumulation, should be required. We could not find a significant relationship between 1-year changes in UACR and HbA1c, probably because the number of subjects with abnormal HbA1c values ( $\geq 5.8\%$ ) was small ( $n = 130$  of 1,539, 8.4%). Although previous studies reported a relationship between serum levels of adiponectin and albuminuria in obese subjects (5) and general men (1), we could not observe a relationship between 1-year changes in UACR and adiponectin in general men. Monitoring over a longer period of time might be needed. To our knowledge, this is the first report demonstrating the relationship between 1-year changes in UACR and visceral fat accumulation and adiponectin.

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