Body: Previous studies support that metabolic syndrome (MS) has a role in the development of chronic kidney disease (CKD). We examined this relationship and its possible determinants in a cross-sectional study of subjects (n=125; females 81.8%; age 52.9±11.2; mean±SD) assisted at the Pre-diabetes (Pre-DM) and Endocrine clinics of a Hospital de Clínicas de Porto Alegre. MS was defined according to the new Joint Scientific Statement Harmonization definition as the presence of 3 out of 5 of the following criteria: hypertension, low HDL-cholesterol, high triglyceride levels, hyperglycemia and high waist circumference. Glomerular filtration rate (GFR) was estimated by the CKD Epidemiology Collaboration (CKD-EPI) equation, insulin resistance by HOMA-IR, renal damage by microalbuminuria and endothelial dysfunction by fibrinogen. GFR was lower in subjects with MS than those without MS (92.4±18.8 vs. 102.6±14.3 ml/min per 1.73m^2; P=0.027). GFR decreased with the increasing number of MS criteria (0 or 1 criteria 104.9±14.7 vs 2 criteria 101.5±14.5 vs 3 or 4 criteria 96.3±17.4 vs 5 criteria 83.3±19.6 ml/min per 1.73m^2; P=0.002). Among the MS criteria, GFR was lower in those with hypertension (P=0.004) and hyperglycemia (P=0.005), it was not statistically lower in subjects with hypertrygliceridemia (P=0.064), and it was similar in those with low HDL (P=0.196) and elevated waist circumference (P=0.539) than those without each one of these criteria. Systolic arterial blood pressure (SBP) was inversely related to GFR (r=-0.324; P=0.001). By multiple linear regression analysis, GFR was just associated to increased fasting plasma glucose (R=0.197; P=0.038) and SBP (R=-0.344; P<0.001. The previously described association between MS and CKD was confirmed in our study. This relationship was not related with insulin resistance, renal damage or endothelial dysfunction and it was mostly determined by abnormalities in blood pressure homeostasis and hyperglycemia.