

SP049

INFLAMMATION AND APPARENT TREATMENT RESISTANT HYPERTENSION IN PATIENTS WITH CHRONIC KIDNEY DISEASE

Jing Chen¹, D Bundy Joshua², Lee Hamm L¹, Chi-Yuan Hsu³, James Lash⁴, Edgar R Miller⁵, George Thomas⁶, Debbie L Cohen⁷, Matthew R Weir⁸, Dominic S Raj⁹, Hsiang-Yu Chen¹⁰, Dawei Xie¹⁰, Panduranga Rao¹¹, Jackson T Wright¹², Mahboob Rahman¹², He Jiang¹³

¹1430 tulane ave, #8545, New Orleans, LA, USA, United States of America, ¹1430 tulane ave, #8545, New Orleans, United States of America, ²Northwestern University Feinberg School of Medicine, Chicago, IL, USA, United States of America, ³University of California San Francisco School of Medicine, San Francisco, CA, United States of America, ⁴University of Illinois College of Medicine, Chicago, IL, USA, United States of America, ⁵Johns Hopkins University School of Medicine, Baltimore, MD, USA, United States of America, ⁶Cleveland Clinic, Cleveland, OH, United States of America, ⁷University of Pennsylvania, Philadelphia, United States of America, ⁸University of Maryland School of Medicine, Baltimore, MD, United States of America, ⁹Georgetown University School of Medicine, Washington DC, United States of America, ¹⁰University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, United States of America, ¹¹University of Michigan School of Medicine, Ann Arbor, MI, United States of America, ¹²Case Western Reserve University School of Medicine, Cleveland, OH, United States of America and ¹³1440 Canal Street, Ste 2000, New Orleans, LA, USA, United States of America

INTRODUCTION: Apparent treatment-resistant hypertension (ATRH) is highly prevalent and associated with cardiovascular disease (CVD) risk in patients with chronic kidney disease (CKD). We analyzed the association of inflammatory biomarkers with ATRH and its complications in CKD patients.

METHODS: ATRH was defined as blood pressure (BP) $\geq 140/90$ mm Hg while taking ≥ 3 antihypertensive medications or BP $< 140/90$ mm Hg while taking ≥ 4 medications. Analyses included 1,359 Chronic Renal Insufficiency Cohort (CRIC) Study participants with ATRH and 2,008 hypertensive participants without. Logistic regression was used to examine cross-sectional associations of inflammatory biomarkers and ATRH adjusting for demographic, lifestyle, and clinical risk factors and treatments. Cox proportional hazards models were used to assess the impact of inflammatory biomarkers on associations of ATRH with composite CVD and mortality beyond conventional risk factors.

RESULTS: Multivariable-adjusted odds ratio (95% confidence intervals [CI]) of ATRH for the highest tertile vs. lowest tertile of inflammatory biomarker levels was 1.29 (95% CI, 1.05-1.59) for interleukin-6 (IL-6), 1.49 (95% CI, 1.20-1.85) for tumor necrosis factor- α (TNF- α) and 0.77 (95% CI, 0.63-0.95) for transforming growth factor- β (TGF- β). High-sensitivity C-reactive protein, fibrinogen, interleukin-1 β , and interleukin-1 receptor antagonist were not significantly associated with ATRH. Adding inflammatory biomarkers to Cox models did not attenuate the significant association of ATRH with CVD and mortality.

CONCLUSIONS: Our findings show higher levels of IL-6 and TNF- α and lower levels of TGF- β were independently associated with odds of ATRH. Targeting specific inflammatory pathways may improve BP control in CKD patients.