ORAL SECTION

Oral Presentations

ATRIAL FIBRILLATION AND HEART FAILURE

TIME-VARYING SERUM URIC ACID PREDICTS NEW-ONSET ATRIAL FIBRILLATION IN TREATED HYPERTENSIVE PATIENTS

Sverre E. Kjeldsen1, Eran S. Zachs2, Ilbri M. Stokke1, Kristian Wachtell2, Darcy A. Hille3, Aud Høieggen1, Stevo Julius4, Eva Gerdts5, Peter M. Okin2, Richard B. Devereux2. 1University of Oslo, Ullevaal Hospital, Oslo, NORWAY, 2Weill Cornell Medicine, Greenberg Division of Cardiology, New York, NY, USA, 3Merck Research Laboratories, Pennsylvania, North Wales, PA, USA, 4University of Michigan Medicine, Ann Arbor, MI, USA, 5University of Bergen, Department of Clinical Science, Bergen, NORWAY

Objective: The Losartan Intervention For Endpoint reduction in hypertension (LIFE) study was the first clinical outcome trial to suggest that lowering of serum uric acid by a uricosuric drug such as losartan has a beneficial effect on cardiovascular outcome independently of changes in renal function or blood pressure. LIFE also showed less new-onset atrial fibrillation in hypertensive patients receiving losartan vs. atenolol based treatment. Because losartan reduces serum uric acid levels, we investigated relations of serum uric acid with new-onset atrial fibrillation in the study.

Design and method: Hypertensive patients with electrocardiographic left ventricular hypertrophy and no prior atrial fibrillation (n = 8243) were treated for 5.0 ± 0.4 years with losartan or atenolol based therapy. Associations of serum uric acid with new-onset atrial fibrillation documented by Minnesota coding were assessed by Cox models using serum uric acid and systolic blood pressure as time-varying covariates to take into account changes of serum uric acid related to losartan or diuretic treatment, changes in renal function, and aging.

Results: Time-varying serum uric acid was associated with new atrial fibrillation defined by Minnesota code (HR = 1.19 per 16.8 umol/L [1 mg/dL] [95% CIs, 1.12–1.26], P < 0.0001), independent of losartan treatment (HR = 0.75 [95% CIs, 0.61–0.93], P = 0.007), older age (HR = 1.95 per 7.0 years [95% CIs, 1.73–2.20], P < 0.0001), male sex (HR = 1.46 [95% CIs, 1.09–1.94], P = 0.010) and higher Cornell voltage duration product (HR = 1.10 per 1023 msec x mm [95% CIs, 1.01–1.21], P = 0.034). Similar results were obtained in Cox models with serum uric acid levels partitioned according to baseline quartiles and in which atrial fibrillation was defined by physician reports or by both Minnesota coding and physician reports.

Conclusions: Thus, we found a strong relationship between time-varying serum uric acid levels during antihypertensive treatment and new-onset atrial fibrillation in elderly patients with left ventricular hypertrophy. Our data suggest that in-treatment serum uric acid is a strong predictor for new onset atrial fibrillation in hypertensive patients, independent of effects of antihypertensive treatment, age, sex, and electrocardiographic left ventricular hypertrophy. Further research is needed to clarify how serum uric acid may provoke atrial fibrillation.