

paroxysmal AF with/without LVA (PAF−/PAF+), persistent AF with/without LVA (PersAF±). The aim of the current analysis was to investigate NT-proANP and VCAM1 levels in peripheral and cardiac circulation and to analyze potential association with LAA strain.

**Method** The study included 116 patients undergoing first AF catheter ablation. Left atrial appendage (LAA) was analyzed before ablation with mid-esophageal echocardiographic in 2D-speckle tracking imaging. LAA total longitudinal strain (LAA-TLS) was assessed as the absolute difference of the maximal systo-diastolic values in extracted strain-curves. Blood plasma samples from femoral vein and LA were collected before catheter ablation. NT-proANP and VCAM1 were analyzed using commercially available assays.

**Results** There were significant differences between the groups with LAA-TLS ( $P < 0.001$ ), cardiac NT-proANP ( $P = 0.009$ ), and VCAM1 ( $P = 0.048$ ). On univariable analysis, age, gender, PersAF, LAA-TLS, renal function, and cardiac NT-proANP and VCAM1 significantly predicted LVA. However, on multivariable analysis, age (OR 1.097, 95%CI 1.009–1.192,  $P = 0.029$ ), PersAF (OR 4.713, 95%CI 1.131–19.649,  $P = 0.033$ ), LAA-TLS (OR 0.945, 95%CI 0.898–0.995,  $P = 0.032$ ) and VCAM1 (OR 1.002, 95%CI 1.000–1.004,  $P = 0.034$ ) remained significant predictors for LVA.

**Conclusion** Beside age and AF type, LAA-TLS and VCAM1 were significant predictors for LVA. Larger studies analyzing non-invasive predictors for electro-anatomical remodeling in AF patients are needed to prove our results.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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#### Poster n°36

### Analysis of NT-proBNP Baseline Levels in APOLLO as a Predictor of Survival in Hereditary Transthyretin-mediated (hATTR) Amyloidosis

M. Slama

Service Cardiologie, Hôpital Bichat, Paris, France

E-mail address: [prmslama@gmail.com](mailto:prmslama@gmail.com)

**Introduction** Hereditary transthyretin amyloidosis (hATTR) is a multisystemic, fatal disease resulting TTR amyloid deposition. Clinical manifestations include neuropathy as well as cardiomyopathy, a major cause of death. NT-proBNP, cardiac biomarker, has shown prognostic value in cardiac diseases clinically validated. For hATTR and wild-type ATTR, survival in patients with NT-proBNP levels  $> 3000$  ng/L was linked with poorer survival.

**Method** APOLLO, phase 3, randomized (2:1), double-blind study of patisiran 0.3 mg/kg or placebo IV q3W in patients with hATTR with polyneuropathy. Fifty-six% of patients had cardiac involvement defined by prespecified criteria: left ventricular (LV) wall thickness  $\geq 13$  mm and absence of aortic valve disease or hypertension.  $n = 225$ : mean age 61 years, 57% non-V30M mutation, NT-proBNP median 756.4 ng/L. To assess the prognostic significance of baseline factors on survival, a Cox regression analyses were conducted. NT-proBNP was evaluated as a continuous variable following logarithmic transformation as well as a binary variable using a cut off value of 3000 ng/mL.

**Results** Median survival follow-up duration was 18.7 months. 13 deaths not related to the treatment, 6 (8%) PBO arm and 7 (5%) in patisiran arm. NT-proBNP was the key significant factor predictive of survival based on univariate and multivariate analyses. The risk of death increased with higher baseline NT-proBNP (hazard ratio = 2.9) [95% CI: 1.8, 4.8,  $P$ -value =  $8.7 \times 10^{-7}$ ] per unit increment in log(NT-proBNP). Patients with NT-proBNP  $> 3000$  ng/L ( $n = 29$ ) had a 19.3-fold [95% CI 5.9, 62.8,  $p$ -value =  $8.7 \times 10^{-7}$ ] increased risk for mortality compared with those below 3000 ng/L ( $n = 196$ ).

**Conclusion** Based on the data from APOLLO, baseline NT-proBNP serum levels in hATTR patients are predictive of survival. These data underscore the importance of diagnosing and treating patients early in the course of the disease.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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#### Poster n°45

### Right ventricular dysfunction in heart failure with preserved ejection fraction

T. Filali\*, I. Ghissi, A. Ben Amor, S. Razkallah, R. Geha, P. Barsoum, F. Walylo, J.P. Maroni

Service de Cardiologie, Hôpital Robert-Ballanger, Aulnay-Sous-Bois, France

\* Corresponding author.

E-mail address: [filalithouraya@gmail.com](mailto:filalithouraya@gmail.com) (T. Filali)

**Introduction** The underlying pathophysiology of right ventricular (RV) dysfunction in heart failure with preserved ejection fraction (HFpEF) is still debated. The aim of this study is to assess the prognostic significance of echocardiographic right ventricular abnormalities in HFpEF.

**Method** We enrolled 150 patients with HFpEF in sinus rhythm and no history of chronic lung disease in this observational study. Over a median follow-up of 18 months, 58 patients (38.6%) reached the end point study of hospitalization for heart failure or death (group 1) and 92 remained asymptomatic (group 2).

**Results** While mean ages, sex ratio, BMI, creatinine level, left ventricular (LV) ejection fraction, LV and RV dimensions were similar between the 2 groups, group 1 patients compared to group 2, had higher ratio of early mitral diastolic inflow velocity E to early diastolic mitral annular velocity e' (E/e' ratio:  $17 \pm 6$  vs  $13 \pm 7$ ;  $P < 0.01$ ), higher pulmonary artery (PA) pressures ( $45 \pm 11$  vs  $36 \pm 12$  mmHg;  $P < 0.01$ ) with higher right heart filling pressures. Furthermore, group 1 patients had reduced RV function evidenced by reduced tricuspid annulus systolic velocities obtained at the basal RV free wall ( $8.9 \pm 2.1$  vs  $10.9 \pm 1.9$  cm/s;  $P < 0.01$ ), reduced tricuspid annular plane systolic excursion (TAPSE:  $14.9 \pm 2.4$  vs  $17.8 \pm 2.7$  mm;  $P < 0.01$ ) and reduced RV fractional area change (FAC:  $41 \pm 6$  vs  $48 \pm 7$ ;  $P < 0.01$ ).

**Conclusion** In HFpEF patients, right ventricular dysfunction progresses with increasing afterload PA pressures, and is associated with worse outcome.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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#### Poster n°46

### Impaired systolic function in heart failure with preserved ejection fraction: A specific phenotype?

F. Schrub<sup>1,\*</sup>, E. Oger<sup>1</sup>, L. Lund<sup>2</sup>, C. Linde<sup>2</sup>, E. Galli<sup>1</sup>, E. Donal<sup>1</sup>

<sup>1</sup> CHU Rennes, Rennes, France

<sup>2</sup> Karolinska hospital, Stockholm, Sweden

\* Corresponding author.

E-mail address: [florian.schrub@chu-rennes.fr](mailto:florian.schrub@chu-rennes.fr) (F. Schrub)

**Introduction** Background: Impairment in left ventricular (LV) systolic function has been described in heart failure (HF) with preserved ejection fraction (HFpEF), but the prognostic of HFpEF according to the degree of LV-systolic dysfunction has been weakly