10.2 years, CHA2DS2-VASc 4.9 ± 1.5) and 2,564 (age 73.9 ± 10.5 years, CHA2DS2-VASc 5.2 ± 1.4) treated/not treated with AA. Survival analysis showed a significant mortality reduction with AA adjusting for CHA2DS2-VASc and anticoagulation status (HR 0.65, 95% CI 0.60-0.72, p<0.001). Using propensity score matching, there was a lower mortality rate in patients treated with AA (Figure – HR 0.80, 95% CI 0.71 - 0.90, p<0.001).

Conclusion: There was an association between lower mortality rates in HFpEF patients with AF treated with AA. This may be due to an improvement in the atrial myopathy that complicates both AF and HFpEF as AA have been shown to reduce atrial fibrosis, dilation, and AF severity.

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**PO-628-06**

**EFFECT OF ANGIOTENSIN RECEPTOR NEPRILYSIN INHIBITOR AND EMPAGLIFLOZINE ON ISCHEMIC VENTRICULAR TACHYCARDIA SUBSTRATE: A HIGH-DENSITY MAPPING AND MRI STUDY**

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Background: Angiotensin receptor npeprilysin inhibitor (ARNI) and empagliflozine reduce cardiovascular death in heart failure, but mechanistic data on this effect are scarce.

Objective: To assess the effect of ARNI and empagliflozine on scar remodeling and ventricular tachycardia (VT) inducibility in a swine model of chronic myocardial infarction (MI).

Methods: Left anterior descending artery MI was induced in 40 Landrace Large X White pigs. Thirty-one animals (78%) survived the MI induction procedure and were randomized to receive treatment with beta-blocker (BB) only (group 1, n=7), empagliflozine+BB (group 2, n=8), ARNI + BB (group 3, n=8) or empagliflozine + ARNI + BB (group 4, n=8). Contrast-enhanced (ce)-MRI were performed at 2-day (baseline) and 1-month post-MI and were post-processed with ADAS 3D; ventricular scar subtypes and borderzone (BZ) corridors volume were measured. EP study with endo-epicardial high-density mapping was performed 1-month post-MI.

Results: VT was inducible in 21/31 subjects (68%). Treatment with ARNI (groups 3 and 4) showed a significant reduction of VT inducibility (OR 0.15 [0.026-0.91], p=0.03) compared to non-ARNI groups (100%, 75%, 50% and 50% for groups 1, 2, 3 and 4, respectively), representing a 30% relative risk reduction. Treatment with empagliflozine (group 2) did not reduce VT inducibility (p=0.704). Baseline ceMRI data showed a mean core and BZ scar mass of 5.4±3.1 and 6.1±3.1 grams, respectively, without significant differences in the percentage of core scar. The mean number of corridors was 2.1±2.2 with a mean mass of 1.3±1.0, without significant differences across groups. Treatment with ARNI and empagliflozine significantly reduced the number (+1.0, -1.1, -1.2 and -1.7 corridors for groups 1, 2, 3 and 4, respectively; p=0.021) and mass of corridors (+0.3, -1.6, -0.8 and -0.4 grams for groups 1, 2, 3 and 4, respectively; p=0.012) at the follow-up ceMRI. ARNI and empagliflozine groups tended to reduce overall BZ mass compared to the control group (reduction of the BZ percentage -1.2±4, -4.9±2, -5.6±2, -3.8±4; p=0.102).

Conclusion: Treatment with ARNI reduced VT inducibility by 30% after MI, likely driven by a significant reduction of the number and mass of BZ corridors. Empagliflozine reduced the number and mass of BZ corridors, without impact on VT inducibility.

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**PO-628-07**

**LEFT BUNDLE-BRANCH PACING POST-ATRIOVENTRICULAR JUNCTION ABLATION FOR ATRIAL FIBRILLATION: PROPENSITY-SCORE MATCHING WITH HIS BUNDLE PACING**

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Background: Left bundle branch pacing (LBBP) has emerged as a promising pacing modality to preserve physiological left ventricular activation but has limited prospective data evaluating