Implanted Cardiac-Defibrillator and Pacemaker Lead Function in Patient with Cardiac Amyloidosis

Introduction:
Cardiac amyloidosis (CA) results from myocardial infiltration of amyloid proteins that leads to restrictive cardiomyopathy, heart failure, and conduction abnormalities. Low QRS voltage on surface electrocardiography is characteristic of CA, raising concern for poor R-wave sensing and elevated pacing and defibrillation thresholds in CA patients with Implantable Cardioverter Defibrillator (ICD) or pacemaker. We sought to characterize the performance of implanted pacemaker and ICD leads among a cohort of patients with cardiac amyloidosis.

Methods:
We performed a retrospective analysis of 18 CA patients with ICD and pacemaker at Virginia Commonwealth University. Lead sensing, impedance, and capture threshold parameters were collected at implant and scheduled follow-up in 6-8 weeks and every 6 months thereafter. Mean follow-up was 30.7 ± 32 months. Significant changes in lead parameters were defined as a 50% reduction in sensed electrograms, > 50 ohms change in pacing impedance, and > 2-fold increase in chronic pacing threshold.

Results:
12 patients (66%) had dual-chamber ICD, 2 (11%) had biventricular ICD, 1 (5%) had single right ventricle lead ICD, and 3 (16%) had a pacemaker. The mean LV ejection fraction was 38 ± 14%. Appropriate ICD therapies occurred in 2 (11%) patients and inappropriate therapy occurred in 2 (11%) patients. The mean R-wave sensing amplitude at implant to last follow-up (8.5 ± 5.4 vs 7.79 ± 4.1), n=16. Significant changes were noted in 2 patients (12.5%) with >50% decrease in R-wave amplitude. However, they did not require lead revision for evidence of lead failure. P-wave sensing amplitude at implant to last follow-up (2.69 ± 0.96 vs. 2.47 ± 1.13) and significant difference was detected in 1 patient (8%). Significant difference was observed in pacing impedance; R atrium lead detected 7 patients (58%) with a >50 change in measured impedance (Ω) from implantation to last follow-up. Additional, the right ventricle lead detected 12 patients (75%) with significant changes. No significant difference was noted in right ventricular pacing threshold (0.69 ± 0.28 at implantation vs 1.09 ± 0.41 at last follow-up, at 0.4/0.5 ms).

Conclusion:
We observed significant changes in lead sensing and pacing impedance from implantation to last follow-up in our cohorts. We also noted equal number of appropriate and inappropriate ICD therapies. This study provides some insight into the stability of ICD and pacemakers lead parameters, however more investigation is needed.