Predictors and prognostic value of left ventricle branch block in hypertrophic cardiomyopathy

Cunha NPD[1]; Rigueira J.[1]; Placido R.[1]; Rodrigues T.[1]; Campos P.[2]; Pinto FJ.[1]; Almeida AG.[1]

[1]Santa Maria University Hospital/CHULN, CAML, CCUL, Lisbon School of Medicine, Universidade de Lisboa, Cardiology Department, Lisbon, Portugal
[2]Santa Maria University Hospital/CHULN, Radiology Department, Lisbon, Portugal

Funding Acknowledgements: Type of funding sources: None.

Introduction: Conduction abnormalities as left bundle branch block (LBBB) are common in myocardial disease and contribute to LV dyssynchrony and adverse LV remodeling. The relevance of LBBB in the context of hypertrophic cardiomyopathy (HCM) is unclear. The aim of this study is to find factors that are associated to LBBB in HCM and its impact in prognosis.

Methods: Retrospective single-center study of 36 consecutive patients (pts) with HCM defined by wall thickness≥15 mm in≥1 myocardial segments in CMR; pts with history of uncontrolled hypertension (HTN) and significant valvular disease were excluded.

Demographic, clinical, ECG and CMR data (including ventricular volumes, late gadolinium enhancement (LGE) and ventricular strain using feature tracking analysis (Circle CVi 42) were analyzed. For statistical analysis X2 test, Mann-Whitney and logistic regression model were used.

Results: Patient's median age was 63 years (IQR: 49,5-74,8), 64% men. 69% had controlled hypertension, 46% dyslipidemia and 23% diabetes; family history of sudden death and HCM occurred in 16% and 46% respectively. 42% had genetic study and mutations were identified in 25% (TNNT2: 8%; MYBPC3: 6%).

During a mean follow-up (FUP) of 17 ± 11 months, 24% had HF, 3% thromboembolic events, 26% new onset atrial fibrillation, 20% ventricular tachycardia (VT), 29% received an ICD and 3% died.

On ECG evaluation, 33% had intraventricular disturbance conduction with 12% having LBBB, 49% had LVH criteria.

On CMR, 81% had septal hypertrophy, 11% apical, 3% anterior-wall LVH and 6% lateral-wall hypertrophy. LVOTO was present in 33%. 69% of the patients had LGE (midwall: 61%, subendocardial: 11%, subepicardial: 3%; at segments with LVH: 47%, RV/LV insertion points: 25%, other:19.4%); the median LGE was 13.6g (IQR 6.7-22.4) corresponding to 7.4% of the LV mass (IQR 3.7-10.9). The median of the maximal wall thickness was 19mm (IQR 16.9-20.9), median LVEF was 70% (IQR 35-87); median LV indexed mass of 105 g/m2 (IQR 54.9-160.7). The median longitudinal strain in 4 and 2 chambers was -9.1 (IQR 15.6-4.6) and -9.1mm (IQR -16-2.6), respectively and the median radial strain in 4 and 2 chambers was 15.6 (IQR 6.5-28.2) and 13.7 (3.5-30.1), respectively.

Patients with LBBB had more VT and ICD implantation in follow-up ($p = 0.038$).

The presence of LGE in RV/LV insertion points ($p = 0.019$) and in the area of higher LVH ($p = 0.033$) were the only variables associated with LBBB. The area of LGE involving the RV/LV insertion points was an independent predictor of LBBB ($p = 0.02$, OR 36.0, IC:1.710-757.79).

Conclusion: In our sample, fibrosis in the RV/LV insertion points in CMR was an independent predictor of LBBB, which was associated with ventricular arrhythmias in follow-up. Further prospective studies with larger number of patients are needed to confirm our findings.