268 - Risk stratification in hypertrophic cardiomyopathy using ECG-based clustering and personalized computer simulations

A.Lyon[1*], R.Ariga[2], A.Mincholé[1], E.Zacur[3], P.Laguna[4], V.Grau[3], S.Neubauer[2], H.Watkins[2] and B.Rodriguez[1]

[1]Department of Computer Science, Oxford, UK
[2]Department of Cardiovascular Medicine, Oxford, UK
[3]Institute of Biomedical Engineering, Oxford, UK
[4]Department Biomedical Signal Interpretation & Computational Simulation Group, University of Zaragoza, CIBER-BBN, Spain
**aurore.lyon@cs.ox.ac.uk*

I. Introduction & Aim

Hypertrophic cardiomyopathy (HCM) is a common cardiac genetic disease and a leading cause of sudden cardiac death (SCD) in young adults. Yet, most patients remain asymptomatic and identifying high-risk patients to provide them with appropriate treatment is therefore critical [1]. It remains a challenge as the current electrocardiogram (ECG) biomarkers are not specific [2].

II. Methods

12-lead Holter ECG recordings for 86 HCM patients were analyzed. We developed signal processing and mathematical modelling techniques to extract morphological biomarkers from the ECG waveforms, and identified subgroups in the HCM population by clustering. We then compared left ventricular structure from cardiac magnetic resonance (CMR) imaging and arrhythmic risk for each group. In order to investigate the influence of HCM structural and electrophysiological abnormalities on the ECG, we developed a whole-body personalized modelling framework allowing the simulation of the ECG from CMR images using 3D volumetric meshes.

III. Results

Four distinct ECG phenotypes were identified, including Group 1A (n=21), with primary repolarization abnormalities (normal QRS with inverted T waves), and Group 3 (n=22), with QRS abnormalities and upright T waves. Interestingly, Group 1A had increased HCM Risk-SCD score [3] compared to other groups (3.8%, p=0.0004), and a trend to increased non-sustained ventricular tachycardia (NSVT). Group 3 had similar maximum wall thickness to 1A (21±6mm) but no increase in Risk-SCD or NSVT. The simulations based solely on cardiac anatomy led to normal QRS in Group 3 and upright T waves in Group 1A, showing that hypertrophy alone does not account for the identified ECG abnormalities but other structural changes may play a role. Current work focuses on integrating fiber disarray based on diffusion-tensor imaging to explain QRS abnormalities, and modelling conduction delays in areas of hypertrophy to investigate mechanisms of inverted T waves.

IV. Conclusions

These results suggest that computational ECG phenotyping can become a novel risk factor for stratification in HCM, and highlight the potential of personalized high performance computing simulations in understanding cardiac mechanisms.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

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