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Kiosk 11R-TC-04

Head-to-head Comparison and Temporal Trends of CMR Recommendations in ESC vs. ACC/AHA Guidelines

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Background: Although guidelines from the European Society of Cardiology (ESC) and American College of Cardiology (ACC)/American Heart Association (AHA) have been providing formal recommendations for CMR, its use in everyday clinical practice is not standardized. We aimed to provide a comprehensive head-to-head comparison and temporal analysis of CMR indications between the ESC and ACC/AHA guidelines to identify areas of consensus and divergence across the current landscape of CMR utilization.

Methods: ESC and ACC/AHA guidelines were systematically screened for recommendations related to CMR. We compared class of recommendation (COR) and level of evidence (LOE) for CMR in ESC and ACC/AHA guidelines and then categorized them according to diagnostic subgroups. Temporal evolution of CMR recommendations was analyzed by comparing current with previous recommendations.

Results: The latest ESC guidelines included 104 recommendations regarding CMR: 46 (44.2%) COR I, 33 (31.7%) COR IIa, 22 (21.1%) COR IIb, and 3 (2.9%) COR III. Five (4.8%)

recommendations had LOE-A, 35 (33.7%) LOE-B and 64 (61.5%) LOE-C, six mentioned CMR as free text. The latest ACC/AHA guidelines consisted of 88 recommendations: 53 (60.2%) COR I, 28 (31.8%) COR IIa, six (6.8%) COR IIb and one (1.1%) COR III. No recommendation was assigned a LOE-A (0%), 46 (52.3%) were classified as LOE-B, and 42 (47.7%) LOE-C.

The proportion of COR I and LOE B was higher in ACC/AHA (respectively, 60.2% vs. 44.2%, p-value=0.02; 52.3% vs. 33.7%, p=0.009). The increase of the number of CMR recommendations over time was significantly higher in ESC guidelines (from 59 to 104 vs. from 63 to 88, p=0.03).

Conclusion: ESC guidelines included more recommendations related to CMR use, whereas the ACC/AHA recommendations had higher COR and LOE. The number of CMR recommendations increased significantly over time in both societies, with a greater magnitude in the European guidelines.

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Investigation of the Modulatory Effect of Physical Activity on Genetic Variants Associated with Left Ventricular Mass

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Background: Left ventricular (LV) mass is a known prognostic cardiovascular biomarker with established genetic underpinnings, and is a particularly important phenotype in the context of heart muscle diseases. Physical activity holds interest as a risk factor as in general, it is protective against cardiovascular disease, however, in certain circumstances it can lead to deleterious remodelling. This gene-lifestyle interaction study examines whether physical activity attenuates the effect of genetic variants known to be associated with LV mass.

Methods: Genotype data (number of risk alleles) for 12 variants known to be associated with LV mass were retrieved for all participants in the UK Biobank. Of these, 42,309 had paired CMR and physical activity data. LV mass was indexed to body surface area. Physical activity levels in metabolic equivalent of task (MET)-minutes were determined from self-reported questionnaire data and standardised by categorising it into a dichotomous variable (physically active [top quintile] vs physically inactive [remaining 4 quintiles]). Interaction analyses were performed by including the variant x physical activity interaction term in an additive model, adjusting for age and sex with indexed LV mass (g/m²) as the outcome.

Results: Physical activity significantly attenuated the association between the *CLCN6* variant (rs143800963) and indexed LV mass (beta_{interaction} = 0.519 g/m², p_{interaction} = 0.039; i.e. 0.519 g/m² represents the difference in the LV mass-increasing effect of the risk allele between the physically active and inactive individuals) (Table 1). The magnitude of the effect of the rs143800963 risk allele on LV mass was nearly 2x larger in physically active individuals than in inactive individuals. The *CLCN6* gene, which codes for a voltage-gated chloride channel, forms part of the *MTHFR-CLNCN6-NPPA*

NPBB gene cluster which is known to be associated with regulation of NT-proBNP levels, an important heart failure biomarker.

Conclusion: Physical activity may play a role in modulating genetic predisposition to increased LV mass in certain individuals at specific loci. This study highlights the potential of gene-lifestyle interaction investigations to inform how certain lifestyle factors can confer heightened risks or exert protective effects for certain individuals. Future work will focus on external replication of these findings, and investigating variants associated with known cardiovascular conditions.

Table 1: Effect of the interaction between LV mass-associated variants and physical activity on indexed LV mass

Variant (effect allele)	Gene	Main effect in active individuals (n=11,077)		Main effect in inactive individuals (n=31,051)		Variant x PA Interaction (n=42,309)	
		Beta (95% CI)	p-value	Beta (95% CI)	p-value	Interaction beta (95% CI)	p-value
rs143800963 (C)	CLCN6	1.058 (0.613, 1.503)	3E-06	0.538 (0.287, 0.788)	3E-05	0.519 (0.024, 1.014)	0.039
rs5503451 (T)	MAPT	-0.478 (-0.708, -0.249)	5E-05	-0.263 (-0.39, -0.136)	5E-05	-0.215 (-0.468, 0.039)	0.097
rs199501 (A)	WNT3	0.464 (0.227, 0.702)	0.0001	0.285 (0.153, 0.418)	2E-05	0.188 (-0.075, 0.452)	0.161
rs388498 (G)	CENPW	-0.262 (-0.54, 0.015)	0.0639	-0.456 (-0.61, -0.302)	<1E-06	0.198 (-0.109, 0.504)	0.207
rs2621197 (C)	ADAMTS10	0.183 (-0.4, 0.766)	0.5379	0.605 (0.283, 0.927)	0.0002	-0.413 (-1.057, 0.231)	0.209
rs34183229 (G)	SYNPOL	-0.455 (-0.745, -0.166)	0.002	-0.269 (-0.431, -0.106)	0.0012	-0.17 (-0.491, 0.151)	0.298
rs28502516 (C)	KDM2B	-0.365 (-0.649, -0.081)	0.0119	-0.103 (-0.351, -0.038)	0.0162	-0.142 (-0.456, 0.173)	0.377
rs2255167 (T)	TTN	0.657 (0.4, 0.913)	<1E-06	0.567 (0.424, 0.71)	<1E-06	0.104 (-0.18, 0.389)	0.472
rs3729989 (T)	MYBPC3	-0.404 (-0.699, -0.11)	0.0071	-0.298 (-0.462, -0.134)	0.0004	-0.112 (-0.438, 0.214)	0.501
rs10497529 (G)	CCDC141	1.179 (0.651, 1.707)	1E-05	1.096 (0.801, 1.39)	<1E-06	0.105 (-0.479, 0.69)	0.724
rs56252725 (G)	PDXDC1	0.18 (-0.127, 0.488)	0.2505	0.161 (-0.004, 0.326)	0.0558	0.021 (-0.314, 0.357)	0.901
rs5858541 (A)	IGF1R	-0.297 (-0.509, -0.084)	0.0083	-0.294 (-0.412, -0.176)	1E-06	-0.001 (-0.237, 0.234)	0.991

All models are adjusted for age and sex. LV mass is indexed to body surface area. Beta is the increase in indexed LV mass (g/m²) per effect allele of the variant; interaction beta is the difference in trait per effect allele comparing physically active individuals to inactive individuals

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Kiosk 11R-TC-06

Mr-compatible 12-lead ECG Recording During Atrial Flutter Ablation Procedure in the Interventional Cardiac Magnetic Resonance (iCMR) Suite: Exploring the Magneto-hydrodynamic (MHD) Effect

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Background: The presence of flutter waves in the 12-lead surface electrocardiogram (ECG) is an important diagnosis tool for cavotricuspid isthmus (CTI) dependent atrial flutter (AFL). Recently, it has been shown in several centers that ablation of CTI dependent AFL can successfully be performed entirely in the interventional cardiac magnetic resonance (iCMR) suite using real-time visualization of the heart tissue, intracardiac electrograms (EGM) and a 3-lead-ECG MR-compatible patient monitoring system (Expression MR400, Philips). The magneto-hydrodynamic (MHD) effect caused by blood flow in a static magnetic field plays a crucial role in the use of surface ECG in MRI. The MHD impact on the ECG signal depends on technical and electrophysiological parameters such as electrode configuration, heart rate and arrhythmia. Recently, a 12-lead-ECG system (MiRTLE Medical) has become available for use in MRI

offering new possibilities to study the influence of this effect on the ECG of patients with arrhythmias.

Methods: As previously shown CTI ablation performed in a 1,5 Tesla iCMR suite is feasible and safe using intracardiac signals derived from the ablation catheter tip and surface cardiac signal from the 3-lead ECG MR-compatible patient monitoring system. A case of a CTI ablation performed in the 1,5 Tesla iCMR suite with the use of the 12-lead ECG MR-compatible monitoring system in addition to the 3-lead ECG monitoring system is described exploring the ability to identify flutter waves in real-time. ECG traces were acquired from the MR-compatible 12-lead ECG system outside the scanner during patient preparation and inside the scanner without imaging sequences running during the procedure. No real-time MHD Filtering has been performed during acquisition of the 12-lead ECG. Inside and outside scanner comparisons of the 12-lead ECG have been performed after the case.

Results: A patient with biological aortic valve presented with symptomatic new-onset CTI-dependent atrial flutter and fast ventricular conduction. The patient was in flutter during patient preparation and part of the procedure which allowed the recording of flutter waves. Catheter placement and CTI ablation was performed entirely in the iCMR suite guided by active catheter imaging, intracardiac signals and 3-lead ECG. Procedure time was 80 min with an RF delivery time of 10 min.

Conclusion: Despite the presence of a sternal cerclage and biological aortic valve MRI imaging was feasible. Flutter waves could be clearly identified on the MiRTLE 12-lead ECG during patient preparation outside the scanner bore. The 12-lead ECG recorded inside the 1,5 Tesla scanner showed that the MHD effect is present obscuring the flutter waves but diminishing over the cardiac cycle. Beat detection showed promising results. This encourages for further exploration of the MHD effect in more flutter patients with the perspective to provide interpatient MHD signal comparison also allowing the investigation of the influence of electrophysiological parameters.

