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# Exploring the Multifaceted Nexus of Hypertrophic Cardiomyopathy and Clinical Outcomes

LETTER TO THE EDITOR

To the Editor,

The recent article by Babur Güler et al[1] entitled "Phenotypic, Epidemiologic, and Imaging Features of Hypertrophic Cardiomyopathy: A Single-Center Experience" was read with great interest. The authors reported a comprehensive cohort of 701 patients, providing valuable real-world insight into the phenotypic spectrum of hypertrophic cardiomyopathy (HCM) in Türkiye where data about this subject remain limited. The study was designed as a single-center, cross-sectional, retrospective, and observational study. The study's strengths include its large sample size, systematic phenotypic classification (including the under-recognized latent obstructive subgroup), and extensive use of multimodality imaging, particularly cardiac magnetic resonance (CMR). The high utilization rate of CMR (84%) and the detailed assessment of late gadolinium enhancement (LGE) widely enhance the power of phenotypic characterization. Moreover, the inclusion of genetic data (even in a subset (32%)) adds important translational relevance, especially with the predominance of MYBPC3 and MYH7 mutations aligning with worldwide literature.[2-4]

However, we would like to highlight several points that may further enrich the interpretation and clinical applicability of the findings: First, the relatively short median follow-up duration (13 months) limits the ability to draw conclusions regarding long-term outcomes, particularly sudden cardiac death (SCD) risk and progression toward advanced heart failure.[3-5] Given the evolving paradigm in HCM (where heart failure-related morbidity may increasingly exceed arrhythmic mortality), longitudinal data from this cohort would be of high value. Second, while the study provides detailed phenotypic comparisons, the absence of multivariable analyses limits the ability to identify independent predictors of adverse outcomes. For instance, the observed higher NT-proBNP levels and mitral regurgitation prevalence in the resting-obstructive group are clinically meaningful, but their independent prognostic significance remains unclear. Future analyses incorporating Cox regression or competing-risk models would strengthen causal inference.[5-7] Third, the findings related to apical HCM and the lack of data about the mid-ventricular obstruction phenotype HCM deserve particular emphasis. The high prevalence of LGE (95%) and extensive LGE burden, along with increased apical aneurysm rates, raise important questions regarding current risk stratification models.[2] These observations support the growing notion that apical HCM may not be as benign as historically perceived and may warrant phenotype-specific risk algorithms beyond the current ESC HCM Risk-SCD model. Furthermore, patients with the mid-ventricular obstruction phenotype HCM have not been mentioned in the paper who are shown to be presented with apical aneurysms, arrhythmic complications of HCM, and major adverse cardiac outcomes compared to other phenotypes.[8-10] There was also no data about the patients with the dilated phase (burn-out) of HCM in which the prognosis is generally poorer compared to standard HCM.[11,12] Fourth, the relatively low rate of genetic testing reflects real-world limitations; however, it also introduces potential selection bias in genotype–phenotype correlations. Expanding genetic screening and incorporating polygenic or



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modifier analyses could help clarify the unexpectedly similar mutation rates observed across phenotypes, particularly in apical HCM and response to medical therapy.[13] Finally, an important trending consideration is the absence of patients treated with cardiac myosin inhibitors. As these agents are increasingly integrated into clinical practice, future studies from this cohort may provide valuable insights into their impact on phenotype expression, symptom burden, and biomarker profiles.

In conclusion, this study represents a significant contribution to regional HCM literature and provides a solid basis for future prospective, multicenter, and longitudinal investigations. Further integration of advanced statistical modeling, extended follow-up, and emerging therapies will be essential to fully clarify phenotype-specific risk and optimize personalized management strategies.

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