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Reply to the Letter to the Editor: "Exploring the Multifaceted Nexus of Hypertrophic Cardiomyopathy and Clinical Outcomes"

LETTER TO THE EDITOR
REPLY

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To the Editor,

We would like to express our sincere gratitude to the authors for their insightful comments[1] and interest in our study, "Phenotypic, Epidemiologic, and Imaging Features of Hypertrophic Cardiomyopathy: A Single-Center Experience."[2] Their observations regarding the long-term clinical applicability and the evolving landscape of hypertrophic cardiomyopathy (HCM) provide a valuable extension to our discussion. We would like to address the points raised to further clarify the scope and contributions of our research.

First of all, the primary goal of our investigation was to provide a robust, large-scale, cross-sectional characterization of HCM phenotypes within a tertiary center in Türkiye, where comprehensive regional data have historically been scarce. As reflected in our title and methodology, the focus was centered on "Phenotypic, Epidemiologic, and Imaging Features." Our study was designed to serve as a foundational descriptive registry rather than a primarily prognostic or predictive analysis. While we documented clinical outcomes such as mortality and implantable cardioverter-defibrillator (ICD) interventions, these parameters were intended to offer a contemporary "snapshot" of the disease burden in the cohort at the time of evaluation.

The authors correctly noted the median follow-up of 13 months. While this duration is relatively short for a chronic and slowly progressive condition like HCM,[3] it was sufficient to fulfill our primary objective of phenotypic characterization. We explicitly acknowledged in our discussion that the mortality (2.9%) and ICD data should be interpreted as preliminary and descriptive. Given the relatively low number of clinical events recorded during this initial phase, performing multivariable Cox regression or competing-risk models would have been statistically underpowered and prone to overfitting, potentially leading to misleading prognostic inferences.

We share the authors' interest in the apical HCM subgroup. Our data, which showed a 95% prevalence of late gadolinium enhancement (LGE) and the highest rate of extensive LGE (37%) in this group, strongly support the emerging consensus that apical HCM may carry a higher risk than historically perceived.[4,5] By highlighting these features along with apical aneurysms (9.7%), we aimed to underscore the diagnostic power of cardiovascular magnetic resonance in identifying high-risk markers that might be overlooked by traditional risk models.[6]

We entirely agree with the authors regarding the clinical significance and management challenges associated with mid-ventricular obstruction (MVO) and the "burn-out" phase of HCM. While MVO was not categorized as a separate primary group in our initial report, we acknowledge that the management of these patients remains particularly daunting due to the current scarcity of high-level evidence and standardized treatment algorithms.[7] It is evident that larger datasets and more robust evidence are required to clarify the natural history and optimal therapeutic strategies for this subgroup, and we are in complete agreement

with the authors on this necessity. Similarly, we concur that the "burn-out" phenotype represents a critical frontier in HCM management.[8,9] We intend to focus more intensively on these two specific areas in our upcoming research, as they represent significant gaps in the current literature that demand urgent academic attention.

The authors raise a valid point regarding the potential selection bias introduced by the 32% genetic testing rate. Beyond the logistical hurdles, the more complex issue lies in the interpretation of identified variants and the execution of comprehensive cascade family screening.[10] Although genetic analysis is a standard protocol in the 5-year multidisciplinary cardiomyopathy clinic, several factors have affected the current data yield. The extended processing time for genetic tests, combined with the comprehensive nature of the cohort—which includes patients with dilated cardiomyopathy, arrhythmogenic cardiomyopathy, and non-dilated left ventricular cardiomyopathy—has resulted in a lower rate of finalized genetic results for the hypertrophic cardiomyopathy subgroup at the time of this study. We firmly believe that genetic evaluation should be an integral part of the diagnostic workup for all HCM patients, and we anticipate that publications reflecting the expanded genetic data will increase in the near future.

Regarding the absence of cardiac myosin inhibitor therapy in the cohort, clarification is required regarding the regulatory and clinical context in the region. During the study period (October 2021 to November 2024), these agents were not yet approved for clinical use by national regulatory authorities in Türkiye. Even currently, despite global advancements, access to these specialized therapies remains significantly restricted due to ongoing reimbursement and availability challenges in the domestic healthcare system.

In summary, the study fulfills its intended purpose of providing a detailed regional epidemiological foundation. We believe that these baseline descriptive data are an essential prerequisite for the multicenter, longitudinal investigations suggested by the authors. We are committed to continuing our follow-up to provide the academic community with more robust prognostic insights in the years to come.

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