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# The Long-Term Mortality Predictors in Hypertrophic Cardiomyopathy Patients with Low Risk of Sudden Cardiac Death

## Ani Kardiyak Ölüm Riski Düşük Hipertrofik Kardiyomiyopati Hastalarında Uzun Vadeli Mortalite Belirleyicileri

**LETTER TO THE EDITOR**
EDİTÖRE MEKTUP

To the Editor,

I read with interest the recent article by Kalenderoğlu et al.,[1] entitled Long-Term Mortality Predictors in Hypertrophic Cardiomyopathy Patients with Low Risk of Sudden Cardiac Death. I would like to congratulate the authors on addressing an important and under-researched clinical issue by investigating long-term mortality predictors in patients with hypertrophic cardiomyopathy (HCM) who are considered to be at low risk according to current guidelines.

Identifying advanced age, cerebrovascular accident, and elevated neutrophil count as independent predictors of long-term mortality in patients with an HCM Risk-sudden cardiac death (SCD) score of less than 4% highlights the limitations of the HCM Risk-SCD model. This study emphasizes the need to integrate additional clinical and laboratory parameters into HCM risk models, especially for patients with an HCM Risk-SCD score below 4%.

However, I would like to respectfully offer a few points for your consideration:

As the primary endpoint was all-cause mortality, the results would have been more clearly interpreted if a competing risk analysis had distinguished between cardiovascular and non-cardiovascular causes of death. Given the advanced average age of the study population, such an analysis would clarify whether the identified predictors reflect cardiac-specific risk or systemic vulnerability in general. Additionally, 34% of the study population had coronary artery disease and 19% had undergone revascularization, both of which increase the risk of sudden and non-sudden cardiac death independent of the underlying disease. It is therefore difficult to attribute the increased all-cause mortality risk solely to HCM without a control group. Furthermore, a comparison between patients who experienced long-term mortality and those who survived could add valuable data for the study. An event-based subgroup analysis could provide a clearer understanding of which variables truly differentiate high-risk individuals within this ostensibly low-risk group.

While identifying neutrophil count as an independent risk factor, the reported median neutrophil values across the three groups were within or only slightly above the upper limit of normal. As neutrophil count is a non-specific inflammatory marker, it would be valuable to know whether potential confounders such as subclinical infection, smoking status, or chronic inflammatory conditions were considered or adjusted for in the analysis. Furthermore, in a disease in which chronic low-grade inflammation plays a pathophysiological role, including additional inflammatory markers such as C-reactive protein (CRP) and the neutrophil-to-lymphocyte ratio (NLR) would have provided a more comprehensive assessment of the inflammatory burden and its prognostic significance. Incorporating cardiac magnetic resonance imaging (MRI) data, such as myocardial fibrosis quantification and late gadolinium enhancement, would also likely provide additional prognostic information in this population. Future studies investigating the added value of cardiac magnetic resonance-derived (CMR-derived) fibrosis burden in patients with a low HCM Risk-SCD score could substantially improve long-term risk stratification.

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The prevalence of atrial fibrillation (AF) in the study cohort was also substantial, ranging from 20% to 32% across tertiles. Similarly, coronary artery disease (CAD) was present in approximately one-third of the population. Both conditions are well established as risk factors for adverse outcomes in hypertrophic cardiomyopathy, including thromboembolic events, heart failure, and mortality.[2-4] However, neither AF nor CAD was included or reported in the univariable or multivariable analyses presented in Table 3. Given their known prognostic importance, particularly in an older HCM population, this information would meaningfully enhance the interpretation of the study's findings.

As the study is retrospective and single-center, its nature may limit the generalizability of the results to different populations. To increase the validity and reliability of the findings, prospective studies should include patient groups from different geographic regions and with different clinical characteristics.

In conclusion, Kalenderoğlu et al. have made a meaningful contribution to our understanding of long-term risk in patients with HCM who have low HCM Risk-SCD scores. I once again congratulate the authors and believe that addressing these points would further enhance the clinical applicability and robustness of their findings.

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