

Editorial

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Should We Use Renin-Angiotensin-Aldosterone System Inhibitors Routinely in Patients With Hypertrophic Cardiomyopathy?

Jae-Hyeong Park 💿, MD, PhD

Department of Cardiology in Internal Medicine, Chungnam National University, School of Medicine, Chungnam National University Hospital, Daejeon, Korea

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Correspondence to

Jae-Hyeong Park, MD, PhD

Department of Cardiology, Internal Medicine, School of Medicine, Chungnam National University, Chungnam National University Hospital, 282, Munhwa-ro, Jung-gu, Daejeon 35015, Korea. Email: jaehpark@cnu.ac.kr

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ORCID iDs

Jae-Hyeong Park (D) https://orcid.org/0000-0001-7035-286X

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Conflict of Interest

The author has no financial conflicts of interest.

▶ See the article "Prognostic and Safety Implications of Renin-Angiotensin-Aldosterone System Inhibitors in Hypertrophic Cardiomyopathy: A Real-world Observation over 2,000 Patients" in volume 53 on page 606.

Hypertrophic cardiomyopathy (HCM) is a relatively common inherited disease characterized by pathologic ventricular thickening and decreased compliance with variable and complex clinical phenotypes and prognosis.¹⁾²⁾ Physicians should pay attention to the management of this condition, as appropriate and comprehensive treatment can reduce mortality and morbidity in these patients. In this current issue, Park et al.³⁾ reported their data about the clinical outcomes of using renin-angiotensin-system inhibitors (RASi) in patients with HCM. Although RASi has been recommended as the standard of care for heart failure,⁴⁾ the role of RASi in HCM has not been well studied. They analyzed 2,104 patients with HCM in two tertiary university hospitals and checked their clinical outcomes according to the use of RASi. The authors found that the use of RASi was not associated with poorer clinical outcomes, even though the RASi group had worse baseline clinical profiles. They also showed that the use of RASi may be associated with favorable clinical outcomes in patients without left ventricular outflow tract obstruction (LVOTO), although this did not reach statistical significance.

This result was consistent with the previous study with a sufficient number of patients that HCM patients with hypertension had lower mortality rates than those without hypertension, possibly related to the use of antihypertensive medication.⁵ Patients with HCM are known to have an over-activated neurohumoral status; the use of RASi, a potent neurohormonal modulator, may be beneficial in HCM patients.⁶ The use of RASi did not increase LVOTO, contrary to the authors' fears that RASi use would increase LVOTO due to its vasodilatory effect.

Before considering the routine use of RASi in all HCM patients as a result of this study, we need to consider the limitations of this study. Firstly, this was a retrospective study, which means that it was not possible to control for the reason why RASi was used. Even if the study had adjusted for the presence of hypertension or systolic blood pressure using statistical techniques, it would still not be sufficient. Second, it would have been better if the authors had stratified their study group according to the extent of fibrosis as assessed by cardiac magnetic resonance imaging or the presence/absence of subclinical left ventricular systolic dysfunction as assessed by echocardiography. Third, HCM is a genetic disease and prognosis may be influenced by genetic variation.⁷⁾ If further research can demonstrate the effectiveness of RASi based on genetic variants, it would be beneficial for HCM patients with similar genetic variants.



Data Sharing Statement

The data generated in this study is available from the corresponding author upon reasonable request.

The contents of the report are the author's own views and do not necessarily reflect the views of the *Korean Circulation Journal*. Should we use RASi routinely in patients with HCM? Not yet. However, based on the results of this study, RASi can be used safely in HCM patients, especially in the absence of LVOTO. If future well-controlled prospective studies confirm the efficacy of use of RASi, it will provide evidence for the appropriate use of RASi and further improve clinical outcomes in HCM patients by using RASi.

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