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EDITORIAL
Valvular heart disease

Aortic valve sclerosis in heart failure: marker of severity or harbinger of progression?

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This editorial refers to 'Prognostic significance of early stage aortic valve disease development in heart failure: insights from the MECKI score cohort', by V.A. Myasoedova *et al.*, https://doi.org/10.1093/ehjopen/oeaf066.

Heart failure (HF) remains a major public health burden, with its prevalence steadily increasing as population age and survival after acute cardiac events improves. Despite advances in pharmacological and device-based therapies, the prognosis for many patients with HF remains guarded. Identifying reliable, accessible markers that can help stratify risk and guide the intensity of follow-up is thus a central objective in contemporary cardiology.[1]

Aortic valve (AV) sclerosis, characterized by focal or diffuse thickening and calcification of the AV leaflets without significant obstruction, has long been considered a benign, age-related echocardiographic finding. However, mounting evidence suggests that AV sclerosis is not merely a passive bystander, but may in fact reflect an active process of valvular and vascular remodelling, inflammation, and atherosclerosis.[2,3] In the general population, AV sclerosis has been linked to increased cardiovascular morbidity and mortality, but its significance in the context of established HF has remained unclear.[4]

The study by Myasoedova et al., leveraging the Metabolic Exercise test data combined with Cardiac and Kidney Index (MECKI) score cohort, provides important new insights into this question.[5] The authors evaluated 1397 patients with HF, carefully documenting the presence of AV sclerosis at baseline and tracking its development over time through serial echocardiographic assessments (*Figure 1*). Their analysis focused on the following two key questions: (i) Does the presence of AV sclerosis at baseline predict adverse outcomes in HF? (ii) Does the new development of AV sclerosis during follow-up carry additional prognostic significance?

At baseline, AV sclerosis was present in just over half of the cohort (50.6%). As expected, these patients were older and had more

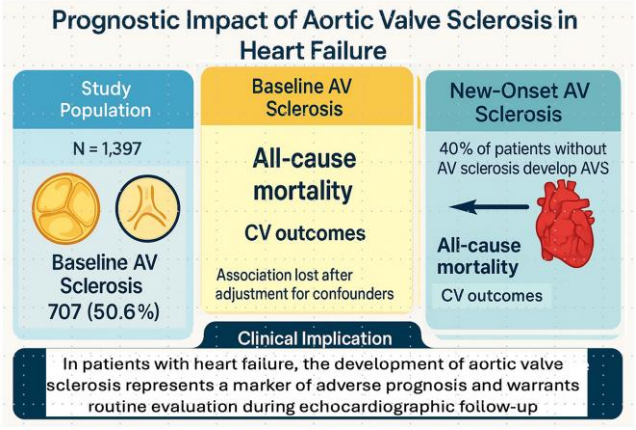

Figure 1 Prognostic impact of aortic valve sclerosis in heart failure (Metabolic Exercise test data combined with Cardiac and Kidney Index score cohort). Baseline aortic valve sclerosis was modestly associated with increased risk, while new-onset aortic valve sclerosis strongly predicted all-cause mortality and cardiovascular outcomes. Routine echocardiographic monitoring may support early risk stratification.

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advanced HF, as reflected by clinical and echocardiographic parameters. Over a median follow-up of 5 years, the presence of AV sclerosis at baseline was associated with increased all-cause mortality and adverse cardiovascular outcomes. However, after adjusting for differences in age and disease severity, this association was attenuated and lost statistical significance. In other words, baseline AV sclerosis appeared to be more a marker of underlying disease burden than an independent risk factor for adverse outcomes.

The most novel and clinically relevant finding emerged from the longitudinal analysis of patients who did not have AV sclerosis at baseline. Among these individuals, more than 40% developed AV sclerosis during follow-up. Strikingly, the new onset of AV sclerosis was associated with a three- to six-fold increase in the risk of all-cause mortality and composite cardiovascular outcomes, even after adjusting for age. This underscores the potential importance of dynamic changes in valvular structure as a marker of disease progression in HF. The clinical implications of these findings are both practical and thought-provoking. First, the data suggest that while AV sclerosis at the time of HF diagnosis may simply reflect advanced age and disease severity, the subsequent development of AV sclerosis during follow-up is a powerful harbinger of worsening prognosis. This distinction is crucial: it is not the static presence of AV sclerosis, but rather its dynamic appearance over time that should alert clinicians to a change in the patient's risk profile.

From a practical standpoint, this means that routine echocardiographic surveillance in patients with HF should include careful assessment of the AV, not only to monitor for the development of significant stenosis, but also to detect the new onset of sclerosis. When AV sclerosis is newly identified in a patient previously free of this finding, it may be prudent to intensify clinical follow-up, optimize medical therapy, and address modifiable risk factors more aggressively. Such a strategy could help prevent further cardiovascular events and potentially improve long-term outcomes, although this hypothesis will need to be tested in future interventional studies. It is also worth noting that AV sclerosis is easily and reproducibly detected by standard transthoracic echocardiography, making it an accessible marker for clinicians in a variety of practice settings. Its presence can be incorporated into existing risk stratification models, such as the MECKI score, to refine prognostic estimates and guide shared decision-making with patients and families.

Nevertheless, several important limitations must be acknowledged. The number of patients who developed AV sclerosis and experienced adverse events was relatively small, leading to wide confidence intervals and limited statistical power. The observational nature of the study precludes definitive conclusions about causality, and the potential for residual confounding cannot be excluded, even after age-matching and adjustment. The frequency and timing of echocardiographic follow-up were not standardized, but rather driven by clinical need, which may have introduced detection bias. Finally, while the association between new-onset AV sclerosis and adverse outcomes is compelling, it remains unclear whether interventions triggered by this finding can actually alter the clinical trajectory of HF.

Despite these caveats, the study by Myasoedova et al. represents an important step forward in our understanding of the interplay between valvular and myocardial disease in HF. It reminds us that the heart is a dynamic organ, and that subtle changes in structure, such as the development of AV sclerosis, can provide valuable clues about underlying pathophysiology and future risk.

In summary, AV sclerosis should no longer be dismissed as a benign, incidental finding in patients with HF. While its presence at baseline reflects the cumulative burden of age and disease, its new development during follow-up is a dynamic marker of disease progression and a strong predictor of adverse outcomes. Clinicians should remain vigilant for the appearance of AV sclerosis in their patients with HF and consider it a call to action: to reassess risk, intensify management, and engage patients in discussions about prognosis and goals of care. Future research should focus on validating these findings in larger, more diverse cohorts, and on testing whether targeted interventions in patients who develop AV sclerosis can improve clinical outcomes. Until then, the message remains clear: in managing HF, paying close attention to the evolving story of the AV may help anticipate complications before they arise and provide patients with the best possible chance for a longer and healthier life.

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References

1. Savarese G, Lund LH. Global public health burden of heart failure. *Card Fail Rev* 2017;**3**:7–11.
2. Agmon Y, Khandheria BK, Meissner I, Sicks JD, O'Fallon WM, Wiebers DO, Whisnant JP, Seward JB, Tajik AJ. Aortic valve sclerosis and aortic atherosclerosis: different manifestations of the same disease? *J Am Coll Cardiol* 2001;**38**:827–834.
3. Di Minno MND, Di Minno A, Ambrosino P, Songia P, Pepi M, Tremoli E, Poggio P. Cardiovascular morbidity and mortality in patients with aortic valve sclerosis: a systematic review and meta-analysis. *Int J Cardiol* 2018;**260**:138–144.
4. Otto CM, Lind BK, Kitzman DW, Gersh BJ, Siscovick DS. Association of aortic-valve sclerosis with cardiovascular mortality and morbidity in the elderly. *N Engl J Med* 1999;**341**:142–147.
5. Myasoedova VA, Salvioni E, Bonomi A, Galotta A. Prognostic significance of early-stage aortic valve disease development in heart failure: insights from the MECKI score cohort. *Eur Heart J Open* 2025.