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Editorial

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Device-Detected Subclinical Atrial Fibrillation as Fire Under the Ashes

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It is well known that clinical atrial fibrillation (AF) can reduce the effectiveness of cardiac resynchronization therapy (CRT) for patients with heart failure (HF) and ventricular dyssynchrony.[1)2)] During AF rhythm, the percentage of biventricular pacing (BiV-P%) can significantly decrease by rapidly conducting dyssynchronous rhythms, fusion beats, and pseudofusion pacing, consequently compromising the inter-/intraventricular resynchronization. In addition, development of AF resulted in loss of atrial mechanical contraction and the inability of atrial synchronous BiV-P, impairing atrioventricular synchrony.[1)3)] However, the clinical implications of device-detected subclinical AF (SCAF), reported in 20% to 30% of patients with CRT, have not been as fully investigated as clinical AF.[4)5)]

In a recent retrospective study in the *Korean Circulation Journal*, Yoon et al.[6)] provided us with valuable information on the adverse effect of device-detected SCAF in patients with CRT. Among 120 CRT patients without a prior history of AF, 19 (15.8%) had device-detected SCAF, defined as atrial high-rate episodes ≥180 beats per minute lasting 6 minutes or longer, during a median follow-up of 25.1 months. Patients with device-detected SCAF exhibited a significantly lower 'optimal BiV-P%' (defined as ≥98%) and a higher incidence of HF hospitalization, cardiovascular death, and all-cause death than those without. Interestingly, patients with device-detected SCAF and those with preexisting AF showed no significant differences regarding the BiV-P% and clinical outcomes.

Unfortunately, in this study, clinical outcomes or BiV-P% was only evaluated according to the presence or absence of device-detected SCAF. More detailed analyses depending on SCAF burden would have been more informative. Additionally, the average BiV-P% was derived only from the last interrogation, not through the entire follow-up duration. However, their results were sufficient to elucidate the importance of device-detected SCAF for better management of patients with CRT.

Device-detected SCAF is also known to be strongly associated with the risk of progression to clinical AF, ischemic stroke, and inappropriate shock in addition to HF aggravation or increased mortality.[4)5)7)] BiV-P% can also be overestimated by the SCAF-induced fusion/pseudofusion beats because these pacing beats are, although ineffective, erroneously counted as BiV-P by most CRT devices.[8)]

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Therefore, more attention needs to be paid to the device interrogation results for the possibility of SCAF, particularly in CRT patients with no or suboptimal response. If the burden of SCAF is not significant, antiarrhythmic drugs may be effective to control SCAF and maximize BiV-P%. Automated CRT algorithms designed to speed up BiV-P rates can help increase BiV-P% during SCAF rhythm.[9] However, when the burden of SCAF increases despite medical treatment, AF ablation may be required. If AF ablation is not feasible or effective, atrioventricular node (AVN) ablation can be an alternative.[2][3] In previous studies, AF patients treated with AVN ablation showed as favorable a prognosis as patients with sinus rhythm following CRT implantation.[2] Finally, anticoagulation may be needed when the SCAF burden exceeds 24 hours because previous data reported that patients with SCAF >24 hours were at increased risk of stroke.[7][10]

In conclusion, device-detected SCAF, which can be elusive or underdiagnosed without device interrogation, may be just as closely associated with various adverse cardiovascular outcomes as clinical AF. Therefore, optimal management of device-detected SCAF may be important to achieve a better prognosis of patients with CRT. Prospective studies are worth conducting to find the optimal cutoff of SCAF burden for more aggressive rhythm control or anticoagulation therapy.

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