Korean Circ J. 2019 Jul;49(7):612-614
https://doi.org/10.4070/kcj.2019.0116
pISSN 1738-5520·eISSN 1738-5555

kcj
Korean Circulation Journal

Editorial

Ventricular Conduction Disturbance in Acute Heart Failure Syndrome: Does It Matter for Prognosis?

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▸ See the article "Prognostic Implication of Ventricular Conduction Disturbance Pattern in Hospitalized Patients with Acute Heart Failure Syndrome" in volume 49 on page 602.

Received: Apr 14, 2019
Accepted: Apr 16, 2019

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Conflict of Interest
The author has no financial conflicts of interest.

The contents of the report are the author's own views and do not necessarily reflect the views of the *Korean Circulation Journal*.

Delayed intraventricular conduction is a common clinical abnormality detected on the electrocardiogram (ECG). It has long been recognized that when associated with heart disease, prolonged QRS duration in an ECG is an independent predictor of adverse outcome.[1] For patients with coronary artery disease plus depressed ventricular function and nonsustained ventricular tachycardia, QRS prolongation resulting from left bundle branch block (LBBB) or intraventricular conduction disturbance (IVCD) has been associated with a 50% increase in the risk of both arrhythmic and total mortality.[2] In hypertensive patients with left ventricular hypertrophy, prolonged QRS duration predicted all-cause and cardiovascular mortality and identified patients at higher risk for sudden cardiac death.[3] Furthermore, nonspecific intraventricular conduction disturbance (NICD) in an ECG is associated with increased mortality and a markedly elevated risk of sudden arrhythmic death in a general population.[4] The prevalence of NICD was reported in 3.8–5.8% of heart failure (HF) patients with reduced ejection fraction.[5] Despite its considerable prevalence, the prognostic impact of NICD in patients with acute heart failure (AHF) has not been well evaluated and compared with that of typical bundle branch blocks (BBBs), especially in East Asia. Additionally, relatively sparse data are available on the comparison of the prognosis of LBBB, right bundle branch block (RBBB), NICD and narrow QRS in the same cohort with AHF.

In this issue of the journal, Lee et al.[6] analyzed data from the Korean Acute Heart Failure registry and investigated prognostic impact of NICD and compared 1-year all-cause mortality of patient with NICD to those of patients with RBBB, LBBB, and normal QRS duration. Authors suggest that NICD was associated with an increased risk of 1-year mortality, whereas the prognostic impacts of LBBB/RBBB was limited compared to previous studies. They used data from the Korean Acute Heart Failure registry[7] with 5,157 patients. In their cohort, NICD was associated with a 75% increased risk for all-cause deaths compared with the narrow QRS group. Hazard ratios of LBBB and RBBB for the primary endpoint were 1.42 (95% confidence interval, 0.91–2.21) and 1.26 (0.85–1.88), respectively. It is very interesting data and big registry enrolling patients with AHF. Wang et al.[1] in the first time suggested the first report using data from a largescale, prospective, multicenter trial to address the predictive value of a prolonged QRS duration (QRS ≥120 ms) in patients with reduced left ventricular ejection fraction who were hospitalized for worsening HF. However, they don't compare directly among LBBB, RBBB and NICD. Tolppanen et al.[8] followed up 982 patients with HF for 3.9

years and revealed that only presence of RBBB and NICD (QRS ≥110 ms) were associated with 1.7-fold and 1.6-fold increased risks of mortality respectively. Since the definition of NICD (QRS ≥110 ms) is different from that in Lee et al.'s study[6] (QRS ≥120 ms), these results cannot be directly compared with present study. Even though they suggest that ventricular conduction disturbance especially NICD was associated with mortality in AHF patients, the pathophysiology and the mechanism of NICD has not been clearly understood yet. They mentioned heterogeneous slow ventricular activation may play a role in the development of ventricular arrhythmia in NICD patients, especially in those with reduced left ventricular systolic function. In contrast to LBBB, the left ventricular activation of NICD is relatively fast through the Purkinje network, but followed by slow activation in the scarred region.[9] This unique activation pattern of NICD might be attributed to the poor response to the cardiac resynchronization therapy.

The above "associations" or "correlates" of ECG may not be specific for patients with HF, but their value has been shown in the literature, and their continuing testing in clinical practice and research could firmly establish whether or not they possess incremental utility in the diagnosis, follow-up, and prognosis of HF. To accomplish this, one needs to implement the above ECG/HF associations in conjunction with other non-ECG parameters, like cardiac imaging-based information on volumes of heart chambers, ventricular ejection fraction, hemodynamic profiles, and humoral (e.g., brain natriuretic peptide) parameters, purported to have independent diagnostic and prognostic utility for patients with HF. The combination of the ECG/HF associations discussed above can materially facilitate the management and prediction of mortality of patients with HF. Of course, such an approach requires more than a single ECG tracing. Incorporation of these ideas in one's "routine" is not cumbersome, necessary information derives from ubiquitously available and cheap technology, and interpretation is instant. It is hoped that the use of the ECG along these lines in the care of patients with HF will lead to further progress.

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