Heart failure is a serious condition because of the structural and/or functional cardiac disorders, in which not only heart failure with preserved ejection fraction (HFpEF) but also heart failure with reduced ejection fraction (HFrEF) are substantially involved. Because of the poor prognosis of patients, especially those with HFrEF, various therapeutic options have been developed during the past 3 decades, such as β-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), implanted devices such as cardioverter-defibrillators (CRT-D) or implanted cardioverter-defibrillators (ICD), and left ventricular assist devices. To monitor the severity of heart failure, we usually examine blood levels of brain natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP); however, a more sensitive monitoring system is sometimes required in the clinical setting.

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In this issue of the Journal, Hanatani et al demonstrate that serum levels of thrombospondin-2 (TSP-2), which is a glycoprotein expressed in the extracellular matrix, developing blood vessels, and basal epidermal keratinocyte layer, correlated with markers of disease severity such as BNP, and that a high TSP-2 level was not only an independent predictor of adverse cardiovascular outcome, but also a stronger indicator of increased cardiovascular risk than the Framingham risk score and plasma BNP level in patients with HFrEF. The expression of TSP-2 is normally low in the myocardium; however, as reported by Hanatani et al, it increases in response to pathological stress stimuli, such as in HFrEF. Taken together, the combination of high TSP-2 and BNP levels identified patients with a significantly higher probability of adverse cardiovascular events.

Circulating TSP-2 levels were comparably elevated in both ischemic and non-ischemic HFrEF patients, whereas the pulmonary arterial wedge pressure was significantly higher in the high TSP-2 group than in the low TSP-2 group, suggesting that high TSP-2 levels can reflect the severe condition of heart failure, independent of the etiology. However, it remains unknown if TSP-2 levels reflect the severity of left ventricular diastolic dysfunction, because there were no significant differences in E/A ratio or deceleration time (DcT) between the 2 groups. Future studies are required to clarify the relationship between circulating TSP-2 levels and left ventricular diastolic dysfunction using other parameters that reflect left ventricular diastolic function. Further, in the current study, serial measurement of TSP-2 levels was not performed in all patients who participated in the study. Thus, it is also unknown if high TSP-2 levels remain high or go down after the pulmonary arterial wedge pressure is improved by the heart failure therapy. This issue also should be clarified in the future studies.

The new approach to patients with HFrEF is indicated in the Figure, and the clinical usefulness of this concept should be also clarified in the near future.

References