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Poor clinical outcome in patients with increased high-sensitive cardiac troponin T in dilated cardiomyopathy: in the context of left ventricular wall stress

Y. Nakashima, Y. Sugano, M. Koga, A. Shibata, N. Ueda, T. Nagai, H. Kanazaki, S. Yasuda, T. Anzai. National Cerebral and Cardiovascular Center, Department of Cardiovascular Medicine, Osaka, Japan

Background: Cardiac troponin T (cTnT) is one of the biomarkers reflecting myocardial injury widely used for the diagnosis of acute coronary syndrome. Recent reports suggested that, in non-ischemic cardiomyopathy as well, cTnT elevation occurs in patients with heart failure, serving as a potent predictor of adverse outcome. However, the background mechanisms accounting for cTnT elevation in this population has not been clarified.

Methods: We included consecutive patients who diagnosed with DCM after in-patient examination including echocardiography and cardiac catheterization between 2011 and 2016 (36 female, mean 53±14 years). Diagnosis of DCM was based on left ventricular ejection fraction (LVEF) <45%, no significant coronary disease, and exclusion of secondary cardiomyopathy such as sarcoidosis and amyloidosis with endomyocardial biopsy. We also excluded patients with renal dysfunction (serum creatinine >3.0mg/dL) from this analysis. High-sensitive cTnT (hs-cTnT) assay was performed in 114 patients who were consequently included in this study. Demographic, laboratory, echocardiographic data, as well as hemodynamic data and survival data, were collected from the patients’ medical records. We calculated LV end-diastolic wall stress (LV EDWS) by using echocardiographic and hemodynamic data based on the following equation: EDWS=(0.334*LV end-diastolic pressure*LV end-diastolic dimension)/(posterior wall thickness)*(1+(posterior wall thickness)/(LV end-diastolic dimension)). We studied the association between hs-cTnT and LV stress, echocardiographic and hemodynamic data including LV EDWS.

Results: Serum hs-cTnT was ranged 0.003–0.124 ng/mL (mean 0.017±0.019 ng/mL). The values of log hs-cTnT were positively correlated with LVEF (r=0.41, p<0.0001), mean PAP (r=0.48, p<0.0001), age (r=0.31, p=0.0038). Based on receiver operating characteristic analysis, patients were divided into two groups; high TnT group (hs-cTnT ≥0.017 ng/mL) and low TnT group (hs-cTnT <0.017 ng/mL). High TnT group showed higher LV EDWS (15.7± 10.5 mmHg, p=0.002), mean PAP (21.9±8.2 vs. 15.7±6.1 mmHg, p=0.0001) and higher LV EDWS (36.5±27.2 vs. 23.7±16.7 kdynes/cm², p=0.02) levels compared to low TnT group, despite comparable cardiac index (2.9±0.8 vs. 3.0±0.6 L/min/m², p=0.27). In survival analysis, high TnT group had worse composite outcome determined by death, LVAS implantation, appropriate ICD discharge, ischemic stroke, and heart failure hospitalization compared to low hs-cTnT group (log-rank p=0.003, Figure).

Conclusions: Patients with high cTnT level had poor outcome in DCM. Elevation of cTnT concentration was strongly associated with worse hemodynamic status including increased LV EDW. Our results suggest that ongoing myocardial injury would be, at least in part, caused by elevated LV wall stress, accounting for poor clinical outcome in patients with increased hs-cTnT level in DCM.