Searching for the key to improve infarcted cardiac wall motion and prevent ventricular remodeling after ST-segment elevation myocardial infarction: Beyond symptom-onset-to-balloon time

Chest pain is the most cardinal symptom for early medical contact in patients with acute myocardial infarction (AMI). In our group, Cho et al. (1) reported that painless ST-segment elevation myocardial infarction (STEMI) was associated with more adverse outcomes that painful STEMI. This was mainly because late detection of ischemia may have significantly contributed to the total ischemic burden. Generally symptom-onset-to-balloon time is regarded as the total ischemic time. Once the ischemic time increases, wall motion changes develop commonly in patients with acute coronary syndrome (ACS), particularly in AMI. In STEMI, ventricular wall motion inevitably deteriorates and sometimes normalizes after successful percutaneous coronary intervention (PCI). Moreover, cardiac remodeling also develops in patients with delayed reperfusion (2). Yoon et al. (3) reported that total mortality was significantly increased in patients with AMI with geometrically progressive left atrial (LA) and left ventricular (LV) dilatation. Authors of the article entitled "Wall motion changes in myocardial infarction in relation to the time elapsed from symptoms until revascularization" published in this issue of Anatolian Journal of Cardiology attempted to investigate important clinical issue concerning the relationship between wall motion changes in myocardial infarction and the time elapsed from the onset of symptoms until revascularization (4). As mentioned in the present manuscript, the fact that the spread of the infarcted zone in STEMI (5) and wall motion abnormality almost complete in the first hour, make it easier to understand why index left ventricular ejection fraction (LVEF) and LV dimensions are not so different (6). This is consistent with the result of Cho et al. (1) in terms of same LVEF (50.2±13.0 vs. 50.6±11.6, p=0.466) between painless and painful STEMI despite of different supposed ischemic time.

LV remodeling after STEMI is often precipitated in other conditions. In a study of 964 STEMI patients (7), adverse LV remodeling group showed a trend toward longer symptom-onset-to-balloon time than non-LV remodeling group (182 vs. 165 min, p=0.06), which was consistent with the present study (ischemic time ≥3 vs. <3 hours). In multivariate analysis, however, discharge heart rate turned out to be an independent predictor of future LV remodeling, not of the symptom-onset-to-balloon time. In addition, age could be a major concern regarding LV remodeling in STEMI. There is a lack of clinical trial data exclusively in elderly patients for specific therapy of adverse remodeling post-STEMI and heart failure (HF). Also HF therapy in the elderly is more challenging because of age-specific biological changes and associated comorbidities and polypharmacy (8). Finally, a recent report mentioned that anti-inflammatory therapy could be a novel treatment option targeting reduction of ventricular remodeling in such an era of modern reperfusion strategies with a goal of door-to-balloon time of <90 min and neuro-hormonal blockade therapies (9). The authors studied rather small number of patients as they mentioned in the study limitation section and this study needs to be continued with a wide range of cardiac functions, dimensions and recent generation coronary stents to make better clinical conclusions.

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