B-TYPE NATRIURETIC PEPTIDE LEVELS – A NEW BIOMARKER IN THE ACUTE CORONARY SYNDROME

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Currently, B-type natriuretic peptide (BNP) and N-terminal B-type natriuretic peptide (NT-pro BNP) are recommended as diagnostic and risk stratification tools in relation to heart failure (1). The main stimulus for their secretion is myocardial wall stress in response to myocyte stretch. It is assumed that the elevated BNP levels indicate the degree of the ischemia - induced left ventricular dysfunction in relation to the pathological left ventricle strain and overload in patients with ACSs (2). However, the relationship between ischemia and LV strain is not ruled out as related to myocardial necrosis as measured by the elevated levels of troponins. It has been suggested that the stunned myocardium, ischemic induced diastolic dysfunction, reversible border zone injury, apoptosis, acute papillary muscle dysfunction and arrhythmia may precipitate the development of acute heart failure in both STEMI and non-STEMI patients (3,4). These pathological changes related to acute ischemia, even without substantial loss of myocardial tissue pointed out a significant threat to coronary patients who developed acute heart failure. BNP has also emerged as a prognostic tool early, within three days, after acute coronary syndromes (ACS) (5,6). Patients with higher plasma BNP concentrations measured within the first week after a first myocardial infarction experience adverse left ventricular remodeling, as assessed by echocardiography (7). Donald S. C. et al, provide information that bedside BNP levels predict CV events at 10 months, independently of echocardiographic abnormalities. The study suggests that BNP enhances risk stratification post ACS (8). BNP was also a good predictor of one year cardiac mortality after myocardial infarction. Bazzino et al., demonstrated the prognostic value of elevated levels of NT-pro-BNP in ACSs, in addition to conventional risk classification and markers of myocardial necrosis. This is in accordance with recently published results confirming the poor prognosis of patients with ACS and signs of heart failure including patients without elevated markers of necrosis (9).

The novel result of the study presented by Kashlov et al. relates the observed significantly increased levels of RIPK3 (Receptor-interacting serine/threonine-protein kinase 3 – a novel regulator of programmed cell death in healthy people and generation of reactive oxygen species), 24 hours after the onset of symptoms and its strong correlation with NT pro-BNP (10). Although troponins do not correspond with the relationship between ischemia and LV strain it seems that the levels of RIPK3 correlate with degree of myocardial necrosis in STEMI patients with heart failure. Longitudinal studies on that relationships in selected population of patient with ACS and heart failure are preferable and needed.

CLINICAL IMPLICATION:

The challenge addressed in the current report is to identify the patients at highest risk and to define the appropriate treatment strategy in patients with ACS who develop acute heart failure concerning the improvement in survival (11,12). The reported findings are valuable in terms of them being in accordance with recent reports suggesting that natriuretic peptide has a potential to be a new prognostic biomarker in patients with STEMI and its measurement might be integrated into the routine evaluation of patients with ACS (13,14).

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REFERENCES