The influence of early post-STEMI inflammation on diastolic function: diagnostic and therapeutic implications

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Introduction: We know that diastolic dysfunction (DD) after ST elevation myocardial infarction (STEMI) is associated with patients prognosis. Systolic dysfunction is easy to diagnose but the identification of DD remains difficult. After STEMI, inflammation can lead to DD and vice-versa. Less information exists on the relationship between DD - inflammation in the specific post-infarction setting, while the potential prognostic and therapeutic impact is important. We focused on the relationship between the level of main inflammatory biomarkers in the first 48 hours post-STEMI and the presence of DD at one month.

Purpose: Our aim was to look for a post-STEMI early inflammatory phenotype that could be able to identify a group of patients at high risk of developing DD at one month.

Methods: The study cohort was constituted by patients admitted to our institution with a diagnosis of STEMI from 2016 to 2019. Serum from patients were collected at 4 time-points: Admission, 4 hours after revascularization (H4), H24, and H48. Inflammatory biomarkers serum levels were assessed using ELISA assays (cytokines, chemokines and endothelial activation biomarkers). Diastolic function and infarct size were assessed at one month by transthoracic echocardiogram and cardiac MRI, respectively. Diastolic function was evaluated using the latest EACVI guidelines. Patients were followed for 12 months, all individual data were collected in a prospective database.

Results: We included 236 patients. 163 patients had normal diastolic function and 73 patients had DD. The probability of having a MACE during the first 12 months after STEMI was higher in the DD group than in the normal diastolic function group (HR = 3.0 CI [1.1-7.9], p=0.01), as well as the level of BNP (197.0 ng/L versus 68 ng/L, p<0.001). There was no significant difference in Left Ventricular Ejection Fraction (LVEF) (58 versus 54%, p=0.0523), or in infarct size (13 versus 17% of left ventricular mass, p = 0.06) between the two groups. In the first 48 hours post-STEMI, in the DD group we found significantly higher seric levels for: CRP AUC (660.2 mg.h.L⁻¹ vs 361.4 mg.h.L⁻¹ p=0.02), IL-6 AUC (206.0 pg.h.mL⁻¹ vs 173.1 pg.h.mL⁻¹ p=0.03), sTNFR1 AUC (27.6 ng.h.mL⁻¹ versus 24.1 ng.h.mL⁻¹, p=0.03), sTNFR2 AUC (44.7 ng.h.mL⁻¹ vs 43.5 ng.h.mL⁻¹, p=0.03), sST2 AUC (30.4 pg.h.mL⁻¹ vs 27.3 pg.h.mL⁻¹, p=0.046), GDF15 AUC (10.8 ng.h.mL⁻¹ vs 8.1 ng.h.mL⁻¹, p=0.050) and IL-10 AUC (174.6 pg.h.mL⁻¹ vs 129.8 pg.h.mL⁻¹, p=0.02). No significant difference was found for Leucocytes, MCP1, MET, HSP 70, VCAM, RANTES, HGF 24-48h between the two groups.

Conclusions: Early inflammation is critical post-STEMI and could play a key role in DD development or progression. The proposed inflammatory biomarker profile has shown a significant association with DD at 1 month, potentially identifying a subgroup of patients at high risk (of MACE and death) and in which tailored anti-inflammatory therapy might be efficient.