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LOCAL INTERLEUKIN-6 RESPONSE IS ASSOCIATED WITH THE RISK OF NO-FLOW IN PATIENTS WITH ST ELEVATION MYOCARDIAL INFARCTION UNDERGOING PERCUTANEOUS CORONARY INTERVENTION

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Aim Although primary percutaneous coronary intervention (PCI) as early reperfusion therapy is one of the most important aspect of the treatment of STEMI, no-reflow after a primary PCI associated with a high incidence of left ventricular (LV) failure and a poor prognosis is still the main limitation of PCI. Various reports have demonstrated that PCI trigger inflammation. We aimed to investigate the association of local interleukin-6 levels and the risk of no flow in STEMI patients treated with primary PCI.

Methods Fifty six (44 male/12 female, mean age: 59±9.9 years) eligible for primary PCI were included in this analysis. Rapid exchange clot extraction catheter (Innovative technologies, Italy) was used in all STEMI patients immediately after guide-wire crossing only if a total occlusion (thrombolysis in myocardial infarction (TIMI) flow 0) existed, then patients successfully received stent implantation (1.3±0.7 per patient). Angiographic no-reflow was defined as coronary TIMI flow grade ≤2 or TIMI flow 3 with a final myocardial blush grade ≤2. Samples of each STEMI patient to determine local inflammation levels were acquired during thrombus aspiration from a total occlusion coronary using clot extraction catheter. Blood samples were also obtained before coronary angiography (pre-CA) and after PCI (post-PCI) from femora artery sheath to clarify

the extent of inflammatory change induced by PCI. Plasma concentrations of inflammatory cytokine interleukin-6 were determined by immunoassay. Comparisons between groups were done by t-test or Mann–Whitney U test (as indicated) for continuous variables and by Fisher's exact test for discrete variables. Correlation analyses were done by Pearson test or Spearman test, as indicated. Multivariable logistic regression analysis was applied to identify whether IL-6 was independently associated with coronary no-reflow. At this scope, in the model we included variables having a significant or borderline association with no-reflow at univariate analysis; furthermore, a stepwise selection method (sle=0.3, sls=0.05) was used to estimate the final predictors of no flow.

Results Twenty one (37.5%) of the patients developed no-reflow or low-reflow phenomenon. A univariate logistic analysis including all the variables evaluated at baseline: sex, age, body mass index, hypertension, family history of coronary disease, diabetes mellitus, smoking, statin therapy, white blood cells count, fasting blood glucose, cholesterol, triglycerides, hs-CRP, systemic interleukin 6, time to balloon, lesion length, reference luminal diameter, thrombus score, number of involved coronary vessels and local interleukin 6, diabetes mellitus (OR 0.321, 95% CI 0.104 to 0.988, p=0.044), hs-CRP (OR 0.959, 95% CI 0.918 to 1.001, p=0.048), systemic IL-6 (OR 0.611, 95% CI 0.381 to 0.981, p=0.034), time to balloon (OR 0.706, 95% CI 0.479 to 1.040, p=0.046), lesion length (OR 0.950, 95% CI 0.911 to 0.991, p=0.014), thrombus score (OR 0.250, 95% CI 0.062 to 1.011, p=0.036) and local IL-6 (OR 0.587, 95% CI 0.438 to 0.785, p<0.001) were independent predictor in this model. Multiple logistic regression analysis identified that advanced time to balloon (OR 0.448, 95% CI 0.204 to 0.984, p=0.045), thrombus score (OR 0.056, 95% CI 0.004 to 0.691, p=0.025) and local IL-6 (OR 0.575, 95% CI 0.374 to 0.884, p=0.012) as independent predictors of no-reflow phenomenon. Local IL-6 was the only independent predictor (OR 1.425, 95% CI 1.052 to 1.931, p=0.0223) in this model, with a stepwise selection method (sle=0.3, sls=0.05) was used to estimate the final predictors of cardiac events, after adjustment of all entered baseline variables.

Conclusions The occurrence of no-reflow phenomenon in STEMI patients after primary PCI was relation to local inflammation response. These findings suggest that local IL-6 response decreased might be beneficial in the management of no-reflow.