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INCREASED LEVELS OF CIRCULATING ENDOTHELIAL PROGENITOR CELLS IN INTRACORONARY BLOOD OF ST-ELEVATION MYOCARDIAL INFARCTION PATIENTS CORRELATE WITH MICROVASCULAR DAMAGE.

i2 Poster Contributions
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Background: Endothelial progenitor cells (EPC) are bone marrow derived elements, whereas microparticles (MP) are fragments derived from activated platelets (CD31+/ CD42+ PMP) endothelial cells (CD31+/CD42- EMP) or from apoptotic cells (CD31+/AnnexinV+ AMP). Both are known to be increased in the context of acute coronary syndromes. We assessed whether a difference in levels of EPC can be detected between intracoronary and peripheral blood soon after the onset of ST elevation myocardial infarction (STEMI), and the correlation of EPC with MP and angiographic indexes of revascularisation.

Methods: Twenty nine STEMI patients undergoing successful primary percutaneous coronary intervention (pPCI) were included. Aortic blood samples from the guiding catheter and intracoronary, translesional blood aspirate from thrombectomy device were sequentially drawn at the beginning of pPCI to measure EPC (CD34+/ KDR+/CD45-), AMP, EMP and PMP by flow cytometry. TIMI flow grade, corrected TIMI frame count (cTFC), and Myocardial Blush Grade (MBG) were measured after PCI to assess revascularisation efficacy.

Results: EPC levels (expressed as %, number of cells per total number of cytometric events) were higher in intracoronary 0.06% (0.02-0.16 IQR, $p<0.001$) than in peripheral blood 0.02% (0.01-0.04). Only intracoronary EPC were related to optimal revascularisation index, resulting higher in MBG0/1 class 0.07% (0.04-0.17, overall ANOVA $p<0.001$) than in MBG2 0.06% (0.03-0.14) and MBG3 0.05% (0.02-0.14, $p<0.001$ for linear trend). No correlation was found with other revascularisation indexes. At the same time a negative correlation was detected between both intracoronary and peripheral AMP and peripheral EPC levels (rho=-0.7, $p<0.001$ in both cases).

Conclusions: Our data show that a rapid intracoronary EPC recruitment takes place in the earlier phases after STEMI onset. This recruitment is inversely related to post-PCI MBG suggesting that EPC are recruited in the culprit artery in attempt to overcome ongoing microvascular dysfunction.

The negative correlation between AMP and EPC levels suggests a possible negative effect of apoptosis on EPC mobilization.