BMPRII signaling of fibrocytes, a mesenchymal progenitor cell population, is increased in STEMI and dyslipidemia

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Introduction: Inflammation is a hallmark feature of ST-elevation myocardial infarction (STEMI). Fibrocytes, a Collagen-I+CD34+CD45+ mesenchymal progenitor cell population accumulate in cardiac tissue of a murine ischemia/reperfusion model. In ACS patients, decreased levels of circulating fibrocytes were found, compared to healthy controls. Bone morphogenic protein receptor II (BMPRII) is involved in the vascular remodeling of lung and heart. Therefore, we studied BMPRII expression in fibrocytes at the culprit lesion site (CLS).

Methods: We sampled blood from the CLS and a femoral site in the course of primary percutaneous coronary intervention (pPCI) from STEMI patients (n=50, male=78%, mean age=61±13y). Another sample was acquired 72h after pPCI (n=21). A cohort of healthy controls (n=20, male=48%, mean age=51±8y) served as controls. Flow cytometry was employed to characterize fibrocytes.

Results: Fibrocytes were increased at the CLS compared to femoral blood (722 [276-1298] vs. 324 [180-589], p=0.0001). 72h after STEMI, peripheral fibrocytes were decreased (246 [151-468] vs. 153 [102-252], p=0.006). Peripheral fibrocyte counts during pPCI were similar to those of controls.

No differences were found in BMPRII expression between coronary and femoral blood of STEMI patients; however, BMPRII expression was higher in patients than controls (MFI 22106 [13142-34125] vs. 13099 [8944-20231], p=0.014). In patients suffering from dyslipidemia, BMPRII on fibrocytes was substantially increased (MFI 26056 [13195-54807] vs. 19913 [13635-22965], p=0.009). 72h after pPCI, BMPRII was significantly upregulated on fibrocytes (MFI 22294 [17973-34125] vs. 31149 [27722-45724], p=0.044).

Conclusions: The more than two-fold increase of fibrocytes at the CLS and subsequent decrease 72h after pPCI in peripheral blood supports the concept of an active process. BMPRII expression is increased in STEMI patients, particularly in patients with dyslipidemia, suggesting lipid-induced inflammation, and the activation of fibrotic vascular remodeling.