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Biochemical markers of inflammatory response and their relation to myocardial injury

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Introduction

Neutrophils have been suggested to be involved in irreversible reperfusion myocardial injury. Biochemical markers of neutrophil infiltration and also other markers of inflammatory response are therefore of interest following reperfusion. One might hypothesize that inflammatory activity contributes to the myocardium at risk (MaR) and myocardial infarct (MI) size or that the inflammatory activation is a response to necrosis.

Purpose

To compare peak and cumulative markers of inflammatory response to MaR, MI size, salvage of MaR, and to improvement of regional function.

Methods

Sixteen patients with a first-time acute myocardial infarction due to occlusion treated by primary PCI were evaluated. Serial blood samples were acquired for analyses of markers for neutrophil infiltration (myeloperoxidase MPO, neutrophil gelatinase NGAL, metalloproteinase-9 MMP-9), of inflammation (interleukin-6 IL-6, interleukin-8 IL-8, tumor necrosis factor-α TNF-α), and of an acute-phase reactant (high-sensitive C-reactive-protein hsCRP). Samples were obtained before PCI and after reperfusion at 1.5 h, 3 h, and 24 h. In twelve of the patients, 99 mTc-tetrofosmin was injected prior to opening of the occlusion and SPECT was performed within three hours for MaR. Late gadolinum-enhanced cardiac magnetic resonance (CMR) was performed the same day for regional function, and at one week also for MI sizing. Regional function was calculated from cine MR images as wall thickness in systole versus diastole in 72 segments. Segments were defined as infarcted, adjacent or remote to infarction and analysed for improvement in function. Salvage was calculated as (MaR-MI)/MaR. Indexes of neutrophil infiltration and inflammation were calculated by combining MPO, NGAL, MMP-9, and IL-6, IL-8, TNF, respectively.

Results

Correlations between peak or cumulative markers of inflammatory response and MaR were not strong, except for peak ($r2 = 0.64$, $p < 0.01$) and cumulative NGAL ($r2 = 0.75$, $p < 0.001$). Correlations between markers of inflammatory response and MI size were equally low, $r2 < 0.50$ for all markers. Salvage of MaR showed a slightly higher correlation only with peak MPO ($r2 = 0.57$, $p < 0.01$). Regarding calculated indexes a higher correlation was showed for neutrophil infiltration by peak ($r2 = 0.56$, $p < 0.01$) and cumulative values ($r2 = 0.61$, $p < 0.01$) and MaR. No statistically significant correlation was found between markers of inflammatory response and improvement of regional function.

Conclusion

No significant relationship was found between markers of inflammatory response and improvement of regional function, or MI size. It is thereby unlikely that the inflammatory activity contributes significantly to MI size or that the magnitude of the infarct determines the inflammatory response. A correlation between markers of inflammatory response and MaR warrants further study.