Background: Elevated plasma levels of C-Reactive Protein (CRP) and Interleukin-6 (IL-6; the cytokine mainly responsible for CRP synthesis by the liver) have been demonstrated in patients with acute myocardial infarction (AMI). Plasma CRP has been correlated with infarct size, whilst plasma IL-6 has been associated with myocardial stunning.

Methods: To assess whether CRP and IL-6 are related to infarct size, myocardial stunning or both, we measured the cumulative release of CRP and IL-6 during the first 48 hours in 44 patients with first AMI. Infarct size was assessed enzymatically (72-hours cumulative LDH release) and by 2-D-echocardiography using wall motion score (WMS) at admission and at 3 months. Myocardial stunning was defined as change in WMS and number of segments recovering to baseline in 3-months follow-up. Patients were divided in 2 groups of 22 patients by median release values of CRP (66 mg/L) and IL-6 (208 mg/L).

Results: In 48 hours (n = 44):

- CRP (mg/L): Release in 48 hours correlated with cumulative LDH (r = 0.38; p = 0.01), CRP release (r = 0.47; p = 0.001) and 3-months WMS (r = 0.44; p = 0.007) and 3-months WMS (r = 0.49; p = 0.001).
- IL-6 (mg/L): Release in 48 hours correlated with cumulative LDH (r = 0.40; p = 0.007) and by 2-D-echocardiography using wall motion score.
- Mean WMS (admission): 7.1 ± 3.0
- Mean WMS (3 months): 3.3 ± 2.0
- Change WMS (adm-3 month): 3.9 ± 2.0
- Recovery (mean # segments): 3.0 ± 2.0

Conclusion: IL-6 and CRP release in first AMI are positively correlated with infarct size. Myocardial stunning is more apparent in both the low IL-6 and CRP release groups.

Dysregulated levels of cytokines in circulating mononuclear leukocytes from patients with acute myocardial infarction

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Vascular endothelial growth factor mRNA synthesis by peripheral blood mononuclear cells in patients with acute myocardial infarction

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Vascular endothelial growth factor (VEGF) is an angiogenic glycoprotein that is upregulated in cardiac myocyte subjected to ischemia. Serum levels of VEGF are reportedly elevated in the subacute phase of acute myocardial infarction (AMI). However, there is no direct evidence that VEGF mRNA is expressed in patients with AMI. To investigate whether VEGF mRNA is expressed in AMI, we measured levels of VEGF mRNA in peripheral blood mononuclear cells (PBMC) obtained from patients with AMI using competitive polymerase chain reaction (PCR).

Methods: Fifteen patients with AMI and 15 healthy controls were enrolled. In this study, PBMC were isolated from all patients on day 14 after onset, and from all controls. Total RNA was extracted from PBMC and reverse transcribed into cDNA. We performed competitive PCR by co-amplifying serial dilutions of GAPDH mutant templates containing a unique Eco RI site. Next, to measure VEGF cDNA semiquantitatively in the samples containing identical amount of GAPDH, we performed competitive PCR similarly by co-amplifying serial dilutions of VEGF mutant templates containing a unique Eco RV site.

Results: Higher levels of VEGF mRNA in the PBMC were observed in the AMI patients (3.0 ± 1.5 fg/pg GAPDH) than in healthy controls (1.8 ± 0.4 fg/pg GAPDH) (p < 0.05). Serum levels of VEGF were significantly correlated with the amount of VEGF mRNA in the PBMC (p < 0.05).

Conclusions: Levels of VEGF mRNA in PBMC were elevated in the subacute phase of AMI and significantly correlated with serum VEGF concentration. Results suggest that VEGF in PBMC is overexpressed in response to some signals during the subacute phase of AMI for the purpose of angiogenesis and healing.