Abstract

An elevated preprocedural plasma or serum C-reactive protein (CRP) level in patients undergoing percutanous coronary intervention (PCI) is associated with short-, intermediate- and long-term outcome. Furthermore, the procedure itself has been shown to provoke an inflammatory reaction as shown by increased plasma CRP levels after PCI. The research programme resulting in this thesis focus on the role of CRP and inflammation in patients undergoing PCI.

In study I 400 consecutive patients with serum levels of troponin T ≤ 0.03 μg/l presenting with stable or unstable angina pectoris were investigated. Twenty-one percent of the patients experienced a myocardial infarction during PCI. The median value of CRP before the procedure was 1.83 (0.12-99.7) mg/l. No difference was seen in CRP levels before PCI between patients without or with myocardial infarction during PCI. Multivariate analysis identified stent implantation, procedure time and complications during the procedure as independent predictors of myocardial infarction during PCI.

In study II 121 patients with stable angina pectoris were enrolled. 100 patients were treated with a thrombin-based (Duett sealing) femoral artery closure device and 121 patients were treated with the FemoStop device. Irrespective of femoral artery closure device serum CRP and serum amyloid A (SAA) levels increased significantly after PCI. The increase was more pronounced in the Duett sealing device group compared with the FemoStop device group.

In study III 100 patients with stable angina pectoris scheduled for elective PCI were prospectively enrolled. Antibodies against different pathogens were examined. Plasma CRP and IL-6 levels were measured before and 6, 24, 48, 72 hours after PCI. Neither infection with single or multiple pathogens nor a minor troponin T elevation after PCI was associated with plasma CRP or interleukin-6 (IL-6) area under the curves (AUCs). Patients treated with stent implantation had higher plasma CRP and IL-6 AUCs compared with patients treated with balloon angioplasty alone

Study 1V 891 consecutive patients presenting with stable or unstable angina pectoris undergoing a variety of PCIs were investigated. Serum concentrations of CRP and troponin T were determined before and the day after PCI. The mean follow-up time after PCI was 2.6 years. In multivariate analysis, patients in the highest tertile of CRP, induced by PCI, had an increased risk (risk ratio (RR) 2.48 [95% confidence interval (CI) 1.42-4.33]) for death or non-fatal myocardial infarction. Furthermore, patients in the second (RR 1.75 [95% CI 1.14-2.75]) and third (RR 2.15 [95% CI 1.37-3.36]) tertiles of the CRP response had an increased risk for coronary revascularization. Patients with periprocedural myocardial infarction with a postprocedural troponin T > 0.14 μg/l had an increased risk for death or non-fatal myocardial infarction (RR 2.65 [95% CI 1.02-6.83]).

Conclusion: Periprocedural factors, whereas not the preprocedural CRP were associated with the risk of myocardial infarction during PCI. However, an elevated serum CRP concentration in response to PCI is a strong independent predictor of death or non-fatal myocardial infarction and coronary revascularization independent on myocardial injury during the procedure. Factors related to the procedure are influenced the CRP response to PCI. The results emphasize the role of CRP in coronary artery disease (CAD), and the need to develop treatments that block the increase in CRP in CAD.

Keywords: C-reactive protein, Duett sealing device, FemoStop, interleukin-6, pathogen burden, percutaneous coronary intervention, prognosis, myocardial infarction, serum amyloid A, troponin T.