RAHEL BEFEKADU WODAJO was born in 1968 in Ethiopia and came to Sweden at the age of 17. She graduated in 2000 at Malmö University as biomedical scientist specializing in pathology. In 2005 she received her masters degree in Biomedical laboratory science at Örebro University. She awarded her Master of Science degree in Medicine 2013 at Örebro University. In 2014, Rahel started her doctoral studies at the Cardiovascular Research Centre, School of medical Sciences, Örebro University. Rahel have been working as a biomedical scientist in the department of clinical chemistry at the Lindesberg Hospital as well as in the department of Pathology and in IVF and fertility unit in Örebro University Hospital. Rahel has throughout this thesis worked as a biomedical scientist in the department of transfusion medicine, section for serology and stem cell.

The rupture of an atherosclerotic plaque in a coronary vessel, and the following formation of a thrombus may lead to an ST-elevation myocardial infarction (STEMI). STEMI causes ischemia in the myocardium, and the following treatment with Percutaneous coronary intervention (PCI) to open the vessel will lead to reperfusion, followed by remodeling of the myocardium during the healing process. These events include interplay between different types of cells and the inflammation, coagulation and complement systems, but the kinetics and predictive capacity of many components are still not fully explored.

This thesis studies different biomarkers in a cohort of patients suffering from STEMI who underwent Percutaneous coronary intervention (PCI) in Örebro in 2011-2012. Blood samples were collected at three time points, at the arrival at the hospital, 1-3 days after PCI and for a smaller group of patients also 3 months after PCI. The study is a sub-study of the TASTE study, so half of the patients were also randomized to thrombus aspiration in conjunction with their PCI. For all patients, it was also recorded whether the culprit coronary vessel was totally occluded or partially patent.

The markers studied are the lysosome protein Cathepsin S (Cat-S), the platelet granule protein thrombospondin 1 (TSP-1), the pentraxins C-reactive protein (CRP) and pentraxin 3 (PTX3), the endopeptidase neprilysin, the soluble forms of TNF-receptor 1 and 2 (sTNFR1 and sTNFR2), markers showing activation of the lectin pathway for complement activation (MASP-1/AT, MASP-1/C1-INH, MASP-2/C1-INH, MASP-2/AT) and common activation markers for complement activation (C3a and sC5b-9).

In this thesis, it is shown that the kinetics for the different markers varies, but that thrombus aspiration or patency of the culprit vessel have little impact on the levels. Survival analysis showed that individuals with plasma levels above the median value for PTX3, sTNFR1 and sTNFR2 at admission and/or at 1-3 days had a significantly increased mortality compared to those with levels below the median value, which indicates that these markers could be interesting for further studies in a material where also analysis of possible interfering factors can be implemented.

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