Chemokines are elevated in ACS and are differentially modulated by heparin treatment.

Platelet CCL5 links acute coronary syndrome and vascular inflammation

Dirk Sibbing1,2; Christian Schulz1,2,3

1Medizinische Klinik und Poliklinik I, Klinikum der Universität München, Ludwig-Maximilians-Universität München, Munich, Germany; 2DZHK (German Center for Cardiovascular Research), partner site Munich Heart Alliance, Munich, Germany; 3Walter-Brendel-Centre of Experimental Medicine, Ludwig-Maximilians-Universität München, Munich, Germany

Correspondence to:
PD Dr. med. D. Sibbing, FESC
Medizinische Klinik und Poliklinik I
Ludwig-Maximilians-Universität München
Marchioninistr. 15, 81377 München, Germany
Tel.: +49 89 4400 73028
E-mail: dirk@sibbing.net or dirk.sibbing@med.uni-muenchen.de

Received: November 3, 2014
Accepted: November 3, 2014
Epub ahead of print: November 13, 2014
http://dx.doi.org/10.1160/TH14-11-0910
Thromb Haemost 2015; 113: 1079

Initiation and progression of atherosclerosis, a chronic inflammatory process of the arterial wall that ultimately leads to ischemic events (e.g. myocardial infarction, stroke), is mediated by a number of extracellular and cellular factors including blood platelets and leukocytes (1). Chemokines (e.g. CCL5 (RANTES), CXCL4) released from activated platelets and neutrophil-derived mediators like myeloperoxidase or azurocidin are key players in atherosclerotic lesion formation. Furthermore and with respect to atherosclerosis in the coronary arteries, it is well known that in the course of these coronary syndromes (ACS) these processes are amplified and accelerated.

In the current issue of Thrombosis and Haemostasis, Blanchet et al. (2) report on the inflammatory role and prognostic value of platelet chemokines and neutrophil-derived mediators in ACS. Key results of this observational study in 204 patients – 115 ACS patients and 89 patients without ACS and angiographic exclusion of coronary artery disease (CAD) – are that chemokines released from blood platelets and mediators released from neutrophils are elevated in ACS and are differentially modulated by heparin treatment. Interestingly, higher CCL5 plasma levels in ACS patients were found to be predictive for the progression of CAD in this cohort. Thus, platelet CCL5, which is one of the most abundant chemokines in platelets (3), may represent a mechanistic link between ACS and atheroprogression. Platelets become activated in the course of ACS, e.g. by binding to exposed atheromatous plaque, and release CCL5, which may in turn drive leukocyte activation and recruitment of monocytes to the inflamed vessel wall. The paper by Blanchet et al. thereby adds to the concept that ACS by itself may accelerate progression of atherosclerosis (4), a potential vicious circle for the patient. Present data and numerous prior investigations have contributed to a better understanding of the inflammatory processes and key mediators in human atherosclerosis. Besides established therapeutic treatment concepts including acetylsalicylic acid and statins for primary and secondary prevention in CAD patients, a number of new therapeutic strategies are likely to arise in the future (5). Such novel treatment options may have the potential for a very specific treatment of cardiovascular disease, thereby maximizing the treatment effect and minimizing side effects.

Conflicts of interest
None declared.

References