P. 52

IDENTIFICATION OF DIFFERENTIALLY EXPRESSED PROTEINS IN ACUTE CORONARY SYNDROMES

V.M. Dardé[1,6], M.G. Barderas[1,7], F. de la Cuesta[1], F. Gil[7], G. Alvarez-Llamas[1], J.J. Nacher[4], L. López-Bescos[4], J. Tuñón[3], J. Egido[2], F. Vivanco[1,5]

[1]Departamento de Inmunología, Fundación Jiménez Díaz;
[2]Laboratorio de Patología Renal y Vascular, Fundación Jiménez Díaz;
[3]Servicio de Cardiología, Fundación Jiménez Díaz, Madrid;
[4]Unidad de Cardiología, Fundación Hospital de Alcorcón, Madrid;
[5]Departamento de Bioquímica y Biología Molecular I,
Unidad de Proteómica, Universidad Complutense, Madrid;
[6]Unidad de Proteómica, Hospital Nacional de Parapléjicos (SESCAM), Toledo;
[7]Dpto. Fisiología Vascular, Hospital Nacional de Parapléjicos (SESCAM), Toledo

Acute Coronary Syndromes (ACS) are triggered by the occlusion of a coronary artery, usually due to the thrombosis of an atherosclerotic plaque. The study of the mechanisms that lead to the plaque thrombosis has been one of the hot spots in cardiovascular research during the last years. A screening for biomarkers in the blood of ACS patients using proteomic tools could provide useful diagnostic and therapeutic information. We have performed a comparative study of plasma from ACS patients combining immunoaffinity depletion with 2-dimensional difference gel electrophoresis (2D-DIGE) and identification by mass spectrometry.

Depleted plasma samples from 28 patients with ACS at day 0, 4, 90 and 180, were compared to samples from 10 stable coronary artery disease (CAD) patients and 10 healthy volunteers, matched for age and sex. DeCyder software revealed statistically significant variations of 47 protein spots in depleted plasma samples from ACS and stable CAD patients in comparison with healthy subjects. The number of differentially expressed spots tended to increase along time, conversely to that observed for circulating monocytes. The identified proteins were involved in different physiological processes, some of whom may play a role in the pathophysiology of the atherotrombotic disease.

We have also studied the effect of intensive treatment with statins in comparison with moderate treatment, assigning patients to receive either 80 mg/d atorvastatin (ATV) or standard therapy during two months after ACS. Expression levels of 15 spots were affected by both treatments, while 14 different spots were normalized by ATV with respect to standard therapy and 4 more spots were only altered by ATV.

Further validation of these findings in larger populations would prove the usefulness of these proteins as novel biomarkers of ACS. These results might help to enrich the current knowledge of molecular mechanisms involved in ACS and improve the existing diagnostic tools.