Diabetes and the Heart

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The French Society of Cardiology (SFC) and the French-speaking Society of Diabetology (ALFEDIAM) co-organize this year a scientific meeting dedicated to the heart in diabetes mellitus. Since myocardial ischaemia in patients with diabetes is frequently discussed, the aim of this 2007 scientific meeting was to give information mainly on cardiomyocyte metabolism, myocardial dysfunction, arterial calcification and novel pathophysiological mechanisms of macrovascular disease in diabetes. In this special issue of Diabetes & Metabolism are reported the main presentations at this conference.

There is now a large body of evidence supporting the existence of "diabetic cardiomyopathy". In their well documented paper, D. Feuvray et al. explain the role of increased fatty acids in disturbing cardiomyocyte metabolism leading to cardiac dysfunction. The mechanisms involved include opening of the cell membrane K<sub>ATP</sub> channel, increased lipid peroxidation and apoptosis. B. Vergès et al report data showing that Nt-ProBNP is, in patients with diabetes, a very strong marker of the poor short-term prognosis after myocardial infarction in patients with diabetes. Nt-proBNP seems to reflect the integration of different risk markers for adverse outcomes (death, heart failure) following myocardial infarction with high informative value.

Recently more attention was given to vascular calcification which is frequent in patients with diabetes. New data on the pathophysiology of vascular calcification are reported in the paper from ZA Massy et al. The presence of vascular calcification could be explained by an imbalance between osteoblast-like and osteoclast-like cell activities in the arterial wall. Many longitudinal studies have demonstrated the predictive value of arterial stiffness, beyond and above classical risk factors for cardiovascular disease. P Boutouyrie describe new techniques for assessing arterial stiffness including measurements of aortic pulse wave velocity and central pressure.

Circulating microparticles derived from apoptotic cells are early markers of vascular dysfunction and may be involved in vascular complications. In their paper, AS Leroyer et al indicate that microparticles levels are increased in patient with diabetes. They present data showing that microparticles, due to their pro-inflammatory properties, could be involved in the development of vascular complications in diabetes by promoting thrombosis, endothelial dysfunction and angiogenesis. During the past years, a lot of interest has risen on Endothelial Progenitor Cells (EPC), which are involved in re-endothelialisation of injured vessels and formation of new vessels in ischemic tissue. JB Silvestre, in his article, shows data indicating that diabetes reduces the availability and the functions of EPC, leading to diminish the EPC-induced post-ischemic vessel growth.

The role of chronic hyperglycaemia in the development and progression of macrovascular disease is discussed by SH Hadjadj et al in the light of the intervention studies performed in patients with diabetes. The available data indicate that there is probably no specific HbA<sub>1c</sub> threshold for macrovascular disease. The on-going trials, such as ADVANCE and ACCORD should give additional information to clarify this point.