EDITORIAL

Coronary recanalisation, myocardial viability, and ventricular remodelling after infarction

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It is important to identify the mechanisms that determine the progression to left ventricular remodelling after an acute myocardial infarction, in order that patients can be treated before the development of overt heart failure

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Chronic heart failure (CHF) is common and in about 70% of cases is secondary to coronary artery disease (CAD).¹ In most of these patients there is evidence of a previous acute myocardial infarction (AMI), especially when the latter is extensive, transmural, and involves the anterior wall of the left ventricle.² Development of overt CHF after AMI is often slow and is preceded by an asymptomatic phase in which changes in the shape, size and properties of the left ventricle occur (remodelling).³ Although different large randomised trials carried out during the past two decades have demonstrated a significant reduction in mortality for heart failure patients treated medically, symptomatic heart failure continues to have a one year mortality close to 45%.^{4–5}

Clinically, it is important to identify the mechanisms that determine the progression to left ventricular remodelling after an AMI and treat them before the development of overt CHF. Overall, there is a linear relation between the number of dysfunctional left ventricular segments and remodelling. Chronic systolic dysfunction after AMI is caused by permanent myocyte loss with scar formation as well as by the presence of hibernating myocardium (that is, dysfunctional but viable myocardium subtended by stenosed coronary arteries which recovers after revascularisation).⁶ Based on available studies, it is logical to assume that the beneficial effect of coronary revascularisation in CAD patients with chronic left ventricular dysfunction derives primarily from recovery of contractile function in hibernating segments which, in turn, may attenuate remodelling.

CORONARY RECANALISATION

In this issue of *Heart*, Bellenger and colleagues⁷ provide evidence that coronary recanalisation, even at a late time after AMI (between three days and six weeks, mean of 26 days), can lead to attenuation of subsequent left ventricular remodelling. The authors studied a subset of patients from the open artery trial (TOAT).⁸ Dobutamine stress cardiovascular magnetic resonance (CMR) was used to assess myocardial viability in patients with anterior myocardial infarction, left ventricular dysfunction, and isolated proximal

occlusion of the left anterior descending coronary artery (LAD), who either underwent late percutaneous coronary intervention with stent to the LAD, or medical treatment alone.⁷ The study demonstrates that in the patients undergoing revascularisation, there was a significant relation between the number of viable segments within the infarct zone and improvement in end systolic volume index and ejection fraction, whereas in the medically treated group there was no relation between the number of viable segments in the infarct zone and the subsequent changes in systolic volume or ejection fraction.

The results of this investigation add to a large number of previous, non-randomised studies that provide a compelling message on the importance of looking for hibernating myocardium in patients with CAD and chronic left ventricular dysfunction.⁹ In this setting coronary revascularisation as a specific treatment for CHF provides not only significant symptomatic relief and improvement of life quality, but also confers a better prognosis.¹⁰ In principle, hibernating myocardium should be suspected in all patients with coronary artery disease and chronic left ventricular dysfunction of any degree ranging from regional dysfunction to ischaemic cardiomyopathy. Myocardial hibernation implies the concept of tissue viability, which can be diagnosed by non-invasive imaging modalities that detect either the presence of contraction reserve or the persistence of metabolic activity within chronically dysfunctional myocardial regions.⁹

SYMPATHETIC NERVOUS SYSTEM ACTIVATION

A number of other mechanisms contribute to left ventricular remodelling among which enhanced activation of the sympathetic nervous system plays a major role. Myocardial β adrenoceptors (β -AR) are downregulated in patients with overt CHF and the degree of receptor downregulation is related to the severity of CHF.¹¹ Furthermore, these patients have higher levels of circulating catecholamines that are inversely related to prognosis.¹²

Positron emission tomography with the non-selective β -AR antagonist S-[¹¹C] CGP 12177 allows the non-invasive measurement of regional myocardial β -AR density in humans in vivo, and Merlet and colleagues¹³ have demonstrated

Abbreviations: AMI, acute myocardial infarction; β -AR, myocardial β adrenoceptors; CAD, coronary artery disease; CHF, chronic heart failure; LAD, left anterior descending coronary artery; CMR, cardiovascular magnetic resonance; TOAT, the open artery trial

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Figure 1 Myocardial β -adrenoceptor downregulation soon after acute myocardial infarction predicts left ventricular (LV) dilatation. β -AR, myocardial β -adrenoceptor density; LV improved, those patients ($n = 41$) whose left ventricular volumes were found to be decreased six months after infarction; LV dilated, those patients ($n = 20$) whose left ventricular volumes were found to be increased six months after infarction. Adapted from Spyrou and colleagues.¹⁵

downregulation of β -AR in patients with CHF caused by idiopathic dilated cardiomyopathy. Using the same technique, our group demonstrated progressive β -AR downregulation in those patients with hypertrophic cardiomyopathy who proceed to left ventricular dilatation and CHF.¹⁴ More recently, in a prospective study of patients with AMI as their first presentation of CAD, we have demonstrated that the degree of myocardial β -AR downregulation, measured in the subacute phase after AMI, is predictive of subsequent left ventricular remodelling assessed by serial measurements of left ventricular volumes up to six months after infarction (fig 1).¹⁵

In summary, non-invasive imaging techniques allow measurements of physiological parameters that are strictly

related to the development of left ventricular remodelling and CHF. Appropriate and timely use of these techniques allows proper patient stratification and optimisation of treatment strategies.

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IMAGES IN CARDIOLOGY

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Cervical extradural haematoma following thrombolysis

A previously well 53 year old man presented with a three hour history of severe chest pain. An ECG showed 4 mm ST segment elevation in the anterior leads. He received reteplase and clopidogrel (aspirin intolerance). Twelve hours later he developed severe neck pain with progressive tetraparesis with a sensory level at C6. Post-thrombolysis heparin and antiplatelet agents were stopped.

Magnetic resonance imaging of the spine (left) showed an extensive posterior extradural haemorrhage within the spinal canal from C2 and T10 with associated spinal cord compression at C5/6 and C6/7.

The patient was transferred as an emergency to the local neurosurgical centre where he underwent emergency evacuation of the haematoma. Seven days later, he had ongoing chest pain with dynamic ECG changes and underwent successful angioplasty to his left anterior descending coronary artery. He has since recovered full function in his upper limbs.

In thrombolysed patients, sudden neck pain should prompt suspicion of the rare, but recognised, complication of a cervical extradural haemorrhage. Urgent neurosurgery can significantly improve neurological prognosis.

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