



Cardiac protection in acute cardiac syndrome

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Editorial

Decades ago, coronary artery occlusion by thrombus formation at the site of a vulnerable atherosclerotic plaque was identified as the precipitating mechanism of acute coronary syndromes. Since then, great efforts have been made in order to design and test strategies for the recanalization of occluded coronary arteries as a strategy to limit infarct size and improve clinical outcomes. Today, over 95% of occluded vessels can be easily reopened in the setting of STEMI. Unfortunately, the clinical impact of these interventions falls short of expectations.

The reduction in mortality, though statistically significant, is of minor entity in absolute terms and limited to patients treated early at tertiary referral centre. From a population point of view, mortality for AMI is still dramatically high, and a large fraction of patients surviving the acute phase is bound to develop heart failure.

Several factors contribute to these disappointing results, including incomplete understanding of the time course of myocardial cell death and underestimation of the contribution of reperfusion injury to final infarct size.

In this issue of *Heart and Metabolism*, experts in ischemia-reperfusion injury, many from my own center, elucidate the mechanisms of ischemic cell death, describe the time course of cellular damage, and discuss the impact of reperfusion on cells previously exposed to ischemia.

The message that can be extrapolated from these contributions is a complex one:

1. Ischemic damage is a time-dependent phenomenon that is completed in a few hours after the occlusion of a coronary vessel. Survival of ischemic tissue may be prolonged by previous exposure to ischemia, or by other interventions, but is certainly shorter than the 12 hours currently accepted as the time limit for primary PCI.
2. Reperfusion injury adds to ischemic injury, but this has appreciable clinical consequences only if flow is re-established when a sizable amount of myocardium is still viable, that is very early, possibly within 2 hours. If flow is re-established later, when the ischemic cell death is completed, reperfusion injury will not impact on final infarct size.
3. Reperfusion injury is triggered by a burst of oxygen free radicals that peaks at 2–5 minutes after restoring flow. Therefore protective agents, to be effective, must be delivered to the target area before restoring flow. •