Patterns of Left Ventricular Dilatation With an Opened Artery After Acute Myocardial Infarction

Missing Links to Long-Term Prognosis

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Further insight into the sequence and significance of LV responses is provided by Bolognese et al. In their examination of 284 consecutive patients undergoing primary percutaneous coronary intervention (PCI) for acute myocardial infarction, Bolognese et al obtained serial echocardiographic and angiographic studies at 24 hours, 1 month, and 6 months. Late follow-up (61±14 months) was available in all but 4 patients. Despite excellent infarct-related artery patency rates at 6 months, 30% of patients showed LV dilatation with >20% increase in end-diastole volume at 6 months compared with 24 hours. Cardiac cath and combined adverse cardiac event rates were significantly higher in patients with angioplasty with stenting. Because of the many uncontrollable variables of myocyte salvage, considerable controversies remain regarding the links between the effectiveness of an opened artery after infarction on long-term ventricular function and consequent survival statistics.7

The most effective interventions to limit or prevent ventricular dilatation after infarction are those addressing the initial myocardial insult through the earliest and most sustained reperfusion therapies.6 The re-establishment of coronary blood flow to the infarcted region, even if delayed in some circumstances, is thought to attenuate ventricular remodeling. Modification of those biochemical and physiological factors that deform the compromised ventricle also influences late ventricular adaptive responses and prognosis. It is now common knowledge that instituting afterload reduction improves ventricular remodeling, attenuates infarct expansion, and provides long-term clinical improvement.1

However, of the three major factors involved in the acute infarct remodeling sequence, cardiologists can exert the most influence only at the onset through the timeliness and completeness of reperfusion with successful thrombolysis and/or angioplasty with stenting. Because of the many uncontrollable variables of myocyte salvage, considerable controversies remain regarding the links between the effectiveness of an opened artery after infarction on long-term ventricular function and consequent survival statistics.7

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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angiography at 1 month and again 6 months later. The quantitation of LV function and size with serial echocardiography is also a key observation linking these phenomena. The study findings are consistent with our current understanding of infarct physiology, myocardial functional recovery, and remodeling. Infarct size, anterior infarct location, and perfusion status of the infarct-related artery, and heart failure on admission, and a restricted pattern of LV filling have all been identified as major predictors of LV dilatation after infarction. These observations demonstrate that after acute myocardial infarction, even with patent infarct-related arteries and a relatively small initial end-systolic volume, many patients remain at risk for ventricular remodeling despite an early favorable examination. Bolognese et al emphasize that acute myocardial infarction patients should be evaluated serially and all available therapies to reduce LV remodeling should be instituted early and maintained indefinitely.

These data also leave us with some questions, some missing links remaining to be answered. While the size of the infarction is crucial to LV remodeling, is the response and preservation of the microcirculation at the time of injury also related to the timing and mechanism of late remodeling? For example, Neumann et al demonstrated that glycoprotein receptor blockade with standard therapy during acute PCI for infarction was associated with improved LV function and coronary hyperemia over a 2 week follow-up. Although angiography was serially performed, this study did not examine TIMI flow or myocardial blush scores or note the incidence of slow or no re-flow during intervention to link microvascular responses to LV recovery.

Why is the infarct size not related to the pattern of LV remodeling? Although the magnitude of creatine kinase enzyme elevation was correlated with LV dilatation, these data were not differentiated among the 3 patterns of dilatation. What mechanisms are responsible for producing these types of dilatation over the follow-up period?

What are the related factors and the meaning of the 3 different patterns of LV dilatation? Although the pattern of dilatation did not provide any additional prognostic information, perhaps the mechanisms associated with these patterns might yield important new relationships regarding methods of myocardial preservation. Beyond establishing a patent infarct-related artery, stabilizing microvascular integrity, and reducing the time to reperfusion, are there adjunctive methods of myocardial preservation, such as intravenous or intracoronary adenosine, that can be used to further limit LV remodeling?

Bolognese et al re-emphasize that the LV remodeling process is directly linked to the infarct size and the extent of wall motion abnormality during the acute phase of infarction. Over time, remodeling produces a hemodynamic improvement of a compensatory nature (lowering LV filling pressure and increasing cardiac output), which seems to occur at the expense of significant increase in left ventricular chamber volumes and ultimately survival. Improving methods of myocardial preservation and protection during acute infarction with less late remodeling will close missing links for patient survival after acute infarction.

References


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