Editors

Patterns of Left Ventricular Dilatation With an Opened Artery After Acute Myocardial Infarction
Missing Links to Long-Term Prognosis
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Ventricular remodeling, the geometric adaptation to injury after acute myocardial infarction, affects the function of both non-infarcted and infarcted muscle, as well as prognosis. Ventricular dilatation bodes especially poorly for late survival.1 It has long been recognized that early infarct expansion is the result of lengthening of the noncontractile region undergoing a stress response with secondary volume overload hypertrophy, a process which maybe progressive over time.2–4 The extent of the initial myocardial damage is linked both to the magnitude and, to a lesser degree, the timing of left ventricular (LV) dilatation and ultimately survival.5 Moreover, ventricular remodeling (enlargement) is influenced not only by infarct size but also the type of infarct healing and coexistent LV wall stresses.5

Further insight into the sequence and significance of LV responses is provided by Bolognese et al.8 In their examination of 284 consecutive patients undergoing primary percutaneous coronary intervention (PCI) for acute myocardial infarction, Bolognese et al8 obtained serial echocardiographic and angiographic studies at 24 hours, 1 month, and 6 months. Late followup (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 as compared with those without LV dilatation. End-systolic volume at 6 months and age were strong predictors of late cardiac death. Three patterns of dilatation, early, late, and progressive, were identified. Early dilatation occurred between 24 hours and 1 month, late dilatation occurred from 1 month to 6 months, and progressive dilatation was defined as a continuum of enlargement from day 1 through 1 and 6 months. These 3 patterns were detected in 15%, 14%, and 13% of the ventricular dilatation group, respectively. However, there was no difference in survival statistics among the 3 patterns of LV dilatation. Thus, LV dilatation in general, but not a specific pattern of dilatation, was associated with a poor long-term outcome.

An opened artery hypothesis was also challenged in this study.8 Similar low restenosis and reocclusion rates during the 6-month follow-up period were present in patients with and without LV remodeling (restenosis rates were 23% versus 24% and reocclusion was 5% versus 6%, respectively, both \( P=NS \)). Restenosis rates among the 3 patterns of LV dilatation were similar (15%, 27%, and 31%, all \( P=NS \)), as were the reocclusion rates (2%, 5%, and 6%, respectively). Thus, at 6 months, there was an unfortunate 30% of patients with patent infarct related arteries who went on to have LV dilation. An open artery also did not link with a specific pattern of dilation.

The investigation of Bolognese et al8 closes some of the links between LV remodeling and patency of the infarct-related artery, in that remodeling, more than late artery patency, influences prognosis even among successfully reperfused patients. This article8 highlights a novel observation, namely that LV remodeling after acute myocardial infarction occurs in a heterogeneous fashion with no specific pattern of LV dilatation differentiating patient survival.

The investigators should be complemented for the detailed long-term study of this relatively low-risk population, treated with optimal reperfusion therapy and having serial coronary...
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, perfusion status of the infarct-related artery, 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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