Correlating infarct size and patient prognosis: are cardiac biomarkers truly insufficient?

I read the recent article by Lonborg et al., on the prognostic value of late gadolinium enhancement-cardiovascular magnetic resonance (LGE-CMR) in ST-segment elevation myocardial infarction (STEMI) patients, with great interest.

Recently, a single troponin measurement at either 72 or 96 h post-infarction provided an accurate estimate of infarct size and the prognostic impact of this has also been demonstrated. As myocyte necrosis culminates in the formation of scar tissue, it is unsurprising that there is also a relationship between magnitude of troponin release and scar burden assessed by LGE-CMR. LGE-CMR allows visualization and measurement of infarct size but, due to logistical and economical barriers, cardiac biomarkers remain the most cost-effective means of estimating the infarct size in contemporary clinical practice. Furthermore, in our times of global austerity, it is imperative that a newer, more expensive test (in this case LGE-CMR) demonstrates just not equivalent but clear superiority over the current test of choice (troponin).

Against this background, the results of the Cox regression analysis are particularly important. The authors highlight that infarct size by LGE-CMR was a predictor of outcome in both univariable and multivariable analyses but neglect to highlight that peak troponin T concentration was similarly an independent predictor of outcome! Thus, both parameters are significant over and above adjustment for the other (and the other variables in the analysis).

The second point regards the composite outcome selected: there were just eight hard events and 27/35 (77%) events were hospitalization for heart failure (HF). While this is an important outcome with associated cost implications, prompt attention in the community for patients with worsening breathlessness or peripheral oedema, as can be provided by HF nurse specialists, can allow appropriate tailoring of medical therapy (e.g. increased dose of diuretics) which has been shown to reduce rates of hospitalization. Consequently, it is difficult to extrapolate these rates across countries due to variations between health care systems. Additionally, the reasons for HF hospitalization are numerous, ranging from a poor social support network to non-compliance with medication. It is for this reason that hard events, such as death or myocardial infarction, are the preferred outcome measure for outcome (prognosis) studies.

Thirdly, and most importantly, the authors have acknowledged that the key clinical question here is whether making a clinical decision based upon the LGE-CMR result improves patient outcome significantly more than if the decision were made based upon ejection fraction or, for that matter, peak troponin concentration. As pharmacological secondary prevention measures would be similar whether the infarct was small or large, the key issue pertains to selection of patients for defibrillator therapy. The authors are correct that a randomized controlled trial is now warranted to determine whether the significantly greater up-front cost of routine LGE-CMR in STEMI patients (compared with the cost of a troponin assay) is outweighed by the incremental clinical benefit this information provides. Until that trial is performed; however, peak troponin T concentration appears an entirely reasonable—and affordable—method for estimating infarct size.

References

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Correlating infarct size and patient prognosis: are cardiac biomarkers truly insufficient?: reply

We welcome the insightful and constructive comments by Dr Benoy N. Shah with regard to our recent paper on the association between final infarct size measured by cardiovascular magnetic resonance (CMR) and long-term outcome in patients with ST-segment elevation myocardial infarction (STEMI).1

Dr Benoy N. Shah questions the use of admission for heart failure as a clinical endpoint, since it may be more prone to bias than reinfection and mortality. While we agree that admission for heart failure is not quite as straightforward to define as reinfection and mortality, admission for heart failure may still be considered as a well-defined endpoint, and is used frequently. Also, the two latter endpoints may also be prone to bias affected by poor social status, comorbidity, and non-compliance to medications including antithrombotic. For the same reason, we adjust for

References
some potential confounding factors in the multivariable analysis. Reinfarction relates to the disease within the coronary artery lumen, whereas heart failure relates to the function and damage to the myocardial. Thus, when it comes to cardioprotection and reduction in infarct size, mortality and heart failure seem to more relevant endpoints than reinfarction. Moreover, 4.7% develops clinical heart failure 1-year following a STEMI and 35% develops heart failure following discharge over 10 years. In elderly, the prevalence of heart failure following acute myocardial infarction suggests recovery of the peri-infarction zone: one-year follow-up by MRI. Circ Cardiovasc Imaging 2009;2:47–55.

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References