A good thing after all? Raised cardiac enzymes after PCI

In this issue of *Acute and Interventional Cardiology* there is a commissioned editorial about the release of cardiac enzymes after percutaneous coronary intervention (PCI) (see pages 3–6).[1] This remains a contentious area and attracts the views of two opposing schools of thought. To the first group the data demonstrate that any enzyme release following PCI is undesirable and associated with adverse outcome. These are the 'enzyme enthusiasts', and they are dedicated to the routine measurement of CKMB or troponin in all patients following PCI. They feel that by knowing enzyme blood levels they can learn more about the PCI that they undertake and the problems that arise from it. Such analysis could potentially lead to modifications in technique or possible patient selection.

The second group, the 'enzyme sceptics', are clear in their minds that whilst substantial elevations in enzymes (for example creatine kinase more than three or five times the upper limit of normal) represent important myocardial damage, in the same way that a new Q wave does, smaller elevations are irrelevant clinically. To the latter group, the routine measurement of post-PCI enzymes in all patients is a costly and clinically irrelevant exercise – most people have gone home long before the result comes back. Furthermore, to the enzyme sceptic, the data are discrepant about the significance of all but the highest enzyme levels and they feel that they detect a problem in patients who develop these high results either during or after the procedure anyway.

It is unsurprising, given the crude mechanical nature of the process of balloon disruption and stent insertion, that some degree of myocardial injury occurs. The friable nature of thrombus and some plaque types, as well as the tendency for the coronary arteries to give rise to side branches, surely make this inevitable. The important question, therefore, becomes: 'does an increase in cardiac enzymes after PCI necessarily indicate something bad?' The data in the literature are diverse and conflicting, particularly for the group with relatively modest elevation of enzymes that are sometimes called 'infarctlets'.[2]

A recently published study has the potential to change our perception of the post-PCI enzyme rise in a radical way.[3] In this paper, a clear cut association was demonstrated between the post-PCI level of CKMB and the degree of expansion of stents during the procedure, as assessed on intravascular ultrasound (IVUS) and defined as the ratio of final lumen over the reference lumen cross-sectional area. Peak CKMB values increased significantly with increasing stent expansion. Furthermore, the level of the CKMB was then shown to be inversely related to the target lesion revascularisation rates at one year follow-up! The authors, conclusion was: 'Increased periprocedural CKMB release appears as a trade-off for optimal stent implantation and lower clinical restenosis'.

This is an exciting and thought-provoking piece of work. Anyone who has performed serial IVUS after stent deployment is familiar with the high frequency of angiographically undetected regions of stent under-deployment. The lessons from the literature regarding the reduction of restenosis by scrupulous attention to the detail of stent sizing and optimal expansion (usually with IVUS guidance)[4] have been both clear and consistent but are possibly being forgotten in the era of the drug- eluting stent.

> ***'A recently published study has the potential to change our perception of the post-PCI enzyme rise in a radical way'***

Perhaps this most recent paper provides not only some decent ammunition for the 'enzyme sceptics', but also reminds us to obey the golden rules of stent deployment... as long as we are prepared to have higher enzyme levels afterwards! It may just be not such a bad thing after all.

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