Greek technology or mythology?

See doi:10.1053/euhj.2002.3174 for the article to which this Editorial refers

The study by Stefanadis et al.[1] in this issue indicates that treatment with a variety of statins reduces the release of heat from atherosclerotic plaques in the human coronary artery. This observation suggests that statins exert a beneficial effect on the inflammatory process that exists in the vascular wall in a variety of acute coronary syndromes.

The finding that this effect was most pronounced in patients with acute myocardial infarction subject to primary angioplasty within 6 h of onset of pain and least in exertional angina patients with in-between values for unstable angina, indicates further that the antinflammatory action of statins is most pronounced in individuals where the active process is most likely to exist. These findings correspond to other reports in which statin therapy reduces other signs of inflammation such as C-reactive proteins[2].

A direct action on infiltrating macrophages is thereby suggested, leading to the supposition that plaque stabilization is achieved, a theory also underlying the clinically demonstrated efficacy of this type of therapy in the large scale CARE[2] and MCRC/BHF[3] trials. It is of interest to note that cholesterol levels changed little in the Greek patients.

Whether this makes thermography a method to detect such lesions, with an eye on specific plaque oriented-therapy, is another matter. The authors reflect on some of the technical limitations that this method is subject to. In particular, the localization of the thermistor tip, verified in part by IVUS and angiography, may be questionable, the more so since it is now generally accepted that the atherosclerotic process occurs more diffusely in the coronary artery wall. So, exactly where is the inflammation?

The observations in the ENCORE[4] and TREND[5] trials where after acetylcholine provocation, abnormal contraction patterns were observed in over 90% of ‘clinically stable’ patients, indicate that thermography may detect the more acute lesions rather than that it can accurately detect diffuse disease in adjacent segments. Nevertheless, it is clear that the efforts made since its introduction in 1998 by this group of Greek investigators is now bearing fruit. Their closing comments, that future prospective studies now ongoing, are needed to confirm the statin’s impact on plaque stabilization (and thereby the predictive value of the thermographic method) are entirely correct. The results are eagerly awaited to see whether Greek technology can eliminate the current mythology. The recently presented data on the LIPS trial, where routine administration of fluvastatin in all PTCA patients drastically reduced all major cardiac events, would seem to suggest so[6].

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References