Letters to the Editor

doi:10.1093/eurheartj/ehi429

Online publish-ahead-of-print 21 December 2006

Gamma-glutamyltransferase, leukotrienes, and cardiovascular risk

It is with great interest that I read the article 'Serum gamma-glutamyltransferase predicts myocardial infarction and fatal coronary heart disease among 28 838 middle-aged men and women' by Lee et al. 1 As pointed out by the authors, previous studies have brought the attention to a possible link between gamma-glutamyltransferase and inflammation in atherosclerosis. 1 In this context, it can be pointed out that, in addition to effects on glutathione metabolism and redox regulation, the gamma-glutamyltransferase enzyme also uses leukotrienes as substrate. 2 Leukotrienes are lipid mediators of inflammation derived from the 5-lipoxygenase pathway of arachidonic acid metabolism. Recent studies have provided evidence for a strong genetic link between this pathway and increased risk of myocardial infarction, and the effects of a leukotriene synthesis inhibitor have been evaluated on biomarkers of cardiovascular risk. 3 Experimental studies have implicated the dihydroxy-leukotriene LTB 4 in pathophysiological reactions of atherosclerosis and restenosis. 4 In addition, increased cysteinyll leukotriene formation has been detected in subjects with early atherosclerosis. 5

Gamma-glutamyltransferase catalyses a transeptidation of the amino acid side chain of the cysteinyll-leukotrienes. 2 This leads to an interconversion of the two vasoactive leukotrienes LTC 4 and LTD 4 , which can influence the pharmacology of leukotriene-induced responses. 2 Leukotriene metabolism could hence propose one possible mechanism behind the link between gamma-glutamyltransferase and inflammation in cardiovascular disease.

References


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Myocardial deformation to determine transmurality of myocardial infarction

We read with interest the article by Becker et al. 1 in which they used speckle (or pixel) tracking echocardiography to measure deformation or strain in patients with myocardial infarction to determine transmurality. Their references to previous work in this area are a little disingenuous. They state that all three earlier studies were ‘experimental’ including our own. 2 In fact our study was almost identical in design, including the use of ce-MRI, with similar results to their own and was not an experimental animal study. We examined consecutive myocardial infarction patients and used tissue Doppler rather than speckle tracking to measure strain rate. We found that a reduced strain (>-0.59 s⁻¹) detected a transmural infarction with high sensitivity (91%) and specificity (96.4%) which is slightly better than Becker and his colleagues results with speckle tracking. 1 Although there are theoretical advantages to speckle tracking particularly the lack of angle-dependency Doppler-based strain rate imaging is widely available and relatively easy to analyse. It is a pity that Becker and colleagues did not do a direct comparison of the two techniques to demonstrate that speckle tracking is indeed superior as they claim.

References