Letters to the Editor

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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 LTb4 in pathophysiological reactions of atherosclerosis and restenosis.4 In addition, increased cysteinyl-leukotriene formation has been detected in subjects with early atherosclerosis.5

Gamma-glutamyltransferase catalyses a transpeptidation of the amino acid side chain of the cysteinyl-leukotrienes.2 This leads to an interconversion of the two vasoactive leukotrienes LTC4 and LTD4, 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 theoretical advantages to speckle tracking. Indeed superior as they claim.

References


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Myocardial deformation to determine transmurality of myocardial infarction?: Reply

We read with interest the letter by Zhang et al.1 discussing their own as well as our article. In their own study, Zhang et al. used myocardial velocity and Doppler derived strain in patients with an acute myocardial infarction to define the transmurality of myocardial infarction.2 Doppler-derived strain and strain-rate analysis have been shown to be very angle dependent and limited by significant noise artefacts.3

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