Platelet activation, gamma-glutamyltransferase and stent restenosis Comment on the article by Ulus et al.

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Ulus et al. [1] report convincing evidence showing that serum gamma-glutamyltransferase (GGT) activity may be an independent marker for stent restenosis. This observation adds to other studies showing that GGT activity is related to the progression of vascular disease towards life-threatening events such as myocardial infarction and stroke [2–4].

The association of serum GGT with stent restenosis prompts two considerations. Firstly, it is conceivable that serum levels of the enzyme may contribute to accumulation of GGT in the diseased arterial wall [5], possibly following the insudation of beta-lipoproteins [6]. Prooxidant reactions promoted by GGT activity within the arterial wall might participate at several levels during disease progression [7].

Secondly, the observation by Ulus et al. may provide additional insight as to the source of elevated GGT levels, in this as well as other conditions with increased cardiovascular risk. Thus, human platelets may contribute to circulating levels of GGT [8,9]. Some evidence indicates that platelet activation may play a role in the pathogenesis of restenosis [10], with the release of growth factors – e.g., serotonin – capable of triggering and maintaining this process [11].

In conclusion, evidence is accumulating that serum GGT activity is related to cardiovascular prognosis, both as a factor contributing to atherogenesis as well as a marker of platelet involvement during vascular complications—that is, a role akin to that recently proposed for another platelet-related marker, soluble CD40 ligand [12].

References


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