Insulin like growth factor-1 and lipoprotein metabolism in stable angina patients

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We read the article: "Association between insulin like growth factor-1 and lipoprotein metabolism in stable angina patients on statin therapy: a pilot study" by Burchardt et al. [1] with interest. The authors concluded that owing to association with products of lipid oxidation, oxidised proteins, and high values of Lp(a); IGF-1 and IGFBP3 levels could be useful indicators of atherosclerosis progression. We believe that these findings should act as a guide for further studies.

IGF-1 plays an important role in the cell protection of multiple systems, where its signal transduction helps to preserve tissues from hypoxia, ischaemia and oxidative stress. IGF-1 and its binding proteins (IGFBP) have association with hypertensive patients with left ventricular hypertrophy and arteriosclerosis, especially coronary artery sclerosis [2, 3]. IGF is produced mostly by the liver and transported by their own binding proteins (IGBP) in the circulation. Seven different IGFBPs have been reported to date, with IGFBP-3 being the most abundant. The effects of these proteins not only bind IGFs, but also have receptors in tissues. An increase in IGFBP-3 levels in patient with a chronic kidney disease (CKD) is shown [4]. IGF levels have been affected in different ways in CKD. It would be better if it could be clearly stated that CKD and liver diseases are excluded from the study.

Conflict of interests: none declared

References

Author’s response

I would like to thank Demirkol et al. for their interest and their comments on our article [1]. As to their remarks, of course we agree on the impact of both — kidney and liver — on IGFBP3 and IGF-1 plasma levels. However, the exclusion criteria from the study were provided in the quoted article [2]. For the sake of thoroughness, we would like to emphasise that patients with documented liver disease or its dysfunction (ALT, AST > ULN) were excluded from the study. Also excluded were patients with chronic kidney disease or their dysfunction qualified when GFR was < 60 mL/min. We must admit that the method of assessing GFR by the MDRD rule used by us has its own limitations. Secondly, GFR 60 mL/min was a priori assumed as the cut-off point for kidney disease. Thus, patients with kidney disease in stage I by Kidney Diseases Outcome Quality Initiative (without GFR limitation) or in stage II (90 < GFR > 60 mL/min) could be included in the study, which is another limitation of our protocol.

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References

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