EDITORIAL COMMENT

C-Rreactive protein/albumin ratio in the assessment of risk for in-stent restenosis: Another small piece in the puzzle of vascular inflammation

A relação da proteína c-reativa/albumina na avaliação do risco de reestenose de stent: mais uma pequena peça novasto puzzle da inflamação vascular

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The study presented by Rencuzogullari et al. in this issue of the Journal 1 is part of the large and important body of evidence on the role of inflammation in cardiovascular disease. 2-5 The authors aimed to investigate the ability of the ratio between serum levels of two important acute-phase proteins, C-reactive protein (CRP) and albumin, to predict in-stent restenosis. There is already an extensive literature on the association between CRP and cardiovascular risk, 6-8 but its ability to predict acute coronary syndromes is modest. 9-11 A positive relationship between elevated serum CRP levels and in-stent restenosis, the clinical focus of the study under consideration, has also been established. 12 At the same time, reduced serum concentrations of albumin have been related to increased cardiovascular risk. 13, 14 Furthermore, the CRP/albumin ratio has been associated with worse prognosis in cancer 15 and can be used as a simple predictor of poor overall health. 16

From a clinical standpoint, assessment of the combination of increased CRP and decreased albumin levels makes sense, seeing that these two molecules are involved in a range of complex and frequently interlinked processes ranging from inflammation to malnutrition, and so in theory the ratio between them should have considerable potential as a prognostic marker. However, Rencuzogullari et al. run a risk by attempting to associate this ratio with in-stent restenosis (a serious albeit uncommon complication of percutaneous revascularization) since, like all indices of inflammatory markers used in cardiovascular assessment, it is non-specific.

The study is retrospective, reviewing around six years of data from a single institution. The population was of patients with ST-segment elevation myocardial infarction who underwent coronary angiography and percutaneous coronary intervention following standard indications. Of these, 448 who underwent repeat angiography due to recurrence of anginal symptoms were included in the study, of whom 25% (110/448) presented in-stent restenosis.

Unfortunately, the study did not use high-sensitivity CRP assays, which are firmly established in the literature as the
gold standard for assessing vascular inflammation. The authors acknowledge that their use of an older test, which is less sensitive and more likely to be influenced by rises in CRP due to non-vascular causes, is a limitation to their study. Their results also include a wide range of laboratory results, from lipid profile to platelet count, as well as platelet distribution width, which is associated with myocardial infarction. The study thus has the characteristics of a general review of in-stent restenosis, even though drug-eluting stents, which were designed to be less prone to restenosis, were not used. This limitation was also pointed out by the authors.

The study concludes that the CRP/albumin ratio is a better predictor than CRP or albumin individually, at least in bare-metal stents. Overall, the work has merit, especially in the emphasis it places on the use of inflammatory parameters in clinical practice. However, despite recent successes in proof-of-concept trials and although inflammation has been the focus of much productive and challenging basic and clinical research over the last twenty years, there is still some way to go before all the accumulated evidence can be brought to bear on daily clinical practice.

To conclude, despite its limitations, the paper under consideration adds another small piece to the puzzle in this challenging area in which research continues apace, even though its conclusions need to be confirmed by further studies.

Conflicts of interest
The author has no conflicts of interest to declare.

References