

Salusin beta, atherosclerosis, and coronary slow flow

To the Editor,

We have read the paper written by Akyüz et al. (1) titled "Relationship of serum salusin beta levels with coronary slow flow" with great interest. In this research, the authors compared serum salusin- β levels between two patient groups, those with normal coronary arteries and those with coronary slow flow (CSF) pattern in the absence of significant coronary artery disease. They concluded that serum salusin- β levels could play a role as a biomarker for the evaluation of CSF. In previous studies, salusin- β has been reported to be related to the development and progression of atherosclerosis, and it has been shown that the circulating levels are high in patients with documented atherosclerosis (2, 3).

On the other hand, atherosclerotic plaques are also abundant for consistent salusin- β release. In a previous intravascular ultrasound study, Cin et al. (4) have demonstrated the association of subclinical atherosclerosis with CSF. Therefore, we believe that the increased serum salusin- β levels in patients with CSF may be the effect of subclinical atherosclerosis and can not be evaluated as a biomarker for CSF. Moreover, in the study group, patients with nonsignificant atherosclerosis have not been excluded, and this might be a factor for the increased serum salusin- β levels observed in this group.

The hypothesis regarding the role of serum salusin- β levels in CSF should be tested between patient groups with similar atherosclerotic plaque burden.

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Author's Reply

To the Editor,

Our study (1) included patients with coronary slow flow (CSF) with nonobstructive coronary lesions and individuals with normal coronary arteriogram findings. After measuring serum salusin- β levels and thrombolysis in myocardial infarction frame count (TFC), we found a positive correlation between the mean TFC and serum salusin- β levels ($r=0.564$; $p<0.001$) (1). Therefore, this finding alone is important for the validity of the study. If we had included a third group, including patients with CAD, it would have validated our study even more. We have mentioned this as one of the study limitations. Endothelial dysfunction and microvascular atherosclerosis are two major determinants of CSF (2). Salusin- β is a marker that indicates the presence of micro- and/or macro-atheroma formation. Most visually normal coronary artery segments have various atherosclerotic plaques (3); consequently, we considered excluding patients with nonobstructive coronary lesions as a futile attempt.

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