Coronary slow flow: a potential prognostic marker correlating with the disease severity in renal failure?

Dear Editor,

We read with great interest the study by Sobkowicz et al. that demonstrated significant reductions in coronary blood flow [as measured with increased thrombolysis in myocardial infarction (TIMI) frame counts (TFC)] in patients with end-stage renal disease (ESRD) regardless of the degree of epicardial coronary artery stenosis [1]. We agree with the authors that coronary slow flow (CSF) in ESRD should not be regarded as a guide in determining the severity of epicardial coronary artery stenosis, but should be considered as a separate phenomenon, particularly associated with microvascular dysfunction and enhanced microvascular resistance due to endothelial dysfunction, myocardial hypertrophy, myocardial fibrosis [1], etc. However, in the setting of chronic renal failure (CRF), CSF may also be associated with milder forms of renal disease and may correlate with the disease severity.

In a previous study by our group including a relatively large number of patients \( (n = 105) \) with mild-to-moderate renal dysfunction [calculated glomerular filtration rate (GFR) values between 20 and 90 mL/min/1.73 m\(^2\)] and angiographically normal coronary arteries, we were able to demonstrate significant reductions in coronary blood flow as measured with increased TFC values compared with the control group \( (n = 102, \text{age- and gender-matched}) \) having normal coronary arteries and GFR values [2]. It is of note that, in the patient group of our study, TFC values for all the three major epicardial coronary arteries were also found to be significantly and negatively correlated with the values of calculated GFR, even after adjusting for other factors (blood pressure, etc.) indicating the independent relationship between these two parameters in renal failure [2]. Our study was quite different from the study by Sobkowicz et al. [1] in terms of study design and results: first, the presence of severe renal dysfunction and the presence of pathologies with potential effects on coronary blood flow (left ventricular hypertrophy, epicardial coronary artery stenosis, etc.) were accepted as exclusion criteria in our study. Second, the patient group was compared with a control group, not with the predefined reference values, and third, there was an independent correlation between CSF and renal function in the patient group that might confer important clinical and prognostic information [2].

Endothelial dysfunction is well known to play a central role in the pathogenesis of CSF, coronary atherosclerosis and CRF indicating the complex relationship among these entities. It may be inferred from the findings in our study [2] that CSF (as measured with increased TFC) may also be encountered in patients with mild-to-moderate renal failure and that the degree of CSF may independently mirror the extent of renal dysfunction, and vice versa. It is also possible that the degree of CSF may help predict adverse clinical outcomes including renal and cardiovascular adverse events, and may also serve as a prognostic marker in patients with renal failure. However, the latter is speculative and warrants large-scale studies to be confirmed.

Conflict of interest statement. None declared.

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Reply

We appreciate very much the interesting comment of Yalta and colleagues concerning our work on the application of the thrombolysis in myocardial infarction frame count method (TFC) in the assessment of coronary blood flow in patients with end-stage renal disease (ESRD) [1]. We agree on the importance of further investigation on the complex relationship between coronary blood flow disturbances and kidney disease (KD). The TFC method was not previously validated in patients with KD. As published by Yilmaz and Yalta [2], even in patients with mild-to-moderate renal failure, velocity of coronary blood flow was reduced according to the TFC method. Moreover, the authors were able to demonstrate a negative correlation between

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