

Authors' comments on: Higher red cell distribution width in patients with slow coronary flow

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Dear Editor,

We would like to thank sincerely Balta et al. (1) for their positive comments and kind interest concerning our original research article that was recently published in *Clinics* (2). As indicated in the authors' comments, red cell distribution width (RDW) is a measurement of the size variation in red blood cells, as well as an index of the heterogeneity of the circulating erythrocytes, which is commonly utilized in the differential diagnosis of underlying anemia (3). More recently, RDW has been shown to be a marker and independent predictor of a variety of coronary artery diseases, including unstable and stable clinical entities and even microvascular disorder (4,5). However, it remains unclear whether anisocytosis might be the cause or a simple epiphenomenon on of the underlying disease (3). Apparently, our published study provided a novel finding regarding the role of RDW in predicting the presence of slow coronary flow.

We agree with authors' comment on the potential impacts of different pathophysiological backgrounds, such as inflammatory status, on RDW (1). The data in our study showed a positive correlation between RDW and plasma C-reactive protein levels, which could be indirect evidence that RDW is influenced by a variety of inflammatory diseases.

In fact, a number of useful biomarkers for predicting the future development of cardiovascular disease, as well as the outcomes in patients with cardiovascular diseases, are clinically available. However, future studies are needed to evaluate whether these biomarkers are useful in patients with slow coronary flow. We previously reported that the plasma concentrations of CRP and interleukin-6 were higher and were positively correlated with the thrombosis in myocardial infarction frame count in patients (n=36) with slow coronary flow, compared with normal coronary flow subjects (n=20), suggesting that an inflammatory response might be involved in the pathogenesis of slow coronary flow (6).

With regard to the time that we used to measure the RDW levels in our study, we appreciate the authors' scientific comments on our manuscript and agree with the authors' point that delaying blood sampling can cause abnormal results in RDW measurement (1). We determined the RDW levels within one hour after the blood samples were collected from all of the patients.

Finally, to the best of our knowledge, the mechanisms involved in the manifestation of slow coronary flow appear multifactorial and are not yet fully understood. Based on the observations reported, we previously hypothesized that an inflammatory process was involved in the development of slow coronary flow (7). We are aware that RDW alone, without any other inflammatory indicators, cannot provide clinicians with sufficient information about the inflammatory status and prognostic indications of patients with slow coronary flow, as the authors indicated (1). Nevertheless, RDW is an easy, inexpensive, and routinely reported test, the assessment of which might allow for the acquisition of significant diagnostic and prognostic information in patients with cardiovascular disorders (3). Further detailed evaluations are needed to build upon the findings reported in our manuscript.

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No potential conflict of interest was reported.

DOI: 10.6061/clinics/2013(09)17