LETTERS TO THE EDITOR

C-reactive protein: not only a marker but also a mediator of myocardial damage following acute myocardial infarction

I have greatly enjoyed reading the recently published article by Ørn et al.1 assessing the relationship between inflammatory mediators, including C-reactive protein, and indices of infarct size and left ventricular remodelling following successful primary percutaneous coronary intervention (PCI) in patients with first-time ST elevation myocardial infarction (MI).

With improved understanding of the critical role of inflammation in atherothrombosis, attention has focused on the inflammatory biomarker C-reactive protein as a risk marker.2 C-reactive protein, an acute-phase reactant, plays an important role in innate immune response, and it is now recognized to be a mediator of atherothrombotic disease.2 As in other types of tissue injury, acute MI (AMI) also generates an acute-phase reaction. The deposition of C-reactive protein in the infarcted region, co-localizing with activated fragments of complement system, indicates that complement activation enhances local inflammation during AMI.3 C-reactive protein has been reported to co-localize with activated complement fragments in infarcted myocardium in patients who died due to AMI.3 Moreover, C-reactive protein is not only a marker of the amount and activity of circulating pro-inflammatory cytokines but may also contribute to inflammation in ischaemic myocardium by activating complement system. Magadle et al.4 showed that pre-procedural serum high-sensitive (hs)-C-reactive protein levels in patients with AMI undergoing primary PCI might be considered a powerful predictor of early complications. Several studies have demonstrated that hs-C-reactive protein measured at either presentation or hospital discharge may have prognostic value in patients with acute coronary syndromes.5 Another clinical study demonstrated that hs-C-reactive protein levels on admission may predict the efficacy of reperfusion in patients with AMI.6

In the recently published article, our group demonstrated that hs-C-reactive protein levels on admission in patients with AMI undergoing primary PCI are likely to be in the causal pathway leading to the development of poor myocardial perfusion, especially when combined with prolonged pain to balloon time.7 In that study, the study population consisted of 75 patients admitted with acute anterior MI and underwent primary PCI in the left anterior descending coronary artery. Myocardial perfusion was evaluated by using TIMI myocardial perfusion grade. In multivariate logistic regression analysis, hs-C-reactive protein levels and pain to balloon time were detected to have statistically significant independent association with poor myocardial perfusion. Adjusted odds ratio was calculated as 1.85 for hs-C-reactive protein (P = 0.003; CI = 1.23–2.80).

In conclusion, when considering the clinical significance of admission high C-reactive protein levels in patients with AMI, it can be concluded that the development of poor myocardial perfusion may partially explain the relation between high C-reactive protein levels and poor clinical outcomes. I considered that poor myocardial perfusion after primary PCI is not only related to procedural factors and clinical characteristics of the patients but may also be related with microvascular damage starting before PCI.

References

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Abnormal left ventricular relaxation in patients with long QT syndrome

We read with great interest the recent report by Haugaa et al.1 and the accompanying editorial by De Ferrari and Schwartz2 on the association between abnormal ventricular repolarization and mechanical dysfunction (prolonged contraction and/or impaired diastolic function) in patients with long QT syndrome (LQTS). We were particularly interested by the statement of Haugaa et al.1 that ‘these findings imply an impairment of diastolic function in a number of symptomatic LQTS mutation carriers’. In keeping with this view, Moss et al.3 also linked the prolonged ventricular repolarization in LQT3 patients (SCN5A-ΔKPQ mutation) with slowed left ventricular (LV) relaxation.3 In these patients, the mean QTc was 57.8 ± 5.5 ms, LV isovolumic relaxation time (IVRT) was 125 ± 27 ms, mitral E-wave deceleration time was 289 ± 80 ms, and mitral E-wave velocity was 57 ± 8 ms, suggesting minor diastolic dysfunction.3 Shortening of the QTc interval by 26 ± 3 ms with ranolazine, a drug that inhibits the late Na current, resulted in significant

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