What about pleiotropic modifiers of the pre-procedural pro-inflammatory and pro-oxidant milieu in patients undergoing drug eluting stent implantation?

Dear Editor,

In the July 2015 issue of your journal, Tanindi et al.[1] presented a study entitled 'Do pre-procedural laboratory parameters predict drug-eluting stent restenosis?', in which the authors investigated the predictors of ISR after implantation of second generation drug eluting stents (DES) among patients with stable angina pectoris. I congratulate the authors on their work, and would also like to draw attention to the following:

1. The exact pathophysiological mechanisms of coronary in-stent restenosis (ISR) have not yet been fully elucidated, but are thought to consist of inflammation, proliferation, and extracellular matrix remodeling.[2,3] There exist several studies on implementing a risk model including clinical, peri-procedural and biological factors for risk prediction and patient risk stratification.[4–6] Although the predictive value of several hematological and biochemical markers which exert either direct and/or indirect pro-inflammatory and pro-oxidant effects have been investigated in this study, only diabetes mellitus and post-procedural residual stenosis were found to be independent predictors of ISR. In the literature, diabetes mellitus is known as the strongest clinical risk factor for ISR.[2]

2. Among biological factors, the study serum CRP levels were shown as the significant predictor of ISR after bare metal stent implantation.[2] However, such an association has not been evidenced after DES implantation, which might be due to the inflammation-altering effects of DES.[6]

3. In the study, 66.6% of patients had hypertension, 41.9% diabetes mellitus and 64.4% hyperlipidemia. However, the medications for all those cardiometabolic risk factors (e.g. statins, anti-hypertensives and anti-diabetics), which might alter the pre-procedural pro-inflammatory and pro-oxidant milieu, have not been reported in the paper. Also, due to its well known anti-inflammatory effects, the rate of previous aspirin usage should have been presented. Furthermore, the authors should have highlighted the reasons in accordance with evidence and/or experience for routine 6–12th month control coronary angiography after percutaneous coronary intervention (PCI) in their clinical practice.

In conclusion, pre-PCI medications for cardiometabolic risk factors may alter pro-inflammatory and pro-oxidant milieu which were known as the important risk factors for ISR.

Uğur Canpolat, M.D.

Department of Cardiology, Hacettepe University Faculty of Medicine, Ankara

e-mail: dru_canpolat@yahoo.com

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