Factors associated with periprocedural myocardial infarction

To the Editor,

We have read with great interest the article by Yao et al. (1) on the association between baseline CRP levels and the occurrence of periprocedural myocardial infarction. It is reported that higher baseline CRP levels are associated with increased periprocedural myocardial infarction incidence. Patient medications, except statins and antiplatelets, were not assessed, and this was reported as a limitation (1).

Smoking status is an important issue because it has several adverse effects on endothelial functions. Moreover, smoking results in the induction of CYP450 enzyme system and in the increased metabolism of clopidogrel (2). Therefore, smoking decreases the antiplatelet effects of clopidogrel, and it may play a significant role in periprocedural myocardial infarction.

Clopidogrel is an effective P2Y12 inhibitor that prevents stent thrombosis and restenosis; however, it does not exhibit a same effect in all patients. Certain patients are resistant to antiplatelet drugs, and there exists a risk of major adverse cardiovascular events among these patients. High-on treatment platelet reactivity (HPR) defines inadequate antiplatelet response in patients undergoing antiplatelet therapy with optimal dose. Patients with HPR are prone to periprocedural stent thrombosis and restenosis. Therefore, such patients should be identified using platelet

reactivity tests (3). Moreover, it has been reported by Patti et al. (4) that inflammation is associated with HPR and increased inflammation is associated with decreased antiplatelet response to clopidogrel.

To conclude, being important determinants of periprocedural myocardial infarction, it would have been better if smoking status and HPR were assessed.

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