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Author`s Reply

To the Editor,

I thank the authors for their interest. The first title of the case report was "Concomitant left main coronary artery and prosthetic mitral valve thrombosis treatment: Improvisation is a must!" to emphasize the uncertainty and need for versatility while treating these patients. There were two patients treated by percutaneous coronary intervention in Yesin et al.'s (1) paper, but there is no detail about the amount and quality of coronary thrombotic material. I think that there are two kinds of thrombotic coronary materials in these patients: easily dispersible and lysable and denser, bulkier coronary thrombotic material. I am not aware of any autopsy or thrombus aspiration study in such patients characterizing thrombus qualities, and Yesin et al. (1) study cannot be accepted as the last verdict in these patients due to limitation in describing coronary thrombus quality and amount. Our case fundamentally differs from their patient group by left main coronary artery occlusion and urgent need for terminating coronary ischemia. In Yesin's study, only 19% of the patients were receiving aspirin, and none were on clopidogrel on admission that reduced the bleeding rate in their protocol. I retrospectively think that low-dose and ultraslow fibrinolytic therapy (25 mg/25 h) was safer in our patient due to aspirin and clopidogrel treatment necessitated by stent implantation and suggested 25 mg/6 h protocol would increase bleeding risk. Heparin infusion was necessitated due to intra-aortic balloon pump (IABP) use in the follow-up. Our patient is also different from the TROIA and PROMETEE patient groups that did not include any patient with IABP (2, 3). As a result, it is very difficult for authors to say "total disagreement on heparin use" by referencing the TROIA and PROMETEE trials because both were not enrolling any patient on IABP. Necessary precautions were taken for tPA stability during a 24-hour infusion. We do not know the thrombus size before the 2nd episode of tPA infusion because TEE was not performed again due to the general condition of the patient and good transthoracic image quality showing stuck mitral leaflet.

In conclusion, it is very difficult for authors to claim low-dose and slow infusion TT to be a better treatment strategy in our patient because their referenced studies did not include any patient on IABP.

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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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Author`s Reply

To the Editor,

Thank you for your careful appraisal. We are extremely glad to receive the insights regarding our article (1), and we appreciate your careful reading and profound comprehension of the periprocedural myocardial infarction incidence. We would like to have the opportunity to respond to the concerns raised in the related letter.

1. We had considered that smoking may play a significant role in periprocedural myocardial infarction when the study had begun; however, no statistical significance was observed for smoking in this cohort.

2. Regarding the role of high-on treatment platelet reactivity (HPR), we are apologetic that we did not detect HPR using platelet reactivity tests before and after antiplatelet drug administration in this retrospective study. We did not regularly detect HPR, because neither platelet function testing nor genetic testing can be recommended for tailoring DAPT, as per the guidelines (2).

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