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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