

Peer Review File

Article Information: <http://dx.doi.org/10.21037/apm-20-1728>

Reviewer Comments:

The manuscript entitled “Platelet-fibrin clot strength measured by thromboelastography could predict hypercoagulability and antiplatelet effects in patients after percutaneous coronary intervention” was reviewed. The manuscript is well written, however, specific comments are listed below.

Response:

We thank the Reviewer for the positive comments about our study. We have followed your suggestions and made every possible effort to address the concerns. Detailed responses are below.

Specific comments

1. Abstract

“c (OMI)” should be “old myocardial infarction (OMI)”.

Response:

We are sorry for this clerical error and have revised it.

2. Methods

Since the authors consider OMI, UA, NSTEMI and STEMI were CAD stage, comparison of these groups should be performed by ANOVA, not T test.

Response:

We thank the Reviewer for the constructive suggestion about our study. We according have revised the sentence as follows: Unpaired two-sides Student's t-test was used to compare normally distributed continuous variables between two groups and the one-way ANOVA with LSD was used to compare among ACS groups (UA, NSTEMI, and STEMI).

3. Results

Some parts of the Result section (ex; line 193-196) should move to the Methods section.

Response:

We thank the Reviewer for the constructive suggestion about our study. We accordingly have moved this section to the methods section: MA_{ADP} was divided into trisections (T1-T3) according to one previous study, which suggested that cut points between 31 mm and 47mm would be the best predictive values for long-term post-PCI ischemic and bleeding events. A therapeutic range of 31 to 47 mm for MA_{ADP} could provide maximum efficacy and safety.

4. Results

Line 198 and 201 (35.7% of NSTEMI, 27.6% of NSTEMI), and line 257 and 260 seems to not correspond to Figure 2 (height of bar).

Response:

We are sorry for this error and accordingly have revised the Figure 2 that is correspond to Table 3 and manuscript.

5. Table 4

“ * ” and “ ** ” in the Table 4 and “*p<0.05, **<0.001” below the Table 4 were not necessary.

Response:

We thank the Reviewer for the constructive suggestion about our study. We accordingly have revised it.

6. Discussion

The authors should mention possible mechanism of effect of different disease condition (UA, NSTEMI or STEMI) on MA-thrombin and MA-ADP.

Response:

We thank the Reviewer for the constructive suggestion about our study. We

accordingly have added the statements as follows: The probable mechanisms could explain these phenomena, of which the exact biologic mechanisms might not be fully understood. UA and NSTEMI are caused by severe coronary lesions and repeated plaque ruptures, inducing platelet activation, and enhancing platelet aggregating function in a relative long term. In comparison, coronary plaque rupture leads to platelet aggregating immediately in STEMI, leading to the formation of coronary thrombus.

7. Discussion

The reviewer totally agree personalized antiplatelet therapy would be needed after PCI. However, according to Figure 1, MA-thrombin and MA-ADP level were overlapped in many patients in each group. So it seems to be difficult to differentiate whether particular patient need more aggressive antiplatelet therapy or not according to MA-thrombin or MA-ADP level. The authors should mention possible strategy of personalized antiplatelet therapy based on current study.

Response:

We thank the Reviewer for the constructive suggestion about our study. We accordingly have mentioned possible strategy of personalized antiplatelet therapy as follows: It is well known that many factors could affect the on-treatment platelet reactivity, including modifiable factors, such as smoking, high body mass index, drug interactions, as well as non-modifiable factors, such as genetic polymorphisms, age,

sex, and chronic kidney disease. Besides, both ischemic risk and bleeding risk should also be taken into consideration when choosing antiplatelet drugs for CAD patients. All these factors could contribute to contemplate strategies of tailored antiplatelet regimens that included the use of more potent P2Y₁₂ inhibitors. According to our results, strengthened antiplatelet therapy should be considered in patients with higher ischemic risk and relatively lower bleeding risk. Prasugrel and ticagrelor, as newer P2Y₁₂ receptor inhibitors, could reduce the thrombotic events without significantly increased bleeding events compared with clopidogrel. Studies also confirmed that few ACS patients (about 3.98%) using ticagrelor were in MAADP >47 mm. Accordingly, current clinical guidelines recommended a potent P2Y₁₂ inhibitor (prasugrel or ticagrelor) as a preference to clopidogrel for ACS patients. Nevertheless, high on-treatment platelet reactivity also observed in patients with prasugrel and ticagrelor, which correlated with the occurrence of ischemic events. Therefore, more exploration is needed in the individualized antiplatelet medication.