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Jailing Polymer Jacketed Guide-wires during Bifurcation Coronary Interventions is Associated with Procedural Myocardial Infarction

RESPONSE TO REVIEWERS

We are grateful to the reviewers for their time and learned comments. Our response to the individual reviewers' comments are listed below:

Reviewer 1

This is an interesting manuscript about the relation of jailing polymer jacketed guide wires (PGW) to procedural myocardial infarction (PMI). The authors demonstrated that jailed PGW might be associated with PMI. This manuscript is nicely structured and well written. I have one minor comment about this manuscript. Please consider the following comment. (Comment) 1. Figure 1 I think the second from bottom probably stand for jailed PGW use. What does the bottommost express?

Response

We thank the reviewer for the encouragement and positive feedback. We apologize for the confusion regarding labels in Figure 1. These have been corrected. The bottommost bar represents use of "kissing balloon angioplasty".

Reviewer 2

Interesting data there are no specific comments.

Response

We thank the reviewer for the encouraging review.

Reviewer 3

The authors present a very interesting point with their manuscript. The results, although the methodology is highly debatable (matching of the control group, PMI definition, type of biomarkers -nowadays CKMB is used less frequently-, type of procedure, renal function..) are reasonable. The only essential issue I missed is the procedure final result. I mean, The final Timi III (main and side) is needed to interpret this paper. In other words, you can jail or not your wire , but if the vessel is closed or the flow is compromised, biomarkers will raise. To conclude, this design is more a hypothesis generator than a proof of concept, and this must be clear in the paper, rather than trying to convince us that it has been done in detail (which is assumed).

Response

We are thankful to the reviewer for his / her suggestions and agree that there are limitations in our data as correctly pointed out.

1. We agree that TIMI 3 flow in both branches is the major determinant of procedural myocardial infarction. Factors such as plaque shift, no or slow reflow etc are logically much more important than the hypothetical question of polymer embolization. Hence our study design only looked at cases where there was no clear cause of myocardial infarction. We had clubbed these few cases with the “no reflow” and “SB occlusion” cases and not included them in the PMI group. We apologize for the lack of clarity in the text. The text in *Methods* section has been edited as shown below to further emphasize this point:
“If there were any unrelated cause for biomarker elevation eg. acute stent thrombosis, no reflow, SB occlusion, < TIMI 3 flow in MV or SB, shock or hypotension in the immediate 24 hours post PCI, acute kidney injury, stroke, bleeding requiring transfusion, pulmonary embolism, access complication causing limb ischemia or sustained arrhythmia these cases were classified as not

having PMI. This was done to focus only on cases without a clear explanation for cause of PMI.”

2. We agree that a retrospective study should only be treated as “hypothesis generating” and apologize if the conclusion seemed over reaching. To clarify this, the following sentence has been added to the *Conclusion* section.

“Given the retrospective design, this finding should be treated as hypothesis generating and hopefully will trigger prospective analysis to confirm or refute this association.”