Effect of targeted temperature management on platelet function in patients with out-of-hospital cardiac arrest due to acute myocardial infarction

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Introduction: Targeted temperature management (TTM) is recommended in the European Resuscitation Council Guidelines for Post-resuscitation Care.¹ However, previous studies report controversial results on the effect of TTM on platelet function (PF) in patients on dual antiplatelet therapy (DAPT) with aspirin and P2Y12 inhibitors. *Hypothesis*: To assess if the effect of DAPT on platelet inhibition in patients undergoing TTM for out-of-hospital cardiac arrest (OHCA) is reduced compared to patients on the same therapy not undergoing this procedure.

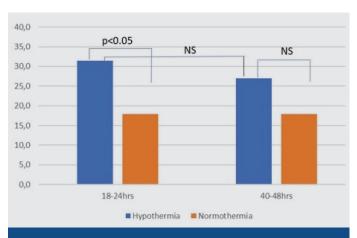


FIGURE 1. Platelet function in the interventional (hypothermia) and control (normothermia) group. NS = non-significant.

1.

Patients and Methods: Clinical characteristics and PF were analyzed in 56 consecutive patients admitted to the Department of Cardiology at the University Hospital Centre between January 2012 and January 2019 under the diagnosis of acute myocardial infarction (AMI). Out of these 56 patients, 17 underwent TTM to 34°C (*Intervention group*) while the other 39 did not (*Control group*). Both groups received the loading dose of DAPT. We analyzed residual PF using the Multiplate® PF analyzer ADP test. Platelet function tests (PFT) were performed 18-24 hours after therapy initiation in both groups and 18-24 hours after return to normal body temperature of 36.5° C in the TTM group. The control group had an average temperature of 36.5° C at the time of the PFT.

Results: There were 39 patients with a mean (SD) age of 63.2 (9.9) years in the Control group and 17 patients with a mean (SD) age of 58.6 (8.2) in the Interventional group. There were no significant differences in the baseline parameters between the groups (history of IHD, hyperlipidemia, diabetes mellitus, smoking, gender, prior medical therapy, current medical therapy). At the first time point (18-24 hours after initiating therapy) the intervention group had a significantly higher residual

PF when compared to the control group (31.5 U vs 17.9 U; p<0.05). This difference is no longer present 18-24 hours after return to normothermia (p=0.2) (**Figure 1**).

Conclusion: Our study shows that TTM decreases the effect of DAPT on PF in patients with AMI undergoing TTM for OHCA. This is in line with the findings published by Uminska *et al*² who showed that TTM severely decreases the bioavailability of P2Y12 inhibitors in this group of patients. These findings indicate that patients with AMI who experienced an OHCA undergoing TTM could have an impaired response to standard DAPT.

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