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The effect of DPP-4i, GLP-1RA, SGLT-2i and long-acting insulin on platelet function in patients with type 2 diabetes mellitus

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Background: Patients with type 2 diabetes mellitus (T2DM) are at higher risk for thrombotic events. Platelet function may be used to assess prothrombotic state in patients with cardiovascular disease.

Purpose: We aimed to investigate whether the administration of novel antidiabetic agents influence platelet function in TDM2 patients.

Patients and methods: We 60 enrolled consecutive patients with T2DM, on stable antidiabetic therapy, who did not achieve therapeutic targets. Subjects were assessed to receive an additional anti-diabetic agent; dipeptidyl peptidase-4 inhibitor (DPP4i, n=14), glucagon like peptide-1 receptor agonist (GLP1RA, n=24), sodium/glucose cotransporter-2 inhibitor (SGLT2i, n=22). Platelet reactivity was measured with PFA-200 collagen/epinephrine (c-EPI) and PFA-200 collagen/ADP (c-ADP) closure time. Glycosylated hemoglobin (HbA1c), c-EPI and c-ADP were assessed at baseline and 3 months after treatment intensification.

Results: There was no difference between the study groups regarding gender, age, hypertension, dyslipidemia, smoking, Hba1c and CADP or CEPI (p=NS for all) at baseline. All groups achieved better glycemic control in terms of HbA1c values between baseline and follow-up (for DPP4i: 7.4±0.2% vs 6.7±0.2%, for GLP1RA: 8.3±0.2% vs 6.9±0.1%, for SGLT2i: 7.5±0.1% vs 6.7±0.1% and for insulin 9.8±0.5% vs 7.7±0.4%, $p<0.001$ for all). After a 3 month-period, treatment intensification with these novel agents did not influence c-EPI and c-ADP values [155.4±6.64 sec vs 152.9±8.28 sec (p=0.678) and 106.6±4.30 sec vs 106.8±3.93 sec (p=0.955) respectively] in whole population. In subgroup analysis, for patients off antiplatelet treatment (n=31), c-EPI was significantly decreased from 148.4±8.5 to 129.8±13.9 sec (p=0.036), but not c-ADP (from 105.4±5.3 to 99.3±4.9 sec, p=0.094). In patients who did receive antiplatelets (n=37), c-EPI and c-ADP were not significantly changed (c-EPI 163.1±10.9 to 179.6±13.9 sec p=0.201 and c-ADP from 106.6±8.2 sec to 114.6±7.3 sec, p=0.318) respectively.

Conclusion: Antiplatelet treatment prevents thrombotic risk in T2DM patients receiving novel antidiabetics. The effects of novel antidiabetics on platelet reactivity -as well as any distinct class properties- merits further investigation.