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Changes in platelet reactivity during TAVI

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THEME: Interventions for Valvular Disease
TOPIC(S): TAVI

AIMS
Although, recent studies described changes in platelet reactivity in days following transcatheter aortic valve implantation (TAVI), precise time course of these changes has not been investigated.

METHODS AND RESULTS
26 consecutive patients with severe and symptomatic aortic stenosis, undergoing TAVI procedure in our institution were enrolled in research. Patients? perioperative clinical characteristics were collected from medical records. All patients who did not have chronic therapy with aspirin and clopidogrel, received loading dose of aspirin (300 mg) and clopidogrel (300 mg) one day before the procedure, followed by their standard maintenance doses (aspirin 100 mg, clopidogrel 75 mg) for the next three months. Patients? platelet reactivity was measured in five time points: just before induction of anesthesia (T1), after heparin administration (T2), 10 minutes after valve implantation (T3), 10 minutes after additional interventional procedures (if necessary) or 20 minutes after valve implantation (T4) and three days after the procedure (T5). Platelet reactivity was measured using impedance aggregometer (Multiplate® analyzer, Roche, Munich, Germany). It was measured in response to three platelet aggregation agonists: arachidonic acid (ASPItest), ADP (ADPtest) and thrombin receptor activating peptide-6 (TRAPtest). Results are presented in Units (U), which is calculated from the height and slope of aggregation curve and represents best overall measure of platelet reactivity. During TAVI procedure, all patients received unfractionated heparin at does 50-70 IU/kg with a target activated clotting time of 250-300 seconds.Mean patient age was 82.3 years with majority patients being male 57.5% (N=15). Mean valve area prior to procedure was 0.71±0.19 and mean transvalvular gradient 45.35±11.14 mmHg. All patients underwent successful TAVI procedure using either self-expandable (N=14, 53.8 %) or balloon-expandable valve (N=12, 46.2 %). Balloon dilatation following initial implantation was performed in 9 cases (34.6 %). Mean post-implantation gradient was 11.75±7.6 mmHg. Significant PVR, defined as more then mild, developed in 7 cases (26.9 %). Mean platelet reactivity before TAVI (T1) was 24.3±22.6 for ASPItest, 47.2±26.7 for ADPtest and 93.1±31.4 for TRAPtest. There was no significant difference between basal measurements and after administering unfractionated heparin (T2). However, 10 minutes after valve implantation (T3), significant reduction in platelet reactivity was observed in all three tests, ASPI 11.6±12.1 (p=0.018), ADP 27.7±16.6 (p<0.001) and TRAP 75.5±26.4 (p=0.016). Subsequent measurements (T4, T5), demonstrated further decline in platelet reactivity, although non-significant compared to T3 (T5: ASPI 8.9±9.5, ADP 15.8±8.5 and TRAP 63.3±21.8).

CONCLUSIONS
Our results show that successful TAVI procedure induces decrease in platelet reactivity regardless of the platelet activation pathway. Decrease was observed 10 minutes after valve implantation and was sustained in the short postoperative period. Further research on a larger number of patients is needed to confirm these results.