Differential Expression of Surface Markers on Activated Platelets in Thyroid Cancer

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Introduction: Platelets (PLTs) are emerging as important non-invasive biomarkers in oncology. Tumor cells modulate platelet activation and aggregation through soluble agonists or direct interaction. Upon activation by agonists like ADP and thrombin, surface expression of CD62p (P-selectin) and CD63 increases, indicating α-granule release. Thrombin receptor-activating peptide (TRAP), acting via protease-activated receptors (PARs), strongly induces CD62p expression, marking a key step in platelet activation. This study examines platelet activation in thyroid cancer (Thy-Ca) patients and healthy donors (HDs), in relation to receptor modification status.

Method: Platelets were isolated from 10 ml of whole blood (n=13; 4 HDs, 9 Thy-Ca) via differential centrifugation and re-suspended in HEPES-Tyrode buffer. The population was split into active and inactive groups, with activation induced by ADP, TRAP, or thrombin for 20 min at room temperature. CD62p (FITC) and CD63 (BV711) expression were assessed using a BD FACS-Celesta flow cytometer (FC) and analysed with FlowJo software (v10.9). Gating based on FSC-A and SSC-A parameters was applied to isolate intact platelets by size and granularity.

Results: FC analysis revealed higher CD62p and CD63 expression in Thy-Ca PLTs at 32.4±0.79% and 3052.11±0.51%, respectively, compared to HD-PLTs at 21.7±0.32% and 2638.58±151.33%. ADP stimulation caused no significant change in HD-PLTs (CD62p: 24.70±0.32%; CD63: 2555±81.6%) but significantly increased expression ($P<0.05$) in Thy-Ca PLTs (CD62p: 56.7±0.86%; CD63: 3114±49%). TRAP stimulation elevated CD62p and CD63 in HD-PLTs to 50.6±0.57% and 2712±18, while Thy-Ca PLTs showed further increases to 81.54±1.06% and 3226.81±48% ($P<0.05$).

Conclusion: Our preliminary findings demonstrate significantly higher CD62p and CD63 expression in Thy-Ca platelets both at baseline and following stimulation with ADP and TRAP ($P<0.05$). However, further studies with larger sample size are required to evaluate platelet activation as a potential marker for cancer therapy.