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PB1832 INFLAMMATION INDUCED COAGULATION IN ACUTE MYELOID LEUKEMIA

Topic: 3. Acute myeloid leukemia - Biology & Translational Research

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Background:

Patients with acute myeloid leukemia (AML) have an increased risk of thrombotic complications in the range of 4.2 - 5.2%.

Aims:

Our hypothesis is that inflammation is responsible for deterioration of coagulation in AML.

Methods:

Quantification of neutrophil extracellular traps (NETs) from peripheral blood of patients with AML by measurement of circulating cell-free DNA (cfDNA) and myeloperoxidase (MPO) activity. Inflammatory cytokines, coagulation factors and chemokines are measured by enzyme-linked immunosorbent assay (ELISA) and flow cytometry in peripheral blood, while fibrinolytic activity with fluorescent tissue-type plasminogen activator (tPA) and urokinase plasminogen activator (uPA) assays.

Results:

The pro-inflammatory cytokines IL-1β and TNF-α were significantly increased in AML, but not the chemokines IL-8 and MCP-1. NETs were increased in the peripheral blood of patients with AML ($p<0.05$) as measured by cfDNA and MPO activity. Regarding coagulation, factor VIII ($p<0.05$) and adhesion molecule P-selectin ($p<0.001$) were increased in plasma. Fibrinolytic activity was 3-fold decreased in the plasma of patients with AML ($p<0.01$) as measured by tPA. In contrast, uPA levels were increased in patients with AML ($p<0.05$). Tissue factor ($CD142^+$) inflammatory microparticles derived from monocytes ($CD14^+$: 5.1±0.6, $p<0.001$), activated monocytes ($CD14^+/CD16^+$: 2.89±0.4%, $p<0.05$) and circulating endothelial cells ($CD31^+/CD144^+$: 4.08±0.5%, $p<0.05$) were increased in AML compared to healthy controls.

Summary/Conclusion:

Chronic inflammation is present in AML in parallel with reduced fibrinolysis and increased coagulation provoking the risk of thrombosis. A panel of the applied inflammatory / procoagulant biomarkers can be used as a predictor of thrombosis in AML.