

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-1 β 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⁺: $2.89 \pm 0.4\%$, $p < 0.05$) and circulating endothelial cells (CD31⁺/CD144⁺: $4.08 \pm 0.5\%$, $p < 0.05$) 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.

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