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Megakaryocytic Emperipolesis in Acute Myeloid Leukemia with Trisomy 8

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A 29-year-old man with myelodysplastic syndromes diagnosed 3 years prior progressed to acute myeloid leukemia following only intermittent red blood cell transfusions and testosterone undecanoate as supportive therapy. Blood tests showed leukocytes 3.3 × 10^9/L, hemoglobin 50 g/L, platelets 3 × 10^9/L and elevated IL-12p70. Bone marrow aspirate showed hypercellularity containing 37.0% blasts positive for CD117, CD34, CD13, CD33, CD38 and HLA-DR, eosinophilia, trilineage dysplasia. Megakaryocytes were normocellular but exhibited predominant granular megakaryocytes, lacking platelet-producing forms, and significant emperipolesis (non-destructive intracytoplasmic migration of diverse hematopoietic cells) in 80% megakaryocytes – a finding absent in initial MDS marrow smears. Cytogenetics revealed 47,XY,+8[20] along with *GATA2* and *U2AF1* mutations and *CFHR3* partial deletion, consistent with initial MDS diagnosis.

Trisomy 8 is one of most frequent cytogenetic abnormality in MDS/AML, often associated with dyshematopoiesis and enhanced inflammatory responses [1,2]. Increased IL-12p70, a proinflammatory cytokine involved in immune regulation and hematopoietic cell interactions, may contribute to the observed emperipolesis. It is plausible that trisomy 8-related genomic instability enhances inflammatory events, upregulating cytokines IL-12p70, which in turn enhances cell adhesion molecules on megakaryocytes promoting emperipolesis by altering megakaryocyte-guest cell interactions—consistent with reports linking proinflammatory cytokines to abnormal cell-in-cell phenomena in hematological malignancies [3-6].

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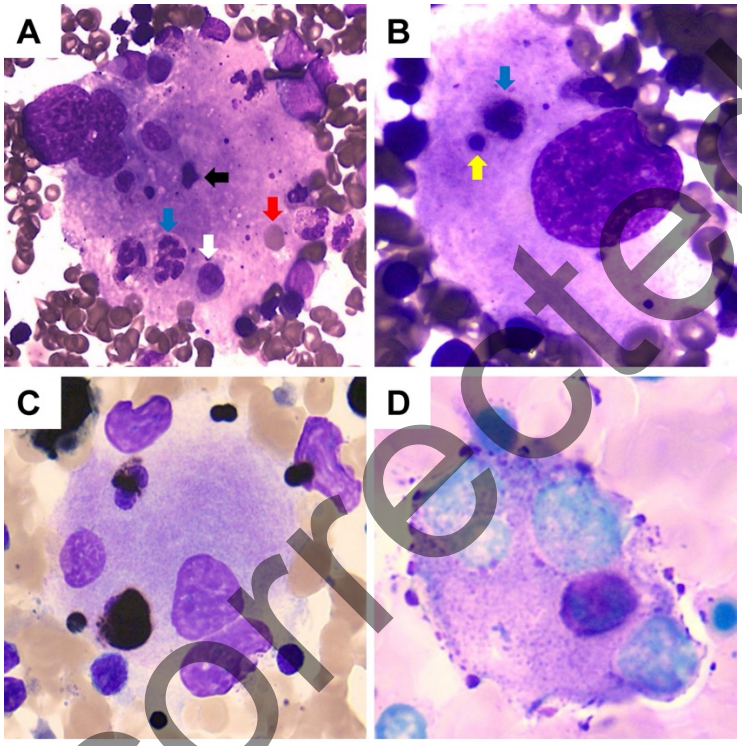

A-B: Wright–Giemsa stain showing megakaryocytes with emperipolesis (blue arrow: granulocytes; white arrow: monocytes; black arrow: lymphocytes; yellow arrow: nucleated red cells; red arrow: erythrocytes); C: POX stain; D: PAS stain.