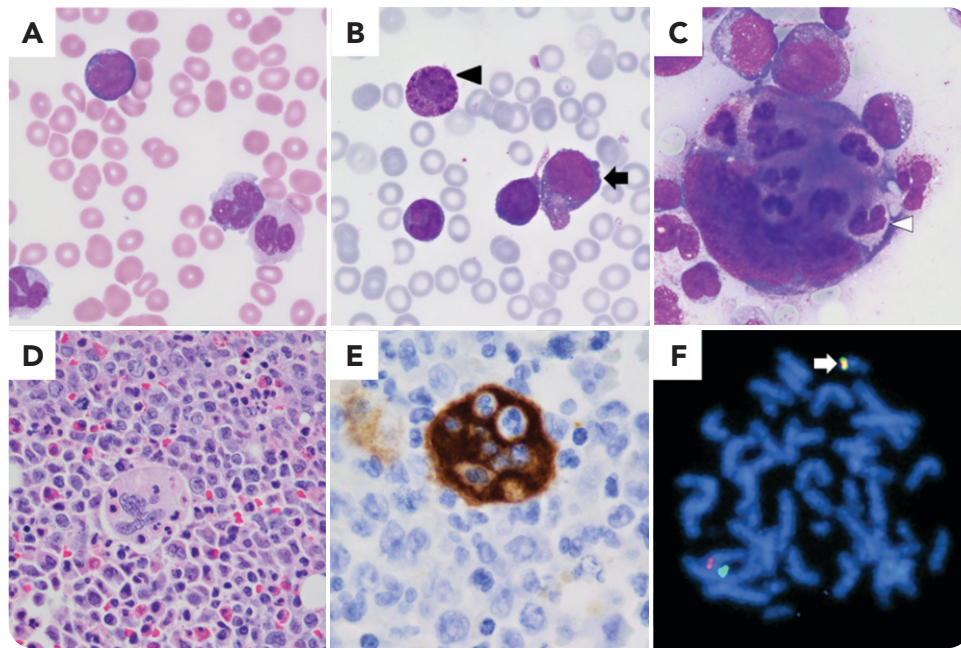


Megakaryocytic emperipolesis as a dyshematopoietic feature in acute myeloid leukemia with *inv(16)*

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A previously healthy 64-year-old woman presented with a 2-week history of bruising, fatigue, and weight loss. Blood work showed circulating blasts, dysplastic neutrophils, and abnormal monocytes (panel A; Wright-Giemsa, 100× objective), and a bone marrow examination was performed. Bone marrow aspirate showed 28% blasts (panel B; black arrow, May-Grünwald-Giemsa [MGG], ×100 original magnification) and dysplastic eosinophil precursors with large metachromatic granules (black arrowhead). Virtually all megakaryocytes in aspirate (panel C; MGG, ×100 original magnification) and biopsy (panel D; hematoxylin and eosin, 50×; panel E; CD61 immunohistochemistry, ×100 original magnification) showed significant emperipolesis, near exclusively of mature neutrophils (panel C; white arrowhead). Karyotyping and metaphase fluorescence in situ hybridization showed evidence of *CBFB-MYH11* fusion (panel F; ×100 original magnification, dual fusion probe; green signal, *MYH11*; red signal, *CBFB*; white

arrow, fusion signal for *CBFB-MYH11*), confirming the diagnosis of acute myeloid leukemia (AML) with *inv(16)(p13.1q22)*.

Emperipolesis refers to the presence of cells in the cytoplasm of another cell without evidence of destruction. Megakaryocytic emperipolesis has been described in benign and malignant hematological pathologies, including cases of myelodysplastic syndrome. This case showed neutrophil dysplasia with morphologically abnormal monocytes and extreme megakaryocytic emperipolesis involving neutrophils. This phenomenon is thought to occur through increased cell-surface P-selectin expression, enhancing neutrophil-megakaryocyte interaction and promoting sequestration. Significant megakaryocyte emperipolesis has not been previously reported in AML with *inv(16)*, likely reflects underlying dyshematopoiesis, and has uncertain functional consequences for affected megakaryocytes.