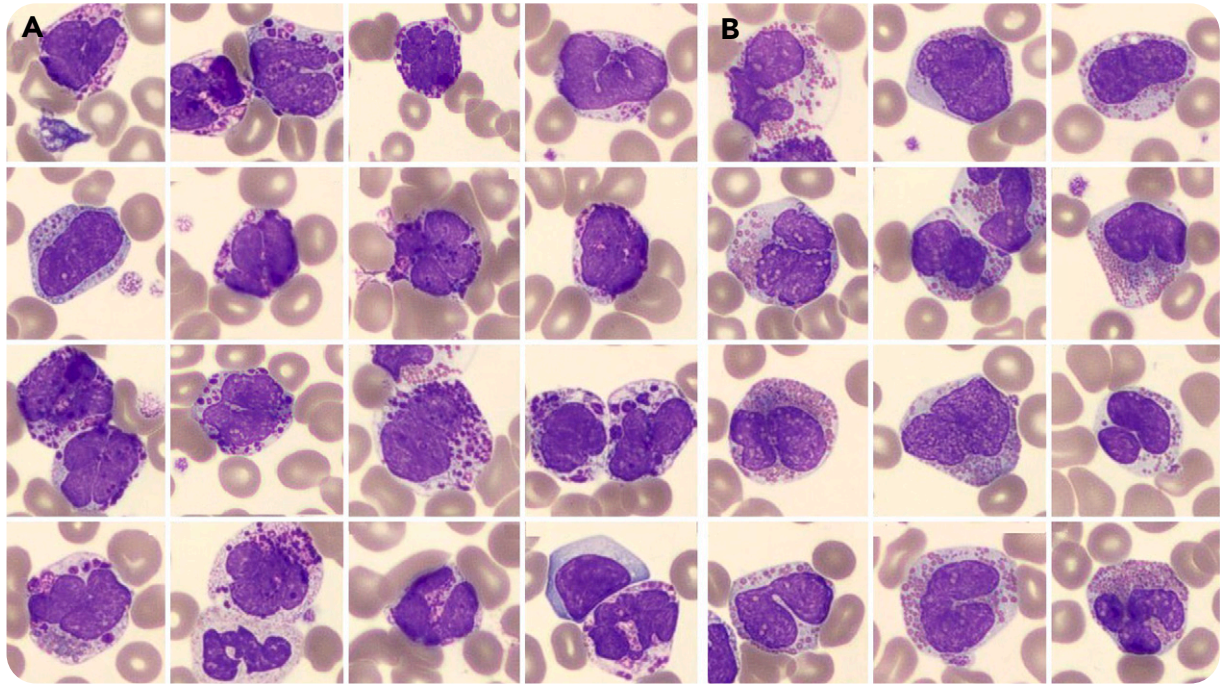


Atypical basophilia

Henk-Jan Boiten and Eva de Jongh, Albert Schweitzer Hospital



A 35-year-old man with a history of ankylosing spondylitis for which he received adalimumab was referred to our clinic because of marked leukocytosis detected from a routine blood test. He experienced occasional night sweats. He was found to have splenomegaly. The complete blood count showed normocytic anemia (13.37 g/dL, 90 fL), normal platelets ($315 \times 10^9/L$), and leukocytosis ($32.8 \times 10^9/L$) with basophilia (51%), eosinophilia (20%), promyelocytes (1%), and blasts (1%). Peripheral blood smear revealed prominent basophilia and atypical eosinophilia (panel A, atypical basophilic granulocytes; panel B, atypical eosinophilic granulocytes, with clearly visible cytoplasm, prominent nuclei, and an increased nuclear-cytoplasmic ratio; original magnification $\times 100$, May-Grünwald Giemsa stain).

Immunophenotyping showed a cell population negative for CD117, CD2, and CD25. Bone marrow biopsy showed increased cellularity with $<10\%$ myeloblasts as well as marked reticulin fibrosis. *BCR-ABL1* fusion was negative. Conventional cytogenetics with gene-specific probes showed an extremely rare $t(9;12)(q34;p13)$ resulting in the *ETV6-ABL1* fusion. No additional cytogenetic abnormalities were observed, including no *PDGFRa* rearrangements.

The *ETV6-ABL1* fusion induces a chronic myeloid leukemia-like disease with a more aggressive behavior and prominent basophilia. The patient received dasatinib with complete molecular response followed by an allogeneic transplantation.