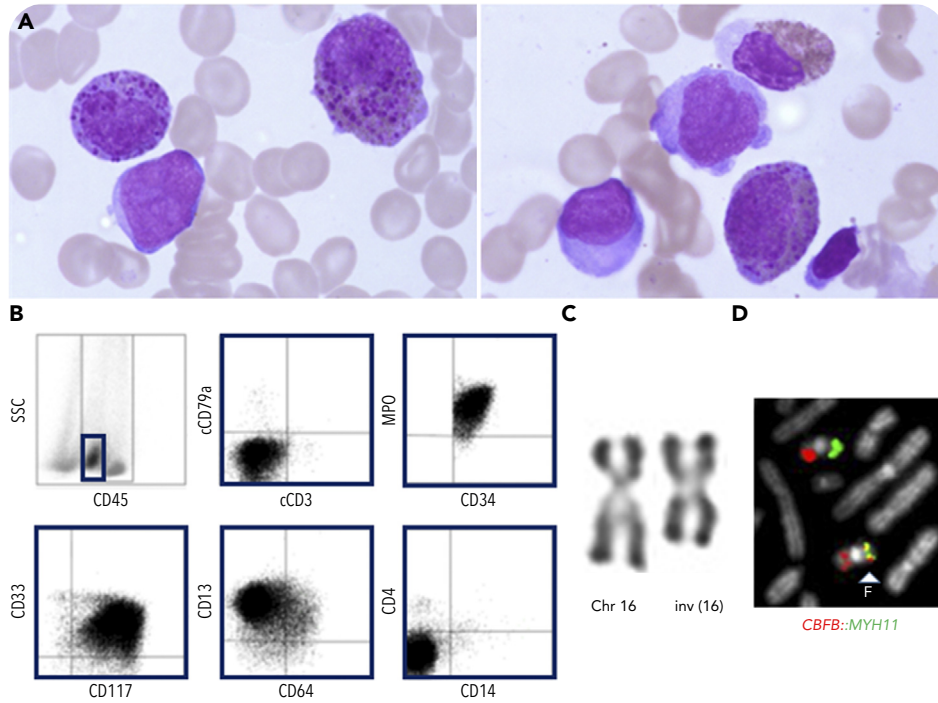


CBFB::MYH11 fusion in a nonmonocytic acute myeloid leukemia

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A 56-year-old woman was admitted to our hospital for anemia (hemoglobin, 108 g/L) and leukopenia ($1.98 \times 10^9/L$) with 10% blasts. Bone marrow aspirate (May-Grünwald-Giemsa stained) revealed 36% blasts and 17% abnormal eosinophils with large coarse basophilic granules (panel A; 100 \times objective; original magnification $\times 1000$), suggesting an acute myeloid leukemia (AML) with *CBFB::MYH11* fusion according to the fifth World Health Organization classification. At the morphologic level, this AML subtype is classically characterized by myelomonocytic differentiation and presence of abnormal eosinophils in bone marrow. In our case, the monocytic population was absent, as confirmed by flow cytometry immunophenotyping, which revealed presence of myeloid blasts (MPO⁺CD33⁺CD13⁺CD34⁺CD117⁺) without monocytic contingent (panel B). Karyotype (R banding) showed the characteristic inversion

of chromosome 16: inv(16)(p13.1q22) in 19 of 20 metaphases (panel C; 63 \times objective; original magnification $\times 630$). Fluorescence in situ hybridization with a *CBFB::MYH11* double-fusion probe (MetaSystems, Altlußheim, Germany) showed a *CBFB::MYH11* fusion signal in 10 of 10 metaphases and highlighted existence of a partial deletion of *MYH11* (panel D; 63 \times objective; original magnification $\times 630$). This fusion was confirmed by reverse transcription polymerase chain reaction.

To our knowledge, cases of nonmonocytic AML with *CBFB::MYH11* fusion remain exceptional. Our case highlights the necessity to search for a *CBFB::MYH11* fusion in all new AML cases so as not to miss those harboring atypical presentation. Indeed, this AML subtype is associated with a favorable prognosis and can benefit from a tailored therapy.