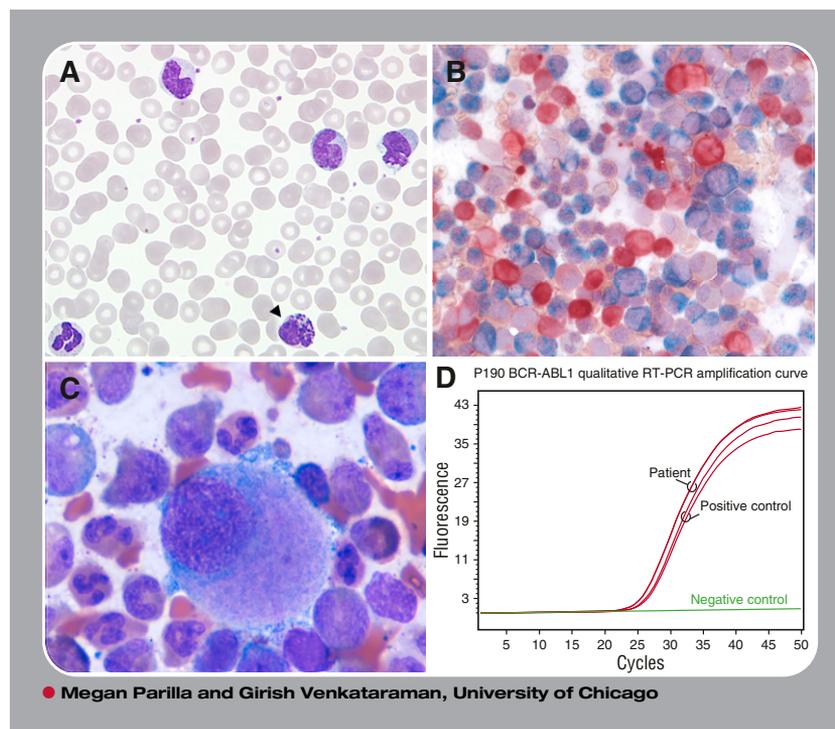


The thin line between CML and CMML



A 57-year-old woman with autosomal dominant polycystic kidney disease, after liver/kidney transplant 2 years ago, presented with complaint of isolated left abdominal discomfort. A complete blood count demonstrated isolated leukocytosis (40 200/ μ L) with normal platelet count (234 000/ μ L) and hemoglobin. A peripheral blood smear demonstrated absolute monocytosis (6400/ μ L), neutrophils without significant dysplasia, and rare basophils (panel A, arrowhead indicates basophil; original magnification $\times 200$, Giemsa stain) without circulating blasts. A bone marrow biopsy was hypercellular with marked granulocytic expansion comprising predominantly mature neutrophils and normal megakaryocytes with rare immature small megakaryocytes on aspirate (panel C; original magnification $\times 400$, Giemsa stain). Combined esterase cytochemical stain shows marked increase in marrow monocytes (panel B, brown; original magnification $\times 200$) with background granulocytic cells (panel B, blue). A concurrently performed peripheral blood *BCR-ABL* molecular assay (testing simultaneously for both P210 and P190 transcripts) detected the P190 fusion variant (panel D) without P210, whereas conventional karyotype showed the classic t(9;22)(q34.1;q11.2).

The chronic myeloid leukemia (CML)-P190 variant often resembles chronic myelomonocytic leukemia (CMML) due to the associated marked monocytosis. However, the lack of granulocytic dysplasia and thrombocytopenia is unusual in CMML. Another rare CML variant with the longer P230 transcript phenotypically presents with neutrophilia and thrombocytosis. In the absence of *BCR-ABL* fusion, some CMMLs with normal-high platelets counts carry concurrent *JAK2* mutations. This case highlights the need to exclude underlying *BCR-ABL* rearrangements before making a diagnosis of CMML.



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