Acute myeloid leukemia pretreated with filgastrim mimicking acute promyelocytic leukemia

A 66-year-old man with a history of hepatitis C virus and liver transplantation presented to the emergency department for shortness of breath. He had received weekly filgrastim for chronic neutropenia, and immediately before admission he had received 3 days of filgrastim and 1 day of pegfilgrastim. A hemogram showed a white blood cell count of 4.54 K/μL with 23% blastic cells. Emergent bone marrow specimen revealed 50% blasts and 43% promyelocytes with Auer rods (panel A). Flow cytometry demonstrated a predominant population expressing myeloid antigens, CD38, CD117, and dim CD7, but not HLA-DR or CD34. These features suggested acute promyelocytic leukemia (APL) and treatment with all-trans-retinoic acid (ATRA) was initiated. Subsequent fluorescence in situ hybridization testing for promyelocytic leukemia (PML)/retinoic acid receptor alpha (RARα) with dual-fusion and RARα break-apart probes was negative (panel B). Molecular testing for PML/RARα messenger RNA by reverse-transcriptase polymerase chain reaction was also negative, but FLT3-ITD was positive. Despite morphologic and immunophenotypic evidence for APL, confirmatory cytogenetic and molecular tests were negative; therefore, acute myeloid leukemia—not otherwise specified was the final diagnosis.

Marrow specimens obtained after exposure to granulocyte colony-stimulating factor preparations should be interpreted cautiously because the features of AML may be altered, allowing confounding overlap with APL. This distinction must be made to avoid unnecessary risk of toxicity—although it is low—as a result of ATRA use in non-APL patients.
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