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Case report

Therapy-related CD7$^+$ acute myeloid leukemia with trisomy 8 following acute monocytic leukemia

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We report a patient who developed CD7$^+$ therapy-related acute myeloid leukemia (t-AML) with trisomy 8 after chemotherapy for AML. [© 2001 Lippincott Williams & Wilkins.]

Key words: Acute myeloid leukemia, CD7, trisomy 8.

Recently, therapy-related acute myeloid leukemia (t-AML) has been described with an increased frequency following primary malignancies.[1] We report a patient who developed CD7$^+$ t-AML with trisomy 8 after chemotherapy for AML.

A 45-year-old female presented with gingival bleeding, general fatigue and palpitation in May 1993. The white blood cell count was 58.5×10^9/l with 9% of blast cells. A bone marrow (BM) aspirate revealed hypercellular marrow with 86.8% of blast cells. The blast cells were not stained with myeloperoxidase and periodic acid Schiff, but were positive with α-naphthyl butylate esterase. The diagnosis of AML-M5b was made according to FAB criteria. Flow cytometric immunophenotypic analysis revealed the blast cells were positive for CD4, CD14, CD33 and HLA-DR, antigens and chromosomal analysis on BM showed a normal female karyotype. She was treated with chemotherapy consisting of behenoyl Ara-C (BH-AC), daunorubicin (DNR), 6-mercaptopurine (6-MP), and prednisolone, and achieved a complete remission. Four courses of consolidation chemotherapy, the same as induction therapy, were carried out and there was no evidence of residual disease. The patient received a total cumulative dose of 7000 mg/m^2 BH-AC, 750 mg/m^2 DNR and 2450 mg/m^2 of 6-MP. Four years later, her peripheral blood examination showed leukocytopenia and thrombocytopenia. BM aspirate revealed hypocellular marrow with 13.0% of blasts that were positive for myeloperoxidase and negative for α-naphthyl butylate esterase. There was no trilineage dysplasia. Chromosomal analysis on BM showed 47,XX, +8 in three of 20 cells and 46,XX in 17 of 20. The blast cells had CD7, CD13, CD33 and HLA-DR antigens. In Southern blot analysis, gene rearrangement study for MLL was in the germline configuration. She was diagnosed with therapy-related hypoplastic myeloid leukemia and treated with low-dose Ara-C. Three months later, the blast cells of peripheral blood increased to 85.0%. Although additional chemotherapy was given, she died because of pneumonia 4 months after diagnosis of t-AML.

In this case, the second leukemia was apparently different from the first in that blast cells had a myeloid feature, trisomy 8 and CD7 antigen. t-AML after treatment for initial AML is extremely rare, but has been reported.[2,3] Cytogenetic abnormalities may play a pathogenic role in the development of therapy-related myelodysplastic syndromes (MDS) and/or leukemias. In particular, trisomy 8 is frequently observed in primary MDS and *de novo* AML. Although this cytogenetic abnormality is not statistically associated with t-AML or t-MDS,[4] it may cause clonal evolution to induce secondary leukemia. Another interesting finding in this case is that the blast cells of t-AML expressed CD7 antigen. It has been reported that CD7 is one of the 'immature' markers and CD7 expression is associated with poor prognosis in AML patients.[5] These karyotypic and immunophenotypic characteristics of blast cells may affect the clinical outcome in this case.

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