

Case Report Section

Paper co-edited with the European LeukemiaNet

A de novo AML with a t(1;21)(p36;q22) in an elderly patient

Paola Dal Cin, Andrew J Yee, Bimalangshu Dey

Department of Pathology, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, USA (PDC); Hematology/Oncology Unit, Massachusetts General Hospital, Boston, MA, USA (AJY, BD)

Published in Atlas Database: March 2007

Online updated version: <http://AtlasGeneticsOncology.org/Reports/0121DalCinID100021.html>
DOI: 10.4267/2042/38459

This work is licensed under a Creative Commons Attribution-Non-commercial-No Derivative Works 2.0 France Licence.
© 2007 Atlas of Genetics and Cytogenetics in Oncology and Haematology

Clinics

Age and sex: 81 years old male patient.
Previous History : no preleukemia ; no inborn condition of note.
Organomegaly : no hepatomegaly ; no splenomegaly ; enlarged lymph nodes ; no central nervous system involvement.

Blood

WBC: $3.3 \times 10^9/l$; Hb: N/A g/dl; platelets: $16 \times 10^9/l$; blasts: 2% (CD34+ myeloblasts).
Bone marrow: 20% myeloid precursors, 16% erythroid precursor, 6% lymphocytes, 55% blasts and 2% plasma cells.

Cytopathology classification

Cytology: AML M0
Immunophenotype: CD33+, CD13+, MPO-, CD41-, CD61-, CD203c- (5% of all blast).
Rearranged Ig or Tcr: N/A
Precise diagnosis: Immunophenotype consistent with the presence of myeloid precursors. Negative markers (CD61, CD41, CD203c) associated with megakaryocytic differentiation; AML M0.

Survival

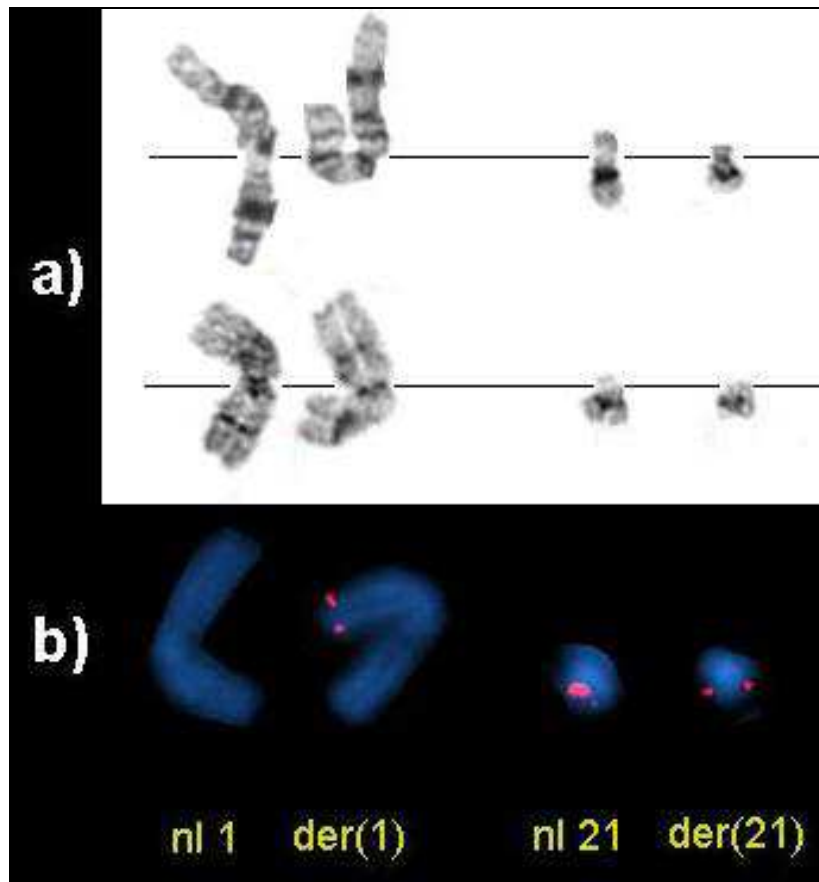
Date of diagnosis: 01-2005.
Treatment: Hydroxyurea and supportive care.
Complete remission: None
Treatment related death: -
Relapse: Patient never achieved complete remission.
Status: Dead 02-2005.
Survival: 1 months.

Karyotype

Sample: Bone marrow; Culture time: 24h; Banding: GTG.
Results: 46,XY,t(1;21)(p36;q22)[15]
Other molecular cytogenetic technics: FISH with LSI (TEL/AML1 ES Dual Color Translocation Probe (Vysis, Inc.) on metaphases (see Fig 2).
Other molecular cytogenetics results: Ish der(1)(dimAML1+), der(21)(dimAML1+).

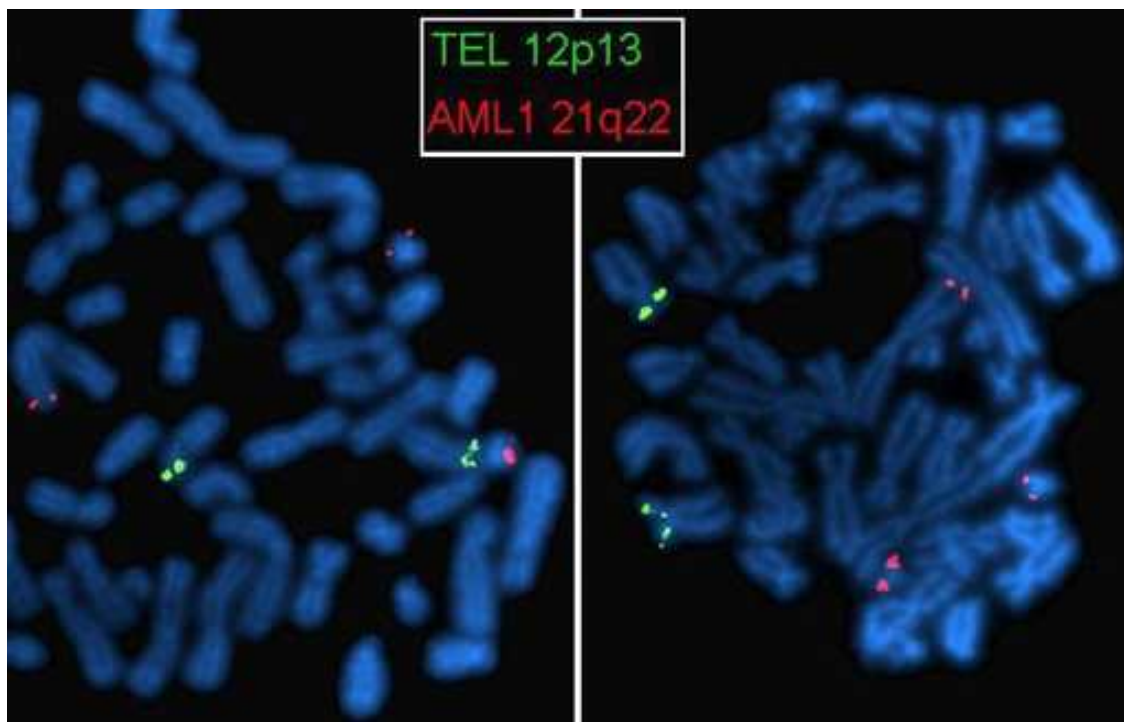
Comments

The t(1;21)(p36;q22) so far reported, is generally observed as the sole chromosomal abnormality (5/6), and is mostly a de novo aberration (4/6). The short survival (one month) of our case, confirms the poor prognosis in these patients carrying this chromosome abnormality.



Partial GTG-banding karyotype showing t(1;21)(p36;q22)) (a)

Partial FISH analysis showing the AML1 hybridization signals on derivative chromosomes 1 and 21, and on the normal chromosome 21 (b)



References

Stevens-Kroef MJ, Schoenmakers EF, van Kraaij M, Huys E, Vermeulen S, van der Reijden B, van Kessel AG. Identification of truncated RUNX1 and RUNX1-PRDM16 fusion transcripts in a case of t(1;21)(p36;q22)-positive therapy-related AML. *Leukemia* 2006;20:1187-1189.

Marian Stevens-Kroef. t(1;21)(p36;q22) - updated. *Atlas Genet Cytogenet Oncol Haematol* 2006;10(3).

Preiss BS, Kerndrup GB, Pedersen RK, Hasle H, Pallisgaard N; Lymphoma-Leukemia Study Group of the Region of

Southern Denmark. Contribution of multiparameter genetic analysis to the detection of genetic alterations in hematologic neoplasia. An evaluation of combining G-band analysis, spectral karyotyping, and multiplex reverse-transcription polymerase chain reaction (multiplex RT-PCR). *Cancer Genet Cytogenet* 2006;165:1-8.

This article should be referenced as such:

Dal Cin P, Yee AJ, Dey B. A de novo AML with a t(1;21)(p36;q22) in an elderly patient. *Atlas Genet Cytogenet Oncol Haematol*.2007;11(3):261-263.
