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

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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 x 10^9/l; Hb: N/A g/dl; platelets: 16x 10^9/l; blasts: 2% (CD34+ myeloblasts).
Bone marrow: 20% myeloid precursors, 16% erythroid precursor, 6% lymphocytes, 55% blasts and 2% plama 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.
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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)
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References


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