Translocation t(2;19)(p11;p12-p13) in childhood with acute myeloid leukemia

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Clinics

Age and sex
11 years old female patient.

Previous history
Preleukemia No preleukemia. Previous malignancy No previous solid tumors.

Organomegaly
No hepatomegaly, no splenomegaly, no enlarged lymph nodes, no central nervous system involvement

Blood

WBC: 194X 10^9/l
HB: 8.6g/dl
Platelets: 17X 10^9/l
Blasts: 66%

Bone marrow: Increased cellularity, no megakaryocytes. Blasts: 63% with Auer rods; promyelocyte: 0%; myelocytes: 5%; metamyelocytes: 6%; neutrophils: 21%; erythropoiesis: 4%; lymphocytes: 1%; monocytes: 0%. Granulopoiesis with myelodysplastic features.

Immunophenotype
Blasts were myeloperoxidase positive, butyrate acetate negative expressing CD 34, CD 33, CD 13, CD 64, CD 7 and HLA DR.

Diagnosis
Diagnosis of acute myeloid leukemia with multilineage dysplasia (WHO classification) subtype LAM- M2 (FAB classification).

Survival

Date of diagnosis: 10-2002
Treatment: Induction treatment: Aracytine: 200 mg/m²/j x 7 days, Novantrone: 12 mg/m²/j x 5 days.
Complete remission: After induction (J30)
Relapse: no
Status: Alive
Survival: 7 +months

Karyotype

Sample: Blood and bone marrow.
Culture time: Overnight unstimulated culture and 72h stimulated culture with mitogen.

Bandung: R-banding

Results:
46, XX, t(2;19)(p11;p12-13) [30] / 46, XX [10]

Other molecular cytogenetics technics:
Fluorescence in situ hybridization (FISH) was performed using a chromosome 2-specific labelled FITC and a chromosome 19-specific labelled Spectrum Orange painting probes (Adgenix, USA) according to the manufacturer’s instructions.
Other molecular cytogenetics results:
FISH confirmed the translocation t(2;19).

**Other Molecular Studies**

Results:
ETO / AML1: negative
No rearrangement of MLL gene.

![Karyotype (R-bands): 46, XX, t(2;19)(p11;p12-13)](image)

**Note:**
Meningeal puncture without blast cells.

**Comments**

We report a translocation t(2;19)(p11;p12-13) occurring in a childhood acute myeloid leukemia, subtype M2. Cytological particularity of this case was the presence of myelodysplastic signs. Rearrangement of 19p13 is a common feature in preB-ALL showing t(1;19)(q23;p13) but not in AML. The t(1;19) involves in 19p13 E2A gene which normally encodes an immunoglobulin enhancer binding proteins. One case was reported showing t(2;19) in acute leukemia. The case was an AML secondary to chemotherapy for ovarian cancer. Karyotype showed complex abnormalities including a t(2;19)(p11;p13) [2]. Variability of the breakpoints at 2p and 19q in our patient, compared with the case described in literature, could be due to the fact that the authors used G banding techniques.

**Other Findings**

Blasts cells (MGG-stained) with Auer rod and degranulated neutrophil (myelodysplastic features) (x100).

Bone marrow (MGG-stained) (x50).
Fluorescence in situ hybridization using painting probes of chromosome 2 labelled FITC (WCP 2) and chromosome 19 labelled Spectrum Orange (WCP 19) (ADgenix, USA).

References


This article should be referenced as such: