An adult case of biphenotypic acute leukemia with t(6;14)(q25;q32)

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Clincis

Age and sex
66 years old male patient.

Previous history
no preleukemia
no previous malignancy
no inborn condition of note

Organomegaly
no hepatomegaly , splenomegaly , enlarged lymph nodes (Slightly enlarged lymph nodes (submental, cervical, axial, mediastinal, inguinal) were detected by computed tomography.), no central nervous system involvement

Blood
WBC : 130.4X 10^9/l
HB : 13.9g/dl
Platelets : 40X 10^9/l
Blasts : 93%
Bone marrow : Hypercellular marrow (NCC 497109/l) with 93.8% blast; monotonous and high nuclear-cytoplasm (N/C) ratio blast cells which had a cleaved nuclear were expanded.%

Cyto-Pathology
Classification

Phenotype
Mixed phenotype acute leukaemia, T/myeloid, NOS

Immunophenotype
Positive for CD2, cyCD3, CD7, CD13, CD15, CD34, HLA-DR, and dimly positive for CD33, MPO, TdT. Negative for CD1a, CD5, CD11b, CD117, TCR-AB, TCR-GD

Rearranged Ig Tcr
Not performed.

Pathology
Acute leukemia compatible.

Electron microscopy
Not performed.

Diagnosis
Mixed phenotype acute leukemia, T/myeloid, NOS.
Survival

Date of diagnosis
04-2015

Treatment
Japan adult leukemia study group T-ALL213-O induction therapy including vincristine (VCR), cyclophosphamide (CPA), daunorubicin (DNR), L-Asparaginase (L-ASP) and Predonisolone (PSL).

Treatment related death: no
Relapse: no
Status: Alive
Last follow up: 09-2015
Survival: 5 months

Karyotype

Sample: Bone marrow
Culture time: 24-48
Banding: G-banding
Results
46,XY,t(6;14)(q25;q32) [20]

Karyotype at Relapse
not applicable

Other molecular cytogenetics technics
fluorescence in situ hybridization (FISH) analysis using IgH 3' flanking region/V probes. 14q32 (IgH) break apart probe is a mixture of two probes, 3'IgH flanking probe and IgH variable region probe as shown in Figure 2a.

Other molecular cytogenetics results
Negative for immunoglobulin heavy chain (IgH) translocation (Figure 2b).

Other Molecular Studies

Technics:
Polymerase chain reaction (PCR) and Sanger sequencing

Results:
Positive for Flt3-internal tandem duplication (ITD).
Negative for c-kit mutation.

FISH analysis was performed by LSI Medience (Tokyo, Japan). (a) A scheme of 3' flanking probe and V probe which is modified from the technical information of LSI Medience corporation (Tokyo, Japan) web site. (b) Negative result for IgH gene rearrangement in this case.
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Comments

We present an adult case of biphenotypic acute leukemia with t(6;14)(q25;q32). Chromosome translocations involving 14q32 are generally represented by B cell neoplasms, because the immunoglobulin heavy chain (IgH) gene is located in this region. However, BCL11B gene also located in 14q32 was shown to be involved in this translocation (Bezrookove et al., 2004). BCL11B, a member of the Kruppel family of zinc finger transcription factors, plays a critical role in T cell development and functions as a tumor suppressor (Wakabayashi et al., 2003). The partner gene of this translocation is unknown. The 28S ribosomal DNA (RN28S1) was reported as a candidate fusion partner (Kobayashi et al., 2014), but this gene is not located in 6q25. The phenotype of haematological malignancies with t(6;14)(q25;q32) is variable. These include acute lymphoblastic leukemia (ALL) (Heerema et al., 1990, Batanian et al., 1996, Georgy et al., 2008, Kobayashi et al., 2014), acute myeloid leukemia (AML) (Raimondi et al., 1989, Bezrookove et al., 2004), chronic T cell neoplasm (Inwards et al., 1990) and chronic lymphocytic leukemia (CLL) (Mayr et al., 2006). In 7 of 9 acute leukemia cases with this translocation, both myeloid and T-cell lineage markers were detected. No immunophenotype was described in the remaining two cases. This translocation may affect expression of T-cell lineage marker, but the role of BCL11B is unclear.

References


This article should be referenced as such: