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Leukemia Section
Short Communication

t(7;14)(p15;q32)

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Abstract

Rearrangements of T-cell receptor (TCR) genes are characteristic chromosomal abnormalities in a variety of T-cell malignancies, particularly in T-cell lymphoblastic leukemia/lymphomas, but TCR-gamma gene or TCL1A (TCR-T-cell leukemia/lymphoma 1A) rearrangements are rare.

Keywords

T-cell lymphoblastic leukemia/lymphoma; t(7;14)(p15;q32); TCL1A; T- cell receptor genes.

Clinics and pathology

Disease

T-cell acute lymphoblastic leukemia/lymphoblastic lymphoma (T-ALL); rarely: acute myeloid leukemia (AML).

	Sex/Age	Diagnosis	Karyotype
3.	F	AML-M2	46,XX,t(7;14)(p15;q32)
10.	M/16	AML-M4	46,XY,t(7;14)(p15;q32)
12.	M/35	BAL	46,XY,add(5)(q13),t(6;16)(q15;q24),t(7;14)(p15;q32)/92,idemx2
1.	M/12	T-ALL	46,XY,-6,t(7;14)(p15;q32),del(8)(q22),del(12)(p12),-13,-16,-18,+4mar
2.	F/10	T-ALL	46,XX,t(6;12)(q21;p13),t(7;14)(p15;q32)
5.	F/4	T-ALL	45,XX,t(1;2)(p22;p14),t(7;14)(p15;q32),dic(12;18)(p11;p11)
6.	M/14	T-ALL	46, XX,t(7;14)(p15;q32)
8.	M	T-ALL	46,XY,?inv(5)(p13q14),del(6)(p21-22),t(7;14)(p15;q32)
9.	F	T-ALL	46,XX,del(5)(q31),del(6)(q15q23),t(7;14)(p15;q32)
11.	F/7	T-ALL	46,XX,del(5),t(7;14)(p15;q32)/45,idem,-8,der(12)t(8;12)(q11;p11), inv(17)(p13q11)
13.	M/55	T-ALL	46,XY,t(7;14)(p15;q32) human immunodeficiency virus infection
15.	M/22	T-ALL	46,XY,t(7;14)(p15;q32) **TRG/TCL1A**
4.	M/14	T-cell lymphoma	t(7;14)(p15;q32)
7.	M/9	T-cell lymphoma	46,XY,t(7;14)(p15;q32)
14.	F/15	MT/NKN	46,XX,t(7;14)(p15;q32)/92,idemx2

Table 1. Clinical findings of patients with t(7;14)(p15;q32). **Abbreviations**: AML-M2, Acute myeloblastic leukemia with maturation; AML-M4, Acute myelomonocytic leukemia; BAL, Bilineage or biphenotypic leukemia; T-ALL, T-Acute lymphoblastic leukemia/lymphoblastic lymphoma; MT/NKN, Mature T- and NK-cell neoplasm.
1-2. Kaneko et al., 1989; 3. Raimondi et al., 1989; 4. Teramura et al., 1989; 5-6. Pui et al., 1991; 7. Toyoda et al., 1991; 8-9. Heerema et al., 1998; 10.Strehl et al., 2001; 11. Chang et al., 2006; 12. Xu et al., 2009; 13. Gill et al., 2011; 14. Narayan et al., 2013; 15. Sugimoto et al., 2014.

Phenotype/cell stem origin

An immature thymocytic pattern: CD7-positive, surface CD3-negative and CD4/CD8- negative.

Etiology

T-cell acute lymphoblastic leukemia/lymphoblastic lymphoma in 11 (Kaneko et al., 1989; Teramura et

al., 1989; Pui et al., 1991; Toyoda et al., 1991; Heerema et al., 1998; Chang et al., 2006; Gill et al., 2011; Sugimoto et al., 2014), among them 1 with human immunodeficiency virus infection (Gill et al., 2011). Additional cases included: 1 mature T- and NK-cell neoplasm (Narayan et al., 2013), 2 AML (Raimondi et al., 1989; Strehl et al., 2001) and 1 biphenotypic leukemia (BAL) (Xu et al., 2009).

Epidemiology

15 patients (9 males and 6 females) aged 4 to 55 years (median 14 years); mainly pediatric cases (12 out of 15 patients; aged 4 to 16 years).

Prognosis

6 of the described patients achieved clinical remission after therapy and had a favorable outcome with survival of 1 to 24+ months (Data from Sugimoto et al., 2014).

Cytogenetics

Note

Because precise breakpoints are often difficult to define, it is possible that the reported t(7;14)(p15;q32) involve various 7p14-7p15.1-p15.3 breakpoints and dispersion of breakpoints throughout the 14q32.1-32.3 region involving various genes.

Cytogenetics morphological

Sole anomaly in 6 T-Acute lymphoblastic leukemia/lymphoblastic lymphoma (Teramura et al., 1989; Pui et al., 1991; Toyoda et al., 1991; Gill et al., 2011; Narayan et al., 2013; Sugimoto et al., 2014) and in both AML patients (Raimondi et al., 1989; Strehl et al., 2001); found in association with 5q deletion in 1 (Heerema et al., 1998) and 12p anomalies in 4 (Kaneko et al., 1989; Pui et al., 1991; Chang et al., 2006).

Genes involved and proteins

TRG (T-cell receptor gamma)

Location

7p14.1

Note

Belongs to the family of T-cell receptor genes encoding T-cell receptor gamma chains, previously often described as located at band 7p14-p15. Note that the breakpoint has been described at 7p15 in a case with TRG+ (Sugimoto et al., 2014).

HOXA@ (homeo box A cluster)

Location

7p15.2

Protein

HOXA cluster genes at 7p15 act as transcription factors regulating gene expression, morphogenesis, and differentiation. Chromosomal disruption of HOXA genes by chromosomal rearrangements such as inv(7)(p15q34) or t(7;7)(p15;q34) that involve TRB at 7q34 and t(7;14)(p15;q11) that disrupt TRD at 14q11 results in HOXA upregulation and ectopic expression in T-cell acute lymphoblastic leukemia/lymphoma, indicating important role for HOXA genes in T-ALL.

TCL1A (T-cell leukemia/lymphoma 1A)

Location

14q32.1

Protein

TCL1A located on 14q32.1 and TCL1B located centromeric of TCL1A belong to the TCL1 family of genes known to be affected by chromosomal rearrangements in T-cell leukemia/lymphoma. TCL1 is a target of chromosomal translocations t(14;14)(q11;q32.1) or t(7;14)(q35;q32.1) and inversions inv(14)(q11q32.1) involving the T- cell receptor alpha /delta locus at 14q11 and TCR beta loci at 14q35 leading to its deregulation in T-cell leukemias, such as T-prolymphocytic leukemia. It is also activated in preleukemic clonal cells and chronic leukemias arising in cases of immunodeficiency syndromes such as ataxia-telangiectasia as a consequence of chromosome translocation involving TRA/TRD locus. TCL1 functions as a coactivator in a PI3-kinase dependent Akt pro-survival pathway.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

The chromosomal translocation t(7;14)(p15;q32) is associated with T-cell acute leukemia/lymphoma mainly, indicating the abnormality may involve genes casually implicated in the development of these types of malignancies. Found often as a sole anomaly or in association with limited additional rearrangements in the majority of reported patients, thus probably involved in the initiation of malignancy. The fusion of TCL1A/TCL1B and TRG genes was confirmed by FISH in 1 patient (Sugimoto et al., 2014), indicating that activation of TCL1 by its juxtaposition to regulatory elements of T-cell receptor gene leading to TCL1 overexpression is involved in leukemogenesis. However, in the remaining patients the genes involved are unknown and probable various genes have been implicated. It is possible that in some patients, the abnormality may involve other genes such as HOXA genes identified in 7p15 region and

TCL1B (TML1) and TCL6 (TNG1, or TNG2) that are overexpressed in T-cell leukemias with 14q32.1 rearrangements.

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