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Review

TAL1 (1p32) deletion in lymphoid malignancies

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Abstract

Review on TAL1 deletion in lymphoid malignancies with data on clinics.

Keywords
TAL1; STIL; SIL; T-cell lymphoblastic leukemia; B-cell lymphoblastic leukemia; Follicular lymphoma; Diffuse large B-cell lymphoma; Multiple myeloma; Plasma cell leukemia; Hodgkin lymphoma; Anaplastic large cell lymphoma; Adult T-cell leukemia/lymphoma

Identity

Note
The deletion of the TAL 1 gene may be due to the deletion of the band 1p32 (del(1)(p32)) or to a submicroscopic interstitial deletion between the TAL1 and STIL genes (Note: STIL is better known as "SIL").
The pathogenesis of the two deletions is different. The 1p32 deletion is probably related to the inactivation of a tumor suppressor gene or genes localized in the band 1p32, while the SIL-TAL1 deletion to deregulation of the TAL1 gene. Del(1)(p32) is found in a variety of B or T lymphoid malignances and the SIL-TAL1 deletion is associated only with T-acute lymphoblastic leukemia (ALL).
Until now 39 cases with del(1)(p32) have been reported: 9 cases with acute lymphoblastic leukemia/lymphoblastic lymphoma, 2 cases with adult T-cell lymphoma/ leukemia (HTLV-1+), 8 cases with follicular lymphoma, 6 cases with diffuse large B-cell lymphoma, 3 cases with anaplastic large cell lymphoma, 2 cases with Hodgkin disease, 7 cases with multiple myeloma and 2 cases with plasma cell leukemia.
The 1p32 deletion can be detected by conventional cytogenetics, and the SIL-TAL1 deletion by fluorescence in situ hybridization, Southern blot analysis and real time PCR.

Clinics and pathology

Deletion of band 1p32

Disease: Acute lymphoblastic leukemia/lymphoblastic lymphoma (ALL)

Epidemiology

The deletion is found in 9 cases ALL - 0.08% of all ALL cases with an abnormal karyotype: 3 cases with T-ALL (Schoch et al, 1996; Jarosova et al, 2016), 5 cases with B-ALL (Kristofferson et al, 1985; Pui et al, 1990; Pui et al, 1992; Ivanov Ofverholm et al, 2016) and 1 case without data on the cell phenotype (Prigogina et al, 1988).
The patients with T-cell phenotype are males. The sex ratio of the cases with B-cell phenotype is M:F=1.5:1. The average age is 22.1 year (range 2-74).

Cytogenetics

In six cases del(1)(q32) are with complex predominantly di- or hyperdiploid karyotypes.
In two cases 1p32 deletion is accompanied with a second anomaly; one case with a T-cell phenotype has t(8;14)(q24;q11) and another with a B-cell phenotype del(12)(p12).
In three cases the deletion is presented as additional deleted chromosome 1.

Disease: Follicular lymphoma (FL)

Epidemiology

The 1p32 deletion is described in 8 cases - 0.53% of all FL cases with an abnormal karyotype) (Yunis et al, 1984; Gaunt et al, 1986; Offit et al, 1989; Goyns et al, 1993; Wiodarska et al, 1994; Horsman et al, 2001; Aamot et al, 2007).
The sex ratio is M:F=1:1.7 The average age is 51.2 year (range 43-62) (the age of 5 cases is reported).

Cytogenetics

The cases are presented predominantly with highly complex di- or hyperdiploid karyotypes. In 7 cases the 1p32 deletion is associated with t(14;18)(q32;q21).
In four cases the anomaly is accompanied with structural aberration of chromosome 6.

Disease: Diffuse large B-cell lymphoma (DBCL)

Epidemiology

Six cases are reported - 0.42% of all DBCL cases with an abnormal karyotype (Fukuhara et al, 1983; Ebrahim et al 1990; Weisenburger et al, 1996; Ichinohasama et al, 2000; Fan & Rizkalla, 2003; Kaneko et al, 2011).
The sex ratio is M:F=2:1. The average age is 51.5 year (range 36-65) (the age of 4 cases is reported).

Cytogenetics

The reported cases are predominantly with highly complex hyperdiploid or hypertetraploid karyotypes. In four cases the 1p32 deletion is associated with structural anomalies affecting 3q including the BCL6 locus 3q27.
In three cases are found T-lineage anomalies - 14q11 rearrangements in two cases and t(2;5)(p23;q35) in one case and in two the B-lineage anomalies - 14q32 rearrangements.

Disease: Multiple myeloma (MM)

Epidemiology

Seven cases are described - 0.37% of all MM cases with an abnormal karyotype (Lewis & MacKenzie, 1984; Dewalt et al, 1985; Seong et al, 1998; Weinlander et al, 1998; Lioveras et al, 2004; Wu et al, 2007). The sex ratio is M:F=0.8:1. Age is reported in two cases - 67 and 73 years.

Cytogenetics

All cases are with complex karyotypes. Three have hyperdiploid, one hypertetraploid, one pseudodiploid, and two hypodiploid karyotypes. In two cases an additional 1p32 deletion is found.
In four cases the anomaly is associated with other structural rearrangements of chromosome 1.
Only in one case 14q32 rearrangement is described.

Prognosis

1p32 deletion is major negative prognostic factor for progression free survival and overall survival in the cases with MM (Hebraud B et al, 2014).

Disease: Plasma cell leukemia (PCL)

Cytogenetics

Two cases (males; one 70 year old) are reported - 1.32% of all PCL cases with an abnormal karyotype (Lewis & MacKenzie, 1984; Colovic et al, 2008). Both cases have a complex karyotype (one hypodiploid and another hyperdiplod with 14q32 rearrangement).

Disease: Hodgkin disease (HD)

Cytogenetics

Two cases are reported (males, 52 and 76 years old) - 3.8% of all HD cases with an abnormal karyotype (Schlegelberger et al, 1994; Busson-Le Coniat et al, 1996).
One is with an additional 1p32 deletion and another with a highly complex hyperdiploid karyotype carrying additional structural anomalies of chromosome 1.
Both cases are with deletions of 6q.

Disease: Anaplastic large cell lymphoma (ALCL)

Epidemiology

Three cases are reported (two males and one female; 24, 40 and 52 years old) - 2.0% of all ALCL cases with an abnormal karyotype (Ebrahim et al, 1990; Falzetti et al, 1999; Colleoni et al, 2000). Two of them are with a T-cell phenotype.

Cytogenetics

The three cases are with a complex karyotype.
In one t(2;5)(p23;q35) is found and is associated with an additional 1p32 deletion and in another two copies of add(2)(p23).
In all three cases the 1p32 deletion is accompanied with structural anomalies of chromosome 8, two of them with i(8)(q10).

Disease: Adult T-cell lymphoma/leukemia (HTLV+) (ATCL)

Cytogenetics

Two cases with a complex karyotype are reported (males; 58 and 68 years old) - 0.75% of all ATCL cases with an abnormal karyotype (Sadamory et al, 1986; Sadamory et al, 1991).
In one the 1p32 deletion is presented as an additional anomaly. Both cases have structural aberrations involving chromosome 4.

SIL-TAL1 deletion

Disease: T- Acute lymphoblastic leukemia/lymphoblastic lymphoma (T-ALL)

Phenotype/cell stem origin

Is restricted to TCR of the alpha, beta and TCR delta lineage with a deletion of one or both alleles of the TCR delta gene (Breit et al, 1993a). TAL1 expression appeared to reflect the cortical stage of thymocyte development (late double positive stage) (Ferrando AA et al, 1992).

Etiology

SIL-TAL1 deletion is mediated via illegitimate V(D)J recombination processes of T-cell receptor (TCR) gene (Aplan PD et al, 1990b).

Epidemiology

SIL-TAL1 deletion is observed in 3-26 % of cases with T-ALL (Aplan et al, 1990b; Brown et al, 1990). The anomaly is more frequent in males (D'Angio M et al, 2014).

Clinics

SIL-TAL1 deletion is associated with higher initial WBC count, T-lineage immunophenotype with CD2 expression, predominant cortical T-phenotype, low incidence in adult patients and higher frequency of extramedullary relapse (Bash RO et al, 1993; Stock W et al, 1995; Mansur MB et al, 2009; D'Angio M et al, 2014). Increased risk from developing of tumor lysis syndrome and disseminated intravascular coagulation were also reported (Wang D et al, 2013).

Cytogenetics

SIL-TAL1 deletion represents a submicroscopic deletion of 90 Kb that affected all coding SIL exons. As a result of the deletion the first coding exons of TAL1 gene is juxtaposed to the promotor of SIL gene, causing its abnormal expression (Chen Q et al, 1990b; Aplan PD et al, 1992b). The breakpoint in SIL gene remains constant while several breakpoints of the TAL1 gene have been identified, which leads to formation of two main ($TAL1^{d1}$ and $TAL1^{d2}$ in 95% of the cases) and several rare types of SIL-TAL1 deletions (Breit et al, 1993a). Almost half of the cases with SIL-TAL1 deletion have normal karyotypes. The rest of the cases are presented with hyperdiploid and more frequently with pseudodiploid karyotypes associated with the structural anomalies del(6q) (7 cases), t(11;14)(p13;q11.2)/ t(11;14)(p15;q11.2) (3 cases) and t(8;14)(q24;q11.2) (1 case) (Wang Q et al, 2014; Cocce MC et al, 2015).

Prognosis

The data on the prognosis of the cases with SIL-TAL1 deletion are controversial. Mansur MB et al. (2009) reported negative impact on the patients, while no difference in survival and overall outcome were seen by D'Angio M et al. (2014).

Genetics

Using chromatin immunoprecipitation sequencing and chromosome conformation capture techniques, several scientific groups presented looping models for TAL1 expression in human and murine cell lines and described multiple interactions between TAL1 and their cis-acting regulatory elements (1a and 1b TAL1 promotors, enhancers and CTCF bound elements) (Zhou Y et al, 2013; Lai F et al, 2013; Patel B et al, 2013). Zhou Y et al. (2013) find in TAL1 expressing cell lines that the regulatory hubs which control transcription bring the TAL/SIL common breakpoint regions (TAL^{d}) into close proximity. The authors suggest that the physical proximity between these regions in the committed lymphoid cells may predispose to SIL-TAL1 deletion. However, the question remains: what is the reason that led to the rearrangement of the located in close proximity breakpoint regions? It should be noted that TAL1 gene is not expressed in the dividing double positive thymocytes, but SIL gene is expressed. In addition, the deletion primarily affects the SIL gene (all coding exons are deleted), so it can be assumed that the consequence of SIL-TAL1 deletion is not only deregulation of the TAL1 gene, but also inactivation of the SIL gene. These considerations suggest that the generation of SIL-TAL1 deletion is possibly due to a defective inactivation of the SIL gene which is intended to block the G2/M transition (through impairing the spindle assembly) and is a part of the complex mechanisms that induced the apoptosis during thymocyte negative selection. In this regard, future studies of looping patterns searching to discover the possible interactions leading to suppression of the SIL gene will elucidate the mechanisms of the formation of the SIL-TAL1 deletion as well as the role of the SIL gene in the regulation of the thymocyte apoptosis.

Genes involved and proteins

STIL

Location 1p33

STIL (or SIL: SCL interrupting locus) gene extended over 70 Kb and contained 18 exons.

The gene encodes a large (150 kDa) cytosolic protein implicated in regulation of the mitotic spindle checkpoint. It is required for the procentriole assembly and the regulation of the centriole

duplication. SIL mRNA expression is higher in rapidly proliferating cells and decreased rapidly during terminal differentiation. It is a positive regulator of the sonic hedgehog pathway and plays an important role in embryonic development. It is over expressed in multiple types of cancer and its expression correlates with the expression of mitotic checkpoint.

TAL1

Location 1p33

TAL1 (T-cell acute leukemia 1) is a member of the class II helix-loop-helix (bHLH) family of transcription factors. The gene extended over 16 Kb and contained 6 exons. Have two isoforms: a long TAL1 (L) and short TAL1 (S). After heterodimerization with members of the class I bHLH proteins known as E proteins (TCF3 (E2A), TCF12 (HEB), BHLHE22 (E2-2)), it binds E-box motif and forms complex with other transcription factors, including LMO, GATA1, RUNX1 and LDB1. TALl1 is expressed in hematopoietic stem cells, progenitor cells and erythro-megakaryocyte lineage. It is required for the specification of the haemangioblasts and the blood cell lineages and also plays a key role for the maturation of the megakaryocytes and erythroblasts (Porcher C et al, 1996; Porcher C et al, 2017; Gering M et al, 1998; Schlaeger TM et al, 2005). TAL1 is transcriptionally silenced during normal lymphocyte development including at the stage of CD4+ CD8+ double-positive thymocytes (the stage of maturation arrest of TAL1 positive T-ALL) (Tremblay M et al, 2010; Seita J et al, 2012). The transcriptional targets of the TAL1 in the normal hematopoietic cells are KIT, CDKN1A, DDIT4, KLF1, EPB42, GYPA, UBE2H and MEF2C (Lecuyer E et al, 2002; Lacombe J et al, 2010; Benyoucef A et al, 2015; Kassouf MT et al, 2010; Xu Z et al, 2003). As part of the highly interconnected auto-regulated circuit, it controls the transcription factors LMO2, RUNX2, MYB and GATA2. Except through a SIL-TAL1 deletion, TAL1 deregulation occurs also as a result of the chromosome translocations t(1;14)(p32;q11) and t(1;7)(p32;q34) and through its ectopic expression (60% of the cases) (Ferrando AA et al, 2002). Recently another mechanism oduces a binding motif for MYB transcription factor (Mansour MR et al, 2014). Deregulation of TAL1 inhibits E-proteins heterodimerization leading to block of T-cell differentiation. However, the oncogenic role of TAL1 in T-cell transformation is more complex and is linked to their influence on the function of the core regulatory circuitry (Sanda T et al, 2012) as well as on multiple downstream targets including ARID5B, NKX3-1, MYCN, CDKN2A, ALDH1A2 and MIR223 (Leong WZ et al, 2017; Kusy S et al, 2010; Astolfi A et al, 2014; Hansson A et al, 2003; Ono Y et al, 1998; Mansour MR et al, 2013).

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