t(6;14)(p22;q32)

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Published in Atlas Database: April 2008
Online updated version : http://AtlasGeneticsOncology.org/Anomalies/t0614p22q32ID1480.html
DOI: 10.4267/2042/44457
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Clinics and pathology

**Disease**
B-cell acute lymphoblastic leukaemia (B-ALL)

**Phenotype/cell stem origin**
Pre-B ALL: CD10+ in 14 of 15 cases, CD19+ probably in the remaining case.

**Epidemiology**
15 cases available: 9 male and 6 female patients; mediane age was 16 years (range: 6 - 48 years).

**Clinics**
Complete remission was obtained in 15 of 15 cases.

**Prognosis**
Prognosis looks fair (see survival curve).

**Cytogenetics**

**Cytogenetics morphological**
Major karyotypic anomalies were seen in 13 of 15 cases, an i(9q) in 10/15 cases (a rather rare anomaly otherwise), +5 in 5/15, and del(13q) in 3/15. The syndrome is therefore very homogeneous, at the clinical, cytological, and cytogenetic levels.

**Genes involved and proteins**

**Note**
Deletion of CDKN2A and PAX5 was found in 10 of 10 cases tested, eventhough in cases without isochromosome (9q)

**ID4**
**Location**
6p22
**Protein**
Member of the ID gene family: "Inhibitors of DNA binding". They are transcription factors which act as transcription inhibitory proteins. They are basic helix-loop-helix (bHLH) proteins which contain the bHLH dimerization domain, but lack the DNA binding domain. They form heterodimers with other bHLH proteins and inhibit them. ID4 may play an important suppressive role in tumor progression, and its silencing by hypermethylation favours tumorogenesis.

**IGH**
**Location**
14q32
Result of the chromosomal anomaly

Hybrid gene

**Description**
No hybrid gene, but juxtaposition of ID4 to the IGH enhancer, leading to ID4 overexpression.

**References**


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