t(14;20)(q32;q13) IgH/CEBPB

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Clinics and pathology

**Phenotype/cell stem origin**

CD10+ acute lymphoblastic leukaemia (B-ALL).

**Epidemiology**

Only four cases to date (Martineau et al., 1996; Akasaka et al., 2007), one male patient and 3 female patients, aged 11, 13, 15, and 35 yrs.

**Clinics**

There was a high WBC in 2 of 3 cases.

**Prognosis**

Survival was 7 mths+, 42 mths+, and 87 mths+.

Genetics

**Note**

In the 3 cases reported by Akasaka et al (2007), a CEBPB / IGH involvement was uncovered.

**Genes involved and proteins**

**CEBPB**

**Location**

20q13

**Protein**

DNA-binding protein. CCAAT enhancer-binding protein (CEBP) transcription factors are a family of 6 multifunctional basic leucine zipper (bZIP) transcription factors. The 5 other CEBPs are: CEBPA (19q13), CEBPD (8q11), CEBPE (8q11), CEBPG (19q13), all four equally implicated in leukemias, and DDIT3/CHOP/CEBP zeta (12q13), so far known to be involved in solid tumours (liposarcoma). These transcription factors play a key role in cellular differentiation, in particular in the control of myeloid differentiation. CEBPB is composed of a N-term transactivation domain, a negative regulatory domain, a DNA-binding basic motif, and a leucine-zipper domain in C-term. Different isoforms (LAP: liver activating protein, and LIP: liver inhibitory protein). CEBPB suppresses cell proliferation through repression of E2F target genes; on the other hand, CEBPB may promote tumorization of epithelial cells (Ramji et al., 2002; Nerlov et al., 2007).

**IgH**

**Location**

14q32

Result of the chromosomal anomaly

**Fusion protein**

**Oncogenesis**

Overexpression of the CEBP gene.

References


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