

Leukaemia Section

Short Communication

t(11;14)(q11;q32)

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Abstract

Review on t(11;14)(q11;q32), with data on clinics, and the genes involved.

Keywords

Immunoglobulin translocations, B-cell lymphoproliferative malignancies, multiple myeloma, gene overexpression

Clinics and pathology

Disease

B-cell lymphoid malignancies and multiple myeloma.

Etiology

B-cell lymphoid malignancies in 5: 3 chronic lymphocytic leukemia (CLL) (Schroder et al., 1981; Weisenburger et al., 1987; Bird et al., 1989), 1 plasma cell leukemia (PCL) (Ueshima et al., 1983), 1 mantle cell lymphoma (MCL) (Espinat et al., 1999) and 2 multiple myeloma (MM) patients (Sawyer et al., 1995; Gozzetti et al., 2011).

Epidemiology

2 males and 5 females aged 45 to 78 years, median 63 years.

Prognosis

Chromosome 14q32 translocations that are part of complex karyotypes are associated with an adverse prognosis in B-cell malignancies.

Cytogenetics

Note

The breakpoint on chromosome 11 is 11q13 in the most common t(11;14)(q13;q32), therefore some of

the translocations described as t(11;14)(q11;q32) in early reports may involve 11q13 breakpoint.

Additional anomalies

Found in association with highly complex karyotypes, thus it is unclear if t(11;14)(q11;q32) was a primary aberration in these patients or it appeared as a secondary change during karyotypic progression.

Genes involved and proteins

IGH (Immunoglobulin Heavy Locus)

Location

14q32.33

Note

IGH translocations relocate genes near active regulatory sequences of the partner gene, resulting in their overexpression.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

Translocations involving chromosome 14 at band q32, at the site of the immunoglobulin heavy chain (IGH) locus have been described in a spectrum of B-cell malignancies. In these translocations, various partner chromosomes have been described, including chromosome 11, of which the t(11;14)(q13;q32) that leads to the overexpression of the CCND1 gene is the most common. Chromosome translocations involving centromeric 11q breakpoints are less frequent and have been

described only in sporadic cases of B-cell lymphoid malignancies and multiple myeloma. Although the mechanism of neoplastic transformation remains unknown, deregulation of the translocated partner gene as a consequence of its transposition into the IGH locus may represent a mechanism of oncogene activation.

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