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Leukaemia Section
Short Communication

15q13-15 Rearrangements

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Clinics and pathology

Disease

Acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), chronic lymphocytic leukemia (CML), chronic lymphocytic leukemia (CLL) and non Hodgkin lymphomas (NHL).

Phenotype/cell stem origin

Most ALL are B-lineage, few T-lineage.
AML: most subtypes.
CML: Lymphomas: described in follicular, diffuse large B-cell, mantle cell and anaplastic large cell, as well as Hodgkin lymphomas.

Epidemiology

Rare in childhood ALL (about 1%), more common in infant ALL (about 13%).
Very rare in AML, adult ALL, CLL and lympho-mas.
Occurs in primary and secondary leukemias.
CML: rare, occurs as secondary abnormality or part of complex Ph rearrangement.

Prognosis

Childhood ALL: No increased risk with current treatment regimens. Outcome not described in other diseases.

Cytogenetics

Additional anomalies

Various anomalies result in 15q13-15 breakpoints, most frequently balanced translocations, but also unbalanced translocations and deletions.
Childhood ALL: 15q13-15 breakpoints frequently occur in complex karyotypes. Associated abnormalities of 9p and of 12p 12p, as well as t(9;22)(q34;q11.2) are common. 15q13-15 break-points also have been reported with der(19)t(1;19)(q21;q13). A t(5;15)(p15.1 or p15.3;q11 or q13) occurs primarily in infant ALL, although also was reported in one adolescent ALL.
AML: found as primary abnormality and as secondary abnormality, with various other abnormalities including t(9;22)(q34;q11.2), t(8;21)(q22;q22), as well as part of complex rearrangements with 8q22 and 21q22.
Recurrent aberrations are: t(11;15)(q23;q14) reported in both AML and ALL and t(12;15)(p12-13;q13-15) in AML.
CML: occurs in variant Ph and in secondary abnormalities.

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