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Leukaemia Section

Short Communication

t(14;19)(q32;q13) in acute lymphoblastic leukaemia

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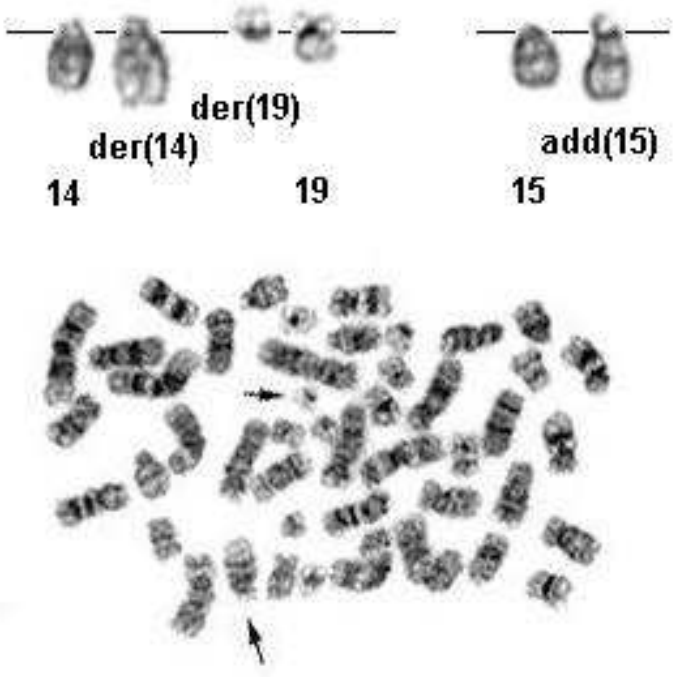
Identity

Note: This abnormality is cytogenetically identical but molecularly distinct from the t(14;19) in CLL and other chronic B-cell lymphoproliferative disorders which results in the juxtaposition of BCL3 with IGH on the der(14) and subsequent over expression of the BCL3 protein.



G-banded metaphase showing the t(14;19)(q32;q13). The derivative chromosomes 14 and 19 are arrowed (bottom). G-banded karyogram showing the t(14;19)(q32;q13) and a add(15q) (top).

Clinics and pathology

Disease

Acute lymphoblastic leukaemia.

Phenotype/cell stem origin

B-lineage immunophenotype and FAB L1.

Epidemiology

Very rare with only 10 cases reported to date. The estimated incidence in childhood and adult ALL is <1%. Among the reported cases there appears to be a female pre-dominance which is unusual for ALL. The age range of patients is 9 to 28 years with a median of 11.5 years. Thus this abnormality appears confined to adolescents and young adults.

Clinics

Typically patients with this abnormality have low white cell count of 9/L.

Prognosis

It is difficult to assess the true prognosis of patients with this abnormality given its rarity, however initial data suggest that the prognosis is better than expected for patients of a similar age.

Cytogenetics

Note

This balanced translocation can usually be identified by G-banding alone. The breakpoint on chromosome is consistently given as 14q32; however the breakpoint on chromosome is more variable ranging from q11 to q13.
The t(14;19) has been described as the sole abnormality in several cases but is more frequently accompanied by additional structural abnormalities; none of which have yet shown to be recurrent.
This abnormality has been reported in a single case with Down Syndrome.

Genes involved and proteins

Note

The involvement of the IGH gene located at 14q32 has been demonstrated via FISH using the LSI IGH Dual Colour Break Apart Rearrangement Probe in all cases tested.
Metaphase and interphase FISH using probes flanking the BCL3 gene have ruled out the involvement of this gene; thus distinguishing it from the cytogenetically identical translocation seen in CLL and other chronic B-cell lymphoproliferative disorders. The target gene on 19q13 is currently thought to lie centromeric of the BCL3 gene, but has not yet been elucidated.

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