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Leukaemia Section
Short Communication

t(14;18)(q32;q21)(IgH/MALT1)

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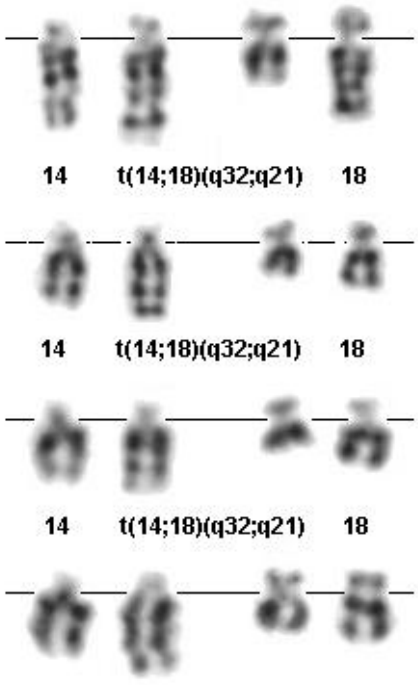

t(14;18)(q32;q21) with additional cytogenetic abnormalities in a hepatic MALT lymphoma. FISH demonstrated an IGH-MALT1 rearrangement.

Clinics and pathology

Disease

t(14;18)/IGH-MALT1 was detected in MALT lymphoma first. The frequencies at which the translocation occurs vary markedly with the primary site of the disease. IGH-MALT1 rearrange-ments were described also in other B-NHLs such as DLBCL (Diffuse Large B-Cell Lymphoma).

Cytogenetics

Cytogenetics morphological

The t(14;18)/IGH-MALT1 is cytogenetically indistinguishable from the t(14;18)/IGH-BCL2. FISH with gene specific probes is suitable to distinguish between these two different rearrange-ments.

Genes involved and proteins

IGH

Location
14q32.33

MALT1

Location
18q21

Protein
Stimulation of either the T cell antigen receptor (TCR) or B cell antigen receptor leads to stimulation of protein kinase C isoforms that phosphorylate the scaffolding protein CARMA1, which subsequently recruits both Bcl-10 and MALT1 to form what is now referred to as the CARMA1-Bcl-10-MALT1 (CBM) 'signalosome'. Once the CBM signalosome is assembled, MALT1 functions as the 'effector' protein and mediates activation of the IKK complex, a multi subunit kinase that phosphorylates the Ikappa B proteins, which bind to and sequester the transcription factor
NF-kappaB in the cytoplasm. Phosphorylation and subsequent degradation of Ikappa B leads to the release of NF-kappaB, which then translocates to the nucleus and regulates the transcription of 'target' genes involved in the immune response to foreign antigens.

Result of the chromosomal anomaly

Hybrid gene

Note

Breakpoints upstream the coding exons of MALT1 resulting in an in-frame deregulation of MALT1.

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