Leukaemia Section
Short Communication

**t(12;15)(p13;q25)**

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**Clinics and pathology**

**Disease**
Acute myeloid leukaemia (AML), FAB M2 subtype.

**Epidemiology**
Only one case to date, a 59-year-old female patient.

**Prognosis**
The patient died 5 months after the onset of the leukaemia.

**Cytogenetics**

**Additional anomalies**
+8 and other anomalies.

**Genes involved and proteins**

**ETV6**
Location
12p13
Protein
ETS-related transcription regulator.

**NTRK3**
Location
15q25
Protein
Tyrosine kinase cell surface receptor.

**Result of the chromosomal anomaly**

**Hybrid gene**
Description
5’ ETV6-3’ NTRK3

**Fusion protein**

**Description**
Encodes a fusion protein with the sterile alpha motif (SAM) oligomerization domain of ETV6 in N-term to the C- term protein tyrosine kinase (PTK) domain of NTRK3.

**Oncogenesis**
Functions as a constitutively active tyrosine kinase. ETV6-NTRK3 is capable of homodimerization, or heterodimerization with ETV6, and subsequent PTK activation, leading to constitutive elevation of cyclin D1, and increased cell cycle progression. ETV6-NTRK3 also leads to constitutive activation of two of the major effector pathways of NTRK3: the Ras-MAPK mitogenic pathway and the phosphatidyl inositol-3-kinase (PI3K) pathway leading to activation of the AKT cell survival factor (Lannon and Sorensen, 2005).

**To be noted**

Note
The same translocation t(12;15)(p13;q25) with the same genes involved (ETV6-NTRK3) can also be found in: Secretory Ductal Breast Carcinoma, in Congenital Mesoblastic Nephroma, and in Congenital Fibrosarcoma.

**References**


This article should be referenced as such: