Leukaemia Section
Short Communication

**t(5;17)(p11;q11) and t(5;17)(q11-12;q11-12)**

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**Abstract**
Review on t(5;17)(p11;q11) and t(5;17)(q11-12;q11-12), with data on clinics.

**Keywords**
Chromosome 5; chromosome 17; Acute myeloid leukaemia

**Clinics and pathology**

**Disease**
Acute myeloid leukemia (AML)

**Epidemiology**
Described in 2 male and 3 female patients aged 28 to 70 years; Acute myeloblastic leukemia with maturation (FAB type M2) in 3 (Huebner et al., 2000; Paietta et al., 1988; Arnaud et al., 2005) and AML-NOS in 2 patients (Suciu et al., 1993; Kerndrup and Kjeldsen., 2001) (Table 1).

**Prognosis**
May represent an unfavorable cytogenetic prognostic category in association with monosomy 7 and/or complex karyotypes.

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**Figure 1.** (A) Partial karyotypes showing the t(5;17)(p11;q11). (B) Fluorescence in situ hybridization with LSI TP53/CEP17 probe (Vysis, Abbott Molecular, US) showing the chromosome 17 centromere (green) and the p53 gene (red) on der(17) chromosome containing the short arms of chromosomes 5 and 17. (C) Hybridization with LSI CSF1R/D5S23/D5S721 hybridizing on 5p13.2 (green) and 5q33 (red) showing the signal for 5p (green) on der(17) and the signal for 5q33 (red) on der(5) chromosome. (D) Simultaneous hybridization with LSI TP53/CEP17 and LSI CSF1R/D5S23/D5S721 probes demonstrating the presence of TP53/CEP17 and 5p13 signals on der(17) and the 5q33 signal on der(5) chromosome containing the long arm of chromosome 17.
**Cytogenetics**

**Additional anomalies**

Complex chromosome rearrangements in all the 5 described patients, found in association with monosomy 7 in 2 (Huebner et al., 2000; Arnaud et al., 2005) and trisomy 8 in 3 patients (Paietta et al., 1988; Suciu et al., 1993; Kerndrup and Kjeldsen., 2001).

**Result of the chromosomal anomaly**

**Fusion protein**

**Oncogenesis**

The reciprocal, apparently balanced t(5;17)(p11-q11;q11) is a rare but non-random anomaly in acute myeloid leukemia, that may be particularly associated with acute myeloblastic leukemia with maturation (AML-M2). The key mechanism of oncogenesis is unknown; however as it presents in association with known anomalies such as monosomy 7 or trisomy 8 in all the described cases, it is likely that it represents a secondary anomaly that developed during the multistep process of leukemogenesis.

**References**


This article should be referenced as such: