Leukaemia Section
Short Communication

del(1p) solely
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Abstract
Deletions of the short arm of chromosome 1 are observed in a broad range of hematological malignances including myeloid disorders, acute lymphoblastic leukemia (ALL), multiple myeloma (MM) and lymphomas. They generally occur as part of complex karyotypes and in advanced disease stages. Their association with complex karyotypes likely reflect an inherent chromosomal instability correlated with a poor prognosis. The occurrence of 1p deletions as a sole anomaly is infrequent and their clinical significance is less well characterized.

KEYWORDS
Chromosome deletions; tumor suppressor genes; gene downregulation; nonrandom 1p deletions.
**Clinics and pathology**

**Disease**
Myeloid malignancies, acute lymphoblastic leukemia (ALL) and multiple myeloma (MM)

**Epidemiology**

Myeloid malignancies mainly (10 patients): 5 patients with myelodysplastic syndromes (MDS) (3 males, 2 females aged 64 to 71 years) (Parlier et al., 1994; Westbrook et al., 2000; Lubbert et al., 2001; Mallo et al., 2008; Wang et al., 2010;) and 5 acute myeloid leukemia (AML) cases (2 males and 3 females aged 16 to 46 years) (Raimondi et al., 1999; Lemez et al., 2000; Coupland et al., 2002; Casas et al., 2004; Gmidène et al., 2012). In addition, there was 1 ALL patient (Zuelzer et al., 1976), a 7-years old T-ALL patient (Kaneko et al., 1989), 1 infant patient with biphenotypic leukemia (Al-Seraiyah et al., 2009) and 7 multiple myeloma cases (3 males and 4 females aged 41 to 72 years) (Ankathil et al., 1995; Calasanz et al., 1997; Pantau et al., 2005).

**Cytogenetics**

**Cytogenetics morphological**

Various breakpoints; found in a form of interstitial or terminal deletion, the most commonly breakpoint described is p11p22 found in 4 out of 7 MM cases; found in a sideline in 1 AML (Raimondi et al., 1999) and in 1 MM case (Pantau et al., 2005).

**Result of the chromosomal anomaly**

**Fusion protein**

**Oncogenesis**

Deletions of the short arm of chromosome 1 represent nonrandom structural aberrations in hematological malignancies, suggesting the existence of tumor suppressor genes encoded in this region. Loss of genetic information from the deleted region may contribute to disease pathogenesis via dosage reduction of genes leading to their aberrant expression. Most 1p deletions involve large regions containing a certain fraction of genes, therefore multiple tumor suppressive genes might cooperate in an additive or synergistic way leading to their simultaneous downregulation.

**References**


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