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Leukaemia Section
Short Communication

der(1;14)(p10 or q10;p10 or q10)

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Abstract

Review on der(1;14)(p10 or q10;p10 or q10) translocation, with data on clinics

Clinics and pathology

Disease

Myelodysplastic syndrome (MDS), myeloproliferative neoplasm (MPN), acute myelogenous leukemia (AML), acute lymhoblastic leukemia (ALL), multiple myeloma (MM) and lymphoma.

Phenotype/cell stem origin

Suggested involvement of a pluripotent stem cell.

Epidemiology

Most of patients (9/14) had a diagnosis of myeloid disorder: AML (2 patients), MDS (2 cases) and chronic myeloproliferative disorders (5 cases). A few other cases included ALL (2 patients), MM (1 case) and 2 cases with lymphoid malignancies.
The male sex is prevalent (9:5); the median age at diagnosis is 59 years (range 10-74 years) (Table 1).

Prognosis

The prognostic effect of an extra 1q chromosome in myeloid disorders may be variable (indolent clinical course in MPD vs a distinct possibility of transformation and poor prognosis in MDS).
The prognosis of patients with other diseases is unknown (sporadic cases reported).

Cytogenetics

Cytogenetics morphological

Whole arm chromosome translocation showing 2 normal copies of chromosome 1, only 1 normal chromosome 14 and a der(1;14) resulting in complete 1q trisomy. The centromeric breakpoints were confirmed only in sporadic cases, revealing an unique centromere derived from chromosome 14 (Busson-Le Coniat et al.,1999; Djordjevic et al., 2005; Fogu et al., 2013). The preservation of both chromosome 1 and 14 centromeres was not confirmed.

Additional anomalies

Sole anomaly in half cases; found in association with del(5q), del(7q), del(12p), del(20q), t(9;22)(q34;q11) in myeloid malignancies and with t(8;14)(q24;q32) in Burkitt lymphoma.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

The unbalanced 1q whole-arm translocation with the recipient acrocentric 14 chromosome results in pure trisomy of the long arm of chromosome 1. The main consequence of 1q trisomy is a genomic imbalance that may account for a proliferation advantage of the neoplastic clone through a gene dosage effect.

Sex/Age	Karyotype	Disease	Reference
M/59	46,XY,+1,der(1;14)(q10;q10)	Post-PV MDS	Swolin et al.,1986
F/ 28	46,XX,+1,der(1;14)(q10;q10),der(19)t(1;19)(q?23;p?13)	ALL	Nylund et al., 1994
M/54	47,XY,+X/35-42,XY,der(1;13)(q10;q10),der(1;14)(p10;q10)	NHL	Hashimoto et al., 1995
F/64	46,XX,der(1;14)(q10;p10)	Post-PV MF	Andrieux 2003
F/59	47,X,add(X)(p22),+7,t(8;14)(q24;q32)/47,idem,add(7)(p22)/ 47,idem,+1,der(1;21)(q10;q10)/47,idem,+1,der(1;14)(q10;q10)	BL	Chan et al., 2003
F/11	46,XX,+1,der(1;14)(q10;q10)/46,idem,add(9)(q34),add(17)(p11)	RAEB	Imashuku et al., 2003
M/69	46,XY,der(1;14)(p10;p10),del(12)(p12)	RAEBt	Harada et al., 2004
F/	46,XX,der(1;14)(q10;q10)	PV	Zamora et al., 2004
M/73	45-46,XY,der(1;14)(p10;q10),del(5)(q13q33),+8,+9,del(11) (q14),del(20)(q11),der(21)t(21;21)x2,-22	RARS	Barouk-Simonet et al., 2005
M/65	46,XY,+1,der(1;14)(q10;q10)	ALL	Adeyinka et al., 2007
M/73	46,XY,+1,der(1;14)(q10;p10)	MM	Adeyinka et al., 2007
M/74	46,XY,+1,der(1;14)(q10;p10)	MPD	Adeyinka et al., 2007
M/43	46,XY,+1,der(1;14)(q10;q10)	CMMoL	Djordjevic et al., 2008
	46,XY,+1,der(1;14)(q10;q10) 46,XY,+1,der(1;14),del(7)(q22) 46,XY,+1,der(1;14),del(7),t(9;22)(q34;q11) 46,XY,+1,der(1;14)t(1;2)(q25;q37),del(7)	AML- M4	Lee et al., 2008

Table 1. Reported cases with der(1;14) centromere translocations involving 1p10 or 1q10.
Abbreviations: ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; CMML, chronic myelomonocytic leukemia; F, female; M, male; MF, myelofibrosis; MDS, myelodysplastic syndrome; MM, multiple myeloma; MPD, myeloproliferative disease; PV, polycythemia vera; PPMF, myelofibrosis after polycythemia vera; RAEB, refractory anemia with excess of blasts; RAEBt, refractory anemia with excess of blasts in transformation; RARS, refractory anemia with ringed sideroblasts.

The consequent deregulation of several genes results in alteration of the balance between proliferation and cell death, suggesting that gene deregulation is the main mechanism of oncogenesis in 1q rearrangements, similar to numerical aberrations, (such as trisomy 8) observed in various hematologic malignancies.

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