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Short Communication

der(1)t(1;1)(p36;q11-q32) in hematopoietic malignancies

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Abstract

Abstract
Review on der(1)t(1;1)(p36;q11-q32) in hematopoietic malignancies, with data on clinics, and the genes involved.

Clinics and pathology

Disease

Chronic myeloproliferative neoplasms (MPN), acute myeloid leukemia, acute lymphoblastic leukemia (ALL) and multiple myeloma (MM).

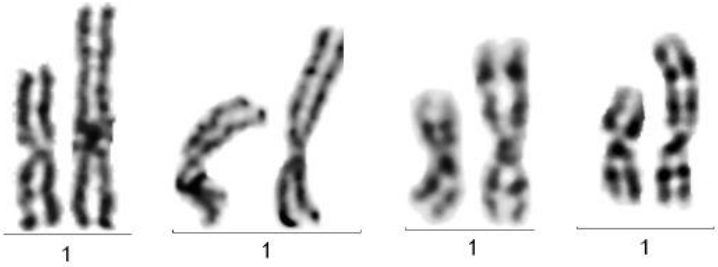
Phenotype/cell stem origin

34 patients, mainly myeloid malignancies (19 cases). 10 patients had lymphoid malignancies (8 ALL, 1 chronic lymphocytic leukemia (CLL) and 1 plasma cell leukemia (PCL) and there were 5 MM cases (Table 1).

Chronic myeloproliferative neoplasms 5 patients: 1 polycythemia vera (PV) who progressed to myelofibrosis (MF) 27 years later (Duhoux et al., 2011), 1 chronic eosinophilic leukemia (CEL) (Duhoux et al., 2011), 1 chronic myeloid leukemia (CML) (Phan et al., 2006) and 2 patients with myelodysplastic syndrome (MDS) (Caramazza et al., 2010; Duhoux et al., 2011).

Acute myeloid leukemia 14 patients, mainly with myelomonocytic-lineage: 1 acute myeloblastic leukemia with maturation (Fugazza et al., 1997), 2 acute myelomonocytic leukemia (Michalova et al., 1999; Duhoux et al., 2011), one of them progressed from MDS (Michalova et al., 1999).

From the 6 acute monocytic leukemia cases (Chessells et al., 2002; Picos-Cardenas et al., 2002; Scrideli et al., 2005; Noguchi et al., 2007; Duhoux et al., 2011; Sarova et al 2013), 1 progressed from MDS (Duhoux et al., 2011), 1 was a case with Down syndrome (DS) (Picos-Cardenas et al., 2002) and there were 2 infants (Chessells et al., 2002; Scrideli et al., 2005), one of them with partial trisomy 1q syndrome (Scrideli et al., 2005). 1 patient was diagnosed with acute megakaryoblastic leukemia (Preiss et al., 2006) and there were 4 AML unspecified patients (Duhoux et al., 2011; Blink et al 2012), 3 of them infants, among them one with DS (Blink et al., 2012).



Acute lymphoblastic leukemia 8 patients (Shikano et al.,1994; Uckun et al 1998; Bruch et al 2003; Paulsson et al 2010; Schmidt-Hieber et al 2010; Duhoux et al 2011; Subramaniyam et al 2011; Safavi et al 2015) with predominantly B-cell phenotype. There were 5 children (Shikano et al.,1994; Uckun et al 1998; Paulsson et al 2010; Duhoux et al., 2011) , one of them an infant patient (Bruch et al., 2003).
Various B-cell hematological neoplasms: 5 MM (Rajkumar et al., 1999; Bang et al., 2006; Vekemans et al., 2010; Sawyer et al., 2014), 1 CLL (Nordgren et al., 2000) and 1 plasma cell leukemia (Bastard et al.,1991) patient.

Epidemiology

20 male and 14 female patients (sex ratio 1.4) with median age of 60 years (range 0 to 83 years). Balanced sex ratio in myeloid malignancies (10M/9F) and male prevalence in ALL cases (7M/1F, sex ratio 7). There were 10 pediatric cases (aged 0 to 12 years) in AML and ALL groups, 6 of them were infants (5 AML and 1 ALL cases).

Prognosis

Patients with 1p36 / PRDM16 translocations and/or terminal 1p36 deletions seems to have a poor prognosis despite a simple karyotype (Duhoux et al., 2011). In addition, there seems to be an over-representation of complex karyotypes that in association with an unbalanced rearrangement may reflect genomic instability correlated with an unfavorable outcome.

Cytogenetics

Cytogenetics morphological

Presents as one normal chromosome 1 and a der(1)t(1;1) chromosome, resulting in partial 1q trisomy. Heterogeneous cytogenetic presentation with q11-32 breakpoints on 1q.

Additional anomalies

Sole anomaly in 6 myeloid disorders: 1 MDS (Duhoux et al., 2011) and 5 AML (Noguchi et al 2007; Duhoux et al 2011). Two or several independent clones were found in 2 MPN (Caramazza et al., 2010; Duhoux et al., 2011) and 2 AML cases (Michalova et al., 1999; Chessells et al., 2002). Associated with chromosome 5 and/or 7 anomalies in 4 AML (Fugazza et al., 1997; Picos-Cardenas et al., 2002; Preiss et al., 2006; Duhoux et al., 2011) and with t(9;22)(q34;q11) in 1 CML (Phan et al 2006) and 2 ALL cases (Uckun etal 1998; Schmidt-Hieber et al 2010). Very complex karyotypes (≥5 abnormalities) were observed in 15 patients (Bastard et al.,1991; Fugazza et al., 1997; Rajkumar et al., 1999; Bang et al., 2006; Phan et al., 2006; Preiss et al., 2006; Nordgren et al., 2000; Schmidt-Hieber et al., 2010; Vekemans et al., 2010; Duhoux et al., 2011; Subramaniyam et al., 2011; Sawyer et al., 2014; Safavi et al 2015) while there seems to be an over-representation of complex anomalies in AML, ALL and B-cell hematological neoplasms.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

The chromosomal translocation der(1)t(1;1)(p36;q11-q32) is a nonrandom event in both myeloid and lymphoid neoplasias (Lestou et al., 2003; Duhoux et al.,2012). While the cytogenetic presentation is heterogeneous and there is a marked variability in 1q and 1p36 breakpoints, the common impact of this rearrangement is duplication of 1q sequences through formation of an unbalanced translocation.
In addition, 1p36 alterations are frequent in hematological malignancies, suggesting that the region is harboring gene(s) important in oncogenesis. One of the genes that may be functionally disrupted is the PRDM16 (PR domain containing 16; also known as MEL1 and MDS1/EVI1-like) gene, the main target of 1p36 rearrangements (Duhoux et al., 2012; Matsuo et al., 2015). Among them, PRDM16 have been found to be rearranged with the DUSP10 (dual specificity phosphatase 10) gene in der(1)t(1;1)(p36.3;q21) in AML (Noguchi et al.,2007), raising the possibility that PRDM16 may be activated by chromosomal rearrangements involving genes on 1q, at least in some patients.
Other candidate genes that have been mapped in the 1p36 region are tumor suppressor genes that might be deleted as a consequence of the unbalanced rearrangement.
Terminal 1p36 deletions are frequent in both myeloid and lymphoid malignancies, indicating inactivation of tumor suppressor genes may be the common oncogenic mechanism in these diseases (Duhoux et al., 2011).
It is likely, that patients with der(1)t(1;1)(p36;q11-32) undergo mixed duplication and deletion events associated with formation of the unbalanced translocation.

Age / Sex	Disease	der(1)t(1;1)(p36;q11-q32)	Ref.
Chronic myeloproliferative neoplasms			
F/40	CML	47,XX,der(1)t(1;1)(p36;q31),del(3)(p21p23),add(4)(q35),+der(7)add(7)(p22)add(7)(q33),-8, t(9;22)(q34;q11),add(10)(p13),+mar	1
M/68	MDS	47,XY,+11/46,XY,der(1)t(1;11)(p36;q13)	2
F/60	CEL	46,XX,der(1)t(1;1)(p36;q12)/46,XX,der(13)t(1;13)(q12;p13)/46,XX,der(22)t(1;22)(q12;p13)	3
M/48	MDS	46,XY,der(1)t(1;1)(p36;q12)	4
M/75	PV-MF	48,XY,+8,+9/48,idem,der(1)t(1;1)(p36;q12)	5
Acute myeloid leukemia			
F/68	AML-M2	45,XX,der(1)t(1;1)(p36;q11),-5,-7,-11,der(17)t(17;18)(p13;q11),+r,+mar/44,XX,-5,-7,-17,+mar, dmin amplified c-MYC sequences	6
M/64	MDS-AML-M4	46,XY,del(5)(q22q33) At diagnosis 46,XY,r(18) 45,XY,der(1)t(1;11)(p36;q23),-11,dmin During transformation 46,XY,dmin	7
M/0	AML-M5	46,XY,der(1)t(1;1)(p36;q21)/45,idem,-Y/47,idem,+8/46,XY,der(21)t(1;21)(q21;p11)	8
M/3	AML-M5	46,XY,der(1)t(1;1)(p36;q32),-7,+21c/46,idem,del(9)(p22)	9
F/1	AML-M5	46,XX,der(1)t(1;1)(p36;q32),der(6)t(6;8)(p25;q13),+11,der(11;18)(q10;q10)dup(1)(q23q44) Partial trisomy 1q "syndrome"	10
F/60	AML-M7	62,XX,-X,der(1)t(1;1)(p36;q22),-2,-3,-4,-5,-7,der(7)t(1;7),+8,-9,-11,-13,-14,der(14)t(5;7;14), der(14)t(14;22)(p11;q11),+15,-16,-20,-21	11
M/25	AML-M5a	46,XY,der(1)t(1;1)(p36;q21) DUSP10-PRDM16 fusion	12
F/1	AML	46,XX,der(1)t(1;1)(p36;q21) Telomeric 1p36 probes deleted	13
M/1	AML	47,XY,der(1)t(1;1)(p36;?q31),+21c Telomeric 1p36 probes deleted	14
M/65	AML	45,XY,der(1)t(1;1)(p36;q25),-5,-5,-9,add(17)(p13),der(21)t(5;21)(q11;q22),+2mar	15
F/65	AML-M5	46,XX,der(1)t(1;1)(p36;q22) Telomeric 1p36 probes deleted	16
M/60	AML-M4	46,XY,der(1)t(1;1)(p36;q12)	17
F/1	AML	47,XX,der(1)t(1;1)(p36;q21),t(5;6)(p15;p23),+21c	18
F/50	AML-M5	46,XX,der(1)t(1;1)(p36;q12),t(9;11)(p22;q23)†	19
Acute lymphoblastic leukemia			
M/4	T-ALL	46,XY,der(1)t(1;1)(p36;q21),del(4)(q31),add(12)(q24),t(14;18)(q22;q21)	20
M chi	ALL	46,XY,der(1)t(1;1)(p36;q21),t(9;22)(q34;q11)	21
F/0	B-ALL	46,XX,der(1)t(1;1)(p36;q11),ins(11;2)(q23;q11q11) MLL-LAF4 fusion	22
M/**12**	B-ALL	55,XY,der(1)t(1;1)(p36;q21),+4,+4,+6,+10,+14,+17,+18,+21,+21/56,idem,+8	23
M	B-ALL Post BMT relapse	45-46,XY,der(1)t(1;1)(p36;q21),t(3;5;10)(p13;q34;p13),t(6;21)(q21;q22), der(8)t(8;8)(p21;q23),-9,t(9;22),-17/45-46,XY,der(1),t(3;16)(p13;q22),add(4)(q22),t(5;14)(q35;q11), der(8),-9,t(9;22),add(10)(q24),add(11)(p11),-17,+der(22)t(9;22)x2	24

M/3	B-ALL	55,XY,der(1)t(1;1)(p36;q12),add(2)(p?),+4,+6,+10,+14,+16,+18,+21,+21,+mar/63-64,idem,+X,+5,+9,+11,+12,+15,+17,+19,+22 Telomeric 1p36 probes deleted	25
M/65	B- ALL	50-51,X,+X,-Y,der(1)t(1;1)(p36;q32),der(1)t(1;12)(q32;q15),der(4)del(4)(q21q25)t(4;6)(p16;q13),i(6)(p10),+7,t(8;9)(q24;p13),der(10)t(8;10)(q24;p13),+11,der(12)t(12;18)(q24;q21)t(14;18)(q32;q21)x2,dup(13)(q22q34)x2,t(14;18),der(17)t(8;17)(?;p13),der(18)t(10;18)(p?13;q?21), der(18)t(14;18)x2 t(14;18)(q32;q21)	26
M/83	T-ALL	46,X,-Y,+del(X)(q26),der(1)t(1;1)(p36;q12)del(1)(q11q21),t(3;22)(q27;q11)/47,idem,+5 L N	27
Various hematological neoplasms			
F/43	B-PCL	41,X,-X,der(1)t(1;1)(p36;q22),t(5;8)(q14;q24),del(6)(q11), der(8)t(8;14)(p21;q11),-13,-14,-16,-18,-20,-20,+mar†	28
F/53	MM	42-44,XX,del(1)(p32p34),der(1)t(1;1)(p36;q12),del(5)(p15),-8,-11,-12,-13,der(17)t(17;21)(p11;q11),-19,-20,-22,+5-6mar	29
M/57	CLL	44,X,Y,der(1)t(1;11)(p36;q13),der(2)t(2;6)(q33;q23),dic(3;6)(q10;p10),der(5)del(5)(p14)del(5)(q31),der(7)t(Y;7)(q11;q21),der(8)t(8;11)(q22;?),del(11)(q12),der(12;14)t(12;14)t(11;14;12)(q13;q32q10;q13),der(12)t(8;12),der(13)t(11;13)(q21;q34),der(17)t(17;18)(p13;q11),-18	30
F/46	MM	45,XY,del(1)(p12p22),der(1;16)(q10;p10),+der(1)t(1;1)(p36;q25),del(2)(q31),del(5)(q31),add(9) (p24),der(12)t(1;12)(q12;q24),-13,del(14)(q22),add(17)(p13),+19,add(21)(p13),-22/46,idem,+18	31
M/70	MM	45,XY,del(1)(p13p32),-8,-13,der(14)t(1;14)(q12;q21),-20,+r,+mar/46,XY,del(1)(q25q43),der(1),t(1;1)(p36;q21)del(1)(p11p32),ins(7;?)(q31;?),del(8)(q12),-13,der(14)t(1;14),-20,+2mar	32
M	MM	42-47,X,der(Y)t(Y;1)(q12;q12),del(1)(p12p22),der(1)t(1;1)(p36;q12)dup(1)(q12q44),t(4;5)(p16;p13),t(8;22)(q24;q11),+12,del(12)(q14),-13,-14/53-63, idem,+1,-der(1),add(2)(q37),+3,+5, +6,+7,+t(8;22),+9,+11,-14,+16,+20,inc†	33
F	MM	46,X,-X,add(1)(q42),add(1)(p22),der(2)t(2;13)(p24;q12),+3,del(3)(p12p21),add(4)(p11),del(5)(p13),+6,add(6)(q12),add(9)(q32),+11,der(12)add(12)(p13)add(12)(q24),der(12)add(12)(p11)? dup(12)(q22q24)add(12)(q24),-13,-13,+15,del(17)(p11),+19,add(19)(p13),add(19)(p13)/46-47, idem,add(15)(p13),-add(19)(p13)/48-49,idem,+X,-add(1)(p22),+der(1)t(1;1)(p36;q12),+2,der(2)t(2;13),add(3)(q27),del(7)(q32),add(9),+del(9)(q12),+12,der(12),der(12),+add(12)(p13), t(12;13)(q24;q12),+del(?13)(q12q22),-14,-add(19)(p13)†	34

Table 1. Reported patients with der(1)t(1;1)(p36;q11-32).
Abbreviations: Ref., references; M., male; F., female; CML., chronic myeloid leukemia; MDS., myelodysplastic syndrome; CEL., chronic eosinophilic leukemia; PV., polycythemia vera; MF.,myelofibrosis; acute myeloid leukemia; AML-M2., acute myeloblastic leukemia with maturation., AML-M4., acute myelomonocytic leukemia., AML-M5., acute monoblastic leukemia; AML-M7., acute megakaryoblastic leukemia AML., acute myeloid leukemia; ALL., aAcute lymphoblastic leukemia/lymphoblastic lymphoma; BMT., bone marrow transplantation; PCL., plasma cell leukemia; MM., multiple myeloma; CLL., chronic lymphocytic leukemia. 1.Phan et al., 2006; 2.Caramazza et al., 2010; 3-5, 13-17,25.Duhoux et al., 2011; 6.Fugazza et al., 1997; 7.Michalova et al., 1999; 8.Chessells et al., 2002; 9.Picos-Cardenas et al., 2002; 10.Scrideli et al., 2005; 11.Preiss et al., 2006; 12.Noguchi et al., 2007; 18.Blink et al., 2012; 19.Sarova et al., 2013; 20.Shikano et al.,1994; 21.Uckun et al., 1998; 22.Bruch et al., 2003; 23.Paulsson et al., 2010; 24.Schmidt-Hieber et al 2010; 26.Subramaniyam et al., 2011; 27.Safavi et al., 2015; 28.Bastard et al.,1991; 29.Rajkumar et al., 1999; 30.Nordgren et al., 2000; 31.Bang et al., 2006; 32.Vekemans et al., 2010; 33-34.Sawyer et al., 2014.

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