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Review

dup(1)(q11-q44) in myeloid malignancies

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Abstract

Review on dup(1)(q11-q44) in myeloid malignancies, with data on clinics

Clinics and pathology

Disease

Myelodysplastic/myeloproliferative neoplasms (MDS/MPN) and acute myeloid leukemia (AML).

Phenotype/cell stem origin

Chronic myeloproliferative disorder in 58 cases and AML in 52 patients.

Chronic myeloproliferative neoplasms: 5 polycythemia vera (PV) (Carbonell et al., 1983; Mamaeva et al., 1983; Rege-Cambrin et al., 1987; Jarosova et al., 1988), 3 essential thrombocythemia (ET) (Knuutila et al., 1983; Richard et al., 1987; Lofvenberg et al., 1990), 10 myelofibrosis (MF) (Schmid and Kohler., 1984; Lawler and Swansbury.,1985; Akahoshi et al., 1987; Fonatsch and Gradl., 1988; Lofvenberg et al., 1990; Andrieux et al., 2003), among them 4 with post-PV myelofibrosis (Andrieux et al 2003) and 2 after therapy for a previous malignancy (Lawler and Swansbury., 1985; Akahoshi et al., 1987). There were 2 chronic myelomonocytic leukemia (CMMoL) (Fonatsch et al., 1991; Trakhtenbrot et al., 2010) cases and 2 patients with unspecified chronic myeloproliferative disorders (Haubenstock et al., 1985; Kerman et al., 1986).

Myelodysplastic syndrome: 25 patients (Anderson and Bagby., 1982; Papenhausen et al., 1984; Yunis et al., 1986; Palmer et al., 1987; Fonatsch et al., 1991; Kanamaru and Tamura., 1993; Ohyashiki et al., 1994; Tien et al., 1994; Satake et al., 1997; Alter et al., 2000; de Souza Fernandez et al., 2000; Kearns et al., 2004; Alfaro et al., 2008; Bacher et al., 2009; Vundinti et al., 2010; Kolquist et al., 2011; Quentin et al., 2011), 8 of them with Fanconi anemia (FA) (Alter et al 2000; Vundinti et al 2010; Quentin et al., 2011).

Chronic myeloid leukemia (CML) was diagnosed in 11 patients (Kohno and Sandberg., 1980; Smadja et al., 1984; Hild and Fonatsch., 1990; Gozzetti et al., 2003; Herens et al., 2003; Lee et al., 2003; Schoch et al., 2003; Abruzzese et al., 2007; Palandri et al., 2009; Sun et al., 2011; Fabarius et al., 2011). In 8 patients dup(1q) was found in association with t(9;22)(q34;q11) (Kohno and Sandberg 1980; Smadja et al., 1984; Hild and Fonatsch., 1990; Lee et al., 2003; Schoch et al., 2003; Palandri et al., 2009; Sun et al., 2011; Fabarius et al., 2011) and it was detected in a Ph-negative clone while on imatinib therapy in 3 patients (Gozzetti et al., 2003; Herens et al., 2003; Abruzzese et al., 2007).

Acute myeloid leukemia: There were 53 patients diagnosed with various forms of AML: 1 AML-M0 (Angelova et al., 2015), 4 AML-M1 (Pui et al., 1990; Tanaka et al., 1997; Taylor et al., 2000; Jekarl et al., 2010), 9 AML-M2 (CG et al., 1988; Tien et al., 1988; Kobayashi et al., 1990; Stuppia et al., 1990; Kudoh et al., 1995; Tamura et al., 1998; Lee et al., 2004; Kim et al., 2009; Beach et al., 2012), 3 AML-M3 (Berger et al., 1988; Cuneo et al 1992; Batzios et al., 2009), 7 AML-M4 (Haas et al., 1985; Misawa et al., 1988; Raynaud et al., 1994; Hda et al., 1996; Forestier et al., 2003; Schmidt et al., 2004; Bao et al., 2006), 5 AML-M5 (Orazi et al., 1988; Soekarman et al., 1992; Harrison et al., 1998; Panagopoulos et al., 2000; Gmidene et al., 2012), 2 AML-M6 (Cuneo et

al., 1990; Creutzig et al., 1996), 5 AML-M7 (Shikano et al., 1995; Creutzig et al., 1996; Alvarez et al., 2001; Strehl et al., 2001; Lundin et al., 2012) and 16 AML unspecified cases (Nacheva et al., 1982; Ghaddar et al.,1994; Nacheva et al., 1995; Kolstad et al., 1996; Pallisgaard et al., 1998; Raimondi et al., 1999; Alter et al., 2000; Blann et al., 2000; de Souza Fernandez et al., 2000; Clavio et al., 2001; Seiter et al., 2001; Kern et al., 2002; Oliveira et al., 2002; Quentin et al., 2011).
Patient's characteristics are reported in Table 1.

Epidemiology

At least 110 reported cases in myeloid malignancies (63 males and 47 females, aged 0 to 84 years; median 45 years); male prevalence in chronic myeloid neoplasms (35M/23F, aged 1 to 84 years; median 51 years); balanced sex ratio in AML (28M/24F aged 0 to 72 years; median 33 years). There were 20 pediatric cases (9M/11F;
There were 8 therapy-related myeloid malignancies developing in patients after cytotoxic and/or radiation therapy for the primary disease: 3 MPN (Lawler and Swansbury1985; Haubenstock et al., 1985; Akahoshi et al., 1987) and 5 AML (Orazi et al., 1988; Pui et al., 1990; Tanaka et al., 1997; Taylor et al., 2000; Schmidt et al., 2004). Also, there was an emergence of cytogenetically abnormal clones with dup(1q) in Ph-negative cell population in CML (Herens et al., 2003; Gozzetti et al., 2003; Abruzzese et al., 2007) and AML patients while on complete remission (Raynaud et al., 1994; Kudoh et al., 1995; Seiter et al., 2001; Batzios et al., 2009., Beach et al., 2012).
Given that these patients had previously received therapy, the role of cytotoxic treatments in these cases cannot be excluded.

Prognosis

The prognosis is likely variable in myeloid malignancies as dup(1q) may be present at all stages in the bone marrow, including hypoplastic marrow without morphological evidence of transformation.
The appearance of dup(1q) in association with leukaemic or myelofibrotic transformation and/or poor-risk genetic features may be predictive of inferior prognosis (Beach et al., 2012).

Cytogenetics

Cytogenetics morphological

Presents as one normal chromosome 1 and a dup(1)(q11-q44) chromosome. The breakpoints in 1q varied from 1q11 to 1q44, with a clustering to 1q21q32 (19 out of 110 cases) and 1q21q42, being duplicated in 13 out of 110 patients.

Additional anomalies

Sole abnormality in 37 patients, mostly in chronic myeloproliferative disorders (3 PV, 3 ET, 6 MF, 1 CMMoL, 14 MDS and 3 CML), and less frequently in AML (7 cases). Associated in combination with an extra chromosome 8 in 11 (Papenhausen et al., 1984;Ohyashiki et al., 1994; Alfaro et al., 2008; Schoch et al., 2003; CG., 1988; Tien et al.,1988; Cuneo et al., 1992; Ghaddar et al., 1994; Creutzig et al., 1996; Tamura et al., 1998; Bao et al., 2006). Loss of chromosome 7 was observed in 3 AML patients (Haas et al., 1985; Hda et al., 1996; Ghaddar et al., 1994) and del(7q) in 4 cases, 3 of them with Fanconi anemia (Alter et al., 2000; Kearns et al., 2004; Quentin et al., 2011). del(5q) was found in 2 AML (Soekarman et al., 1992; Tanaka et al., 1997) and del(20q) was present in 3 (Trakhtenbrot et al., 2010; Kobayashi et al., 1990; Nacheva et al., 1995) cases.

Sex/Age	Disease	Karyotype	
Chronic myeloproliferative disorders			
F	MDS	46,XX,dup(1)(q23q44)	1
M	PV	46,XY,dup(1)(q21q31)	2
M/36	ET	46,XY,dup(1)(q21q32)	3
F/65	PV	46,XX,dup(1)(q21q44),inv(1),inv(9)	4
M/54	MDS	47,XY,dup(1)(q21q32),+8	5
F/45	IMF	46,XX,dup(1)(q21q32)	6
F/84	CMPD	46,XX,dup(1)(q21q32) Therapy for ovary carcinoma	7
F/39	IMF	46,XX,dup(1)(q21q32 Therapy for Hodgkin disease	8
F/69	CMPD	46,XX,dup(1)(q22q31),der(9)t(9;17)(q11;q11),inv(11),-17	9
M/59	RAEB	46,XY,dup(1)(q21q42)	10
F/60	IMF-AML	46,XX,dup(1)(q21q42),t(3;21)(q26;q22) Therapy for breast carcinoma	11
F/29	RAEB1	46,XX,dup(1)(q11q42),dup(6)(p21p25),t(11;16)(q14;p13)	12
F/33	PV	46,XX,ins(11;4)(p14;q26q35)/46,XX,dup(1)(q21q44)	13
M/66	PV	46,XY,dup(1)(q21q44)	14
F/75	ET	46,XX,dup(1)(q11q44)	15

F/51	PV	46,XX,dup(1)(q11q44)	16
M/73	IMF	46,XY,dup(1)(q25q44)	17
F/43	IMF	47,XX,dup(1)(q11q42)	18
M/76	ET	46,XY,dup(1)(q11q42)	19
F/46	RA	46,XX,dup(1)(q21q32)	20
F/24	RA	46,XX,dup(1)(q12q42)	21
M/79	CMMoL	46,XY,dup(1)(q25q42)	22
M/24	RA	46,XY,dup(1)(q23q44)	23
M	RAEB	46,XY,dup(1)(q21q44)/47,idem,+8	24
M	RA	46,XY,dup(1)(q21q42)/92,idemx2	25
M/68	RAEB	45,X,-Y,dup(1)(q21q41)	26
M/1	RA	48,XY,+11,+21/48,idem,dup(1)(q21q42)	27
M	MDS	46,XY,dup(1)(q12-21q24),der(2)t(1;2) FA 46,XY,der(2)t(1;2)(q21;q35-37)/46,idem,dup(1)	28
F	MDS	46,XX,dup(1)(q21q25),dup(1)(q21q42),del(7)(q31),del(11)(q21q25),add(17)(q25),der(20)t(1;20)(q10;q13)46,XX,dup(1)(q21q42),del(7),del(11),der(20)t(1;20 FA	29
M/66	RAEB	46,XY,dup(1)(q12)	30
M/71	PPMF	46,XY,dup(1)(q21q42)/46,idem,del(11)(q21q24)	31
M/66	PPMF	46,XY,dup(1)(q21q44),add(18)(p11)	32
M/77	PPMF	46,XY,dup(1)(q21q32)	33
M/64	PPMF	46,XY,der(6)t(1;6)(q21;p23)/46,XY,dup(1)(q21q42)	34
F	IMF	46,XX,dup(1)(q21q42)	35
M	MDS	46,XY,dup(1)(q41q44),del(7)(q31q35)	36
F/51	RCMD-MDS AML	46,XX,dup(1)(q21q32) / 47,XX,dup(1),+8	37
M/62	RAEB2	47,XY,+1/46,XY,dup(1)(q21q32)	38
M/58	RAEBS	46,XY,dup(1)(q32q21)	39
M/65	Post PV-CMMoL	46,XY,t(14;20;18)(q11;q11;p11)/46,idem,del(13)(q12q22)/48,XY,dup(1)(q22q23),del(20)(q11),+21,+mar	40
M/14	RAEB	46,XY,dup(1)(q12q24) FA	41
M	MDS	46,XY,dup(1)(q21q32)x2	42
M/19	RCMD	46,XY,dup(1)(q21q42)/46,idem,der(1)t(1;3)(p36;q21) FA	43
M/32	MDS	46,XY,dup(1)(q10q43) FA	44
M/29	MDS	45,X,-Y,dup(1)(q24q42) FA	45
M/9	RAEB1	46,XY,dup(1)(q21q41)/46,idem,add(11)(q22),add(19)(p13)/46,idem,del(18)(q22) FA	46
F/7	RAEB1	46,X,add(X)(p21),dup(1)(q12q32),dup(3)(q21q26),add(5)(q33),del(7)(q21),-18,+mar FA	47
Chronic myeloid leukemia			
M/40	CML	46,XY,dup(1)(q12q31),t(9;22)	48
M/41	CML BP	46,XY,dup(1)(q25q32),t(6;19),t(9;22),der(21)t(1;21)(q21;q2?2)	49
F/31	CML	46,XX,dup(1)(q23q32),t(9;22)/46,XX,t(1;5)(q21-22;q31),t(9;22)	50
F/77	CML	46,XX,dup(1)(q11q21) Receiving imatinib	51
F/59	CML	46,XX,dup(1)(q21q42) Receiving imatinib	52
F/35	CML	47,XX,dup(1)(q21q44),+6,inv(9)(p11q12),t(9;22)(q34;q11)	53
M	CML BP	47,XY,dup(1)(q21q44),+8,t(9;22) Receiving imatinib	54
M/70	CML	46,XY,dup(1)(q11q21) Receiving imatinib	55
M	CML	45,X,-Y,dup(1)(q21q32),t(9;22)(q34;q11)	56
M/44	CML	46,XY,dup(1)(q31q21),t(9;22)(q34;q11)	57
F /33	CML BP?	46,XX,t(1;2),del(6)(q21),t(9;22)/46,XX,t(9;22),del(18)(p11)/46,XX,dup(1)(q23q32),del(4)(q21),t(9;22),t(11;12)(q23;q13),del(18)(p11),add(19)(p13)	58
Acute myeloid leukemia			

M/0	AML	46,Y,der(X)t(X;11)(q22;q23)ins(X;11)(q22;p15p13),dup(1)(q11q21),der(11)t(X;11)del(11)(p12p15)	59
M/24	AML-M4	46-47,XY,dup(1)(q11q12),-7,t(9;13)(p22;q32)	60
F/2	AML	47,XX,dup(1)(q44q25),+21c DS	61
M/52	AML-M3	46,XY,dup(1)(q11q22),t(15;17)(q22;q12) At relapse	62
M/72	AML-M2	47,XY,del(6)(p23),+8/47,idem,dup(1)(q11q44)/47,idem,dup(1)(q21q32)	63
M/55	AML-M4	46,XY,dup(1)(q12q42	64
M/16	AML-M5b	48,XY,dup(1)(q12),+3,+9 Therapy for osteosarcoma	65
M/9	AML-M2	49,XY,dup(1)(q25q44),+7,+8,t(8;21)(q22;q22),+13,+13,-14,der(22)t(17;22)(q21;p11)	66
M/32	AML-M6	46,XY,dup(1)(q24q44),del(8)(p12),add(9)(q34),del(13)(q14q22),add(16)(p11)	67
M/64	AML-M2	47,XY,+21 / 46,XY,dup(1)(q11q42),del(20)(q11q13)	68
M/6	AML-M1	46,XY,del(1)(p32),dup(1)(q21q32),add(11)(p15)/46,XY,del(1),t(1;11)(p32;q23) Therapy for mature B-cell neoplasm	69
M/66	AML-M2	44,X,-Y,der(1;11)t(1;11)(q10;q21),dup(1)(q24q25),-4,der(17)t(17;21)(p11;p11),-21,+2r	70
M	AML-M3	47,XY,+8,t(15;17)(q22;q21)/47,idem,dup(1)(q31q42)	71
M/67	AML-M5	45,XY,+X,dup(1)(q21q31),del(5)(q12q34),t(6;9)(q23;q34),8,add(9)(p?),del(14)(q21q31),-15	72
F/64	MDS-AML	47,XX,+8/47,idem,dup(1)(q25q42)/45,idem,+2,-5,del(6)(q21),-7,-17,-18,+mar,dmin	73
M/46	AML-M4	46,XY,dup(1)(q21q32) At complete remission	74
M/58	AML-M2	46,XY,t(8;21)(q22;q22)/45,idem,-Y At diagnosis 46,XY,del(11)(q14q23)/46,idem,dup(1)(q21q44) At complete remission	75
M/23	AML	46,XY,del(20)(q11q13)/46,idem,dup(1)(q23q32),hsr(3)(q27)	76
F/2	AML-M7	47,XX,i(7)(q10),+der(11)t(11;14)(p13;q11),-14,+21/47,idem,dup(1)(q21q24)	77
F/2	AML-M6	49,X,t(X;13)(q11;p11),dup(1)(q25q43),+8,+10,+21c/49,idem,del(9)(p22)	78
M	AML	46,XY,dup(1)(q21q44),add(14)(q?32)	79
F	AML-M4	46,XX,del(1)(q21q44),-7,add(12)(p13),dup(12)(q13q21),+mar	80
F/63	AML-M1	44,XX,dup(1)(q25q44),del(4)(q25q28),del(5)(q13q31),i(11)(q10),der(12)t(11;12)(q23;p12),del(13)(q12q14),-14,-17,add(19)(q13),der(19)t(?11;19)(q13;?q13)ins(19;?)(?q13;?) Therapy for breast cancer	81
F/1	AML-M5b	46,XX,dup(1)(q44q12),del(6)(q21),del(10)(q23),add(11)(q23)	82
F/40	AML	46,XX,dup(1)(q21q44),add(14)(q?)	83
F/53	AML- M2	47,XX,dup(1)(q21q32),+8	84
F	AML	46,XX,t(12;21)(q12;q21)/46,XX,dup(1)(q21q42),r(7)(p22q31),add(13)(q34),der(21)t(7;21)(q32;q22)	85
F	AML	46,XX,dup(1)(q21q44)/46,idem,t(18;21)(q21;q22)	86
M	AML	46,XY,dup(1)(q12q31)/46,XY,der(18)t(1;18)(q12;p11)/46,XY,del(6)(p21p24) FA 46,XY,add(4)(p15)	87
M/2	AML	48,XY,dup(1)(q23q31),+11,+21c DS	88
F	AML	46,XX,dup(1)(q11q21),t(9;22)(q34;q11)	89
F/56	AML-M5a	46,X,t(X;5)(q27;q21),dup(1)(q32q42),inv(8)(p11q13)	90
F/15	AML-M1	48,XX,dup(1)(q21q24),t(6;11)(q27;q23),+19,+21 Therapy for lymphoma	91
F/3	AML-M7	46-47,XX,dup(1)(q32q44),t(1;5)(q32;p15),der(14;21)(q10;q10),+21c DS	92
M/52	MDS-AML	46,XY,dup(1)(q21q31),add(11)(q23)	93
F/50	AML	46,XX,dup(1)(q21q41) After therapy for AML	94
M/43	AML	46,XY,t(8;21)(q22;q22) At diagnosis / 46,XY,dup(1)(q21q32) At relapse	95
F/1	AML-M7	47,XX,der(7)t(1;7)(q22;p22),+21c/47,XX,dup(1)(q22q44),+21c DS	96
M	AML	47,XY,+13/47,idem,dup(1)(q23q42) At relapse	97
M/10	AML	46,XY,dup(1)(q21q42) FA	98
M/4	AML-M4	46,XY,t(11;17)(p15;q21)/46,idem,dup(1)(q25q44)/47,idem,+del(1)(p11)	99
F/33	AML-M2	46,XX,t(8;21)(q22;q22)/46,idem,dup(1)(q21q31)	100
F/32	AML-M4	46,XX,t(3;11)(p25;p15),t(8;16)(p11;p13)/46,idem,dup(1)(q11q44) Therapy for ALL	101
M/50	AML-M4	47,XY,+8/47,idem,dup(1)(q23q32)	102

F/37	AML-M3	46,XX,dup(1)(q21q32) At remission	103
F/70	AML-M2	46,XX,dup(1)(q21q42),t(16;21)(p11;q22)/46,XX,der(7)t(1;7)(q21;q35),t(16;21)	104
M/31	AML-M1	46,X,idic(Y)(q12)x2,dup(1)(q12q42),-16,der(21)t(16;21)(p11;q22)/47,idem,+idic(Y)	105
F/5	AML	46,XX,dup(1)(q23q44),add(3)(q22),del(7)(q11q22),der(10),add(15)(q26) FA	106
M/55	AML- M2	45,X,-Y,t(8;21)(q22;q22) At diagnosis / 46,XY,dup(1)(q21q32) At remission	107
F/62	AML-M5	46,XX,dup(1)(q11q21),-2,der(7)(p?),add(14)(q34),+mar	108
F/3	AML-M7	47,XX,dup(1)(p31p35),dup(1)(q32q44),+21c DS	109
M/60	AML-M0	46,XY,dup(1)(q21q32),dmin	110

Table 1. Reported cases with 1q duplication involving 1q11-q44

Abbreviations: M, male; F, female; MDS, myelodysplastic syndrome; PV, polycythemia vera; ET, essential thrombocythemia IMF, idiopathic myelofibrosis; CMPD, chronic myeloproliferative disorder; RA, refractory anemia; RAEB, refractory anemia with excess of blasts; CMMoL, chronic myelomonocytic leukemia; FA, FanconiÃ-s anemia; PPMF, post polycythemia vera myelofibrosis; RCMD, refractory cytopenia with multilineage dysplasia; RAEBS, refractory cytopenia with multilineage dysplasia and ringed sideroblasts; CML, chronic myeloid leukemia; BP, blastic phase; AML, acute myeloid leukemia; AML-M4, acute myelomonocytic leukemia; DS, DownÃ-s syndrome; AML-M3, acute promyelocytic leukemia; AML-M2, acute myeloblastic leukemia with maturation; AML-M5, acute monoblastic leukemia; AML-M6, acute erythroleukemia; AML-M1, acute myeloblastic leukemia without maturation; AML-M7, ALL., acute lymphoblastic leukemia; acute megakaryoblastic leukemia; AML-M0, Acute myeloid leukemia with minimal differentiation.

1.Anderson and Bagby., 1982; 2.Carbonell et al., 1983; 3.Knuutila et al., 1983; 4. Mamaeva et al., 1983; 5.Papenhausen et al., 1984; 6.Schmid and Kohler., 1984; 7. Haubenstock et al., 1985; 8. Lawler and Swansbury.,1985; 9.Kerman et al., 1986; 10.Yunis et al., 1986; 11.Akahoshi et al., 1987; 12.Palmer et al., 1987; 13-14. Rege-Cambrin et al., 1987; 15.Richard et al., 1987; 16.Jarosova et al., 1988; 17. Fonatsch and Gradl., 1988; 18-19.Lofvenberg et al., 1990; 20-22.Fonatsch et al., 1991; 23.Kanamaru and Tamura 1993; 24-25.Ohyashiki et al., 1994; 26.Tien et al., 1994; 27.Satake et al., 1997; 28-29,87.Alter et al., 2000; 30,89.de Souza et al., 2000; 31-34.Andrieux et al., 2003; 35.Andrieux et al., 2004; 36.Kearns et al., 2004; 37-38.Alfaro et al., 2008; 39.Bacher et al., 2009; 40.Trakhtenbrot et al., 2010; 41.Vundinti et al., 2010; 42.Kolquist et al., 2011; 43-47,106.Quentin et al., 2011; 48.Kohno and Sandberg., 1980; 49.Smadja et al., 1984; 50.Hild and Fonatsch 1990; 51.Gozzetti et al., 2003; 52.Herens et al., 2003; 53. Lee et al., 2003; 54. Schoch et al., 2003; 55.Abruzzese et al., 2007; 56.Palandri et al., 2009; 57.Fabarius et al., 2011; 58.Sun et al., 2011; 59.Nacheva et al., 1982; 60.Haas et al., 1985; 61.de Alarcon et al., 1987; 62. Berger et al., 1988; 63.CG., 1988; 64.Misawa et al., 1988; 65. Orazi et al.,1988; 66. Tien et al.,1988; 67. Cuneo et al., 1990; 68.Kobayashi et al., 1990; 69.Pui et al., 1990; 70.Stuppia et al., 1990; 71.Cuneo et al., 1992; 72. Soekarman et al., 1992; 73.Ghaddar et al., 1994; 74.Raynaud et al., 1994; 75.Kudoh et al., 1995; 76.Nacheva et al., 1995; 77.Shikano et al., 1995; 78.Creutzig et al., 1996; 79.Kolstad et al., 1996; 80.Hda et al., 1996; 81.Tanaka et al., 1997; 82.Harrison et al., 1998; 83.Pallisgaard et al., 1998; 84.Tamura et al., 1998; 85-86.Raimondi et al., 1999; 88.Blann et al., 2000; 90.Panagopoulos et al., 2000; 91.Taylor et al., 2000; 92.Alvarez et al., 2001; 93.Clavio et al., 2001; 94-95.Seiter et al., 2001; 96.Strehl et al., 2001; 97.Kern et al., 2002; 98.Oliveira et al., 2002; 99.Forestier et al., 2003; 100.Lee et al., 2004; 101.Schmidt et al., 2004; 102.Bao et al., 2006; 103.Batzios et al., 2009; 104.Kim et al., 2009; 105.Jekarl et al., 2010; 107.Beach et al., 2012; 108. Gmidene et al., 2012; 109.Lundin et al., 2012; 110.Angelova et al., 2015.

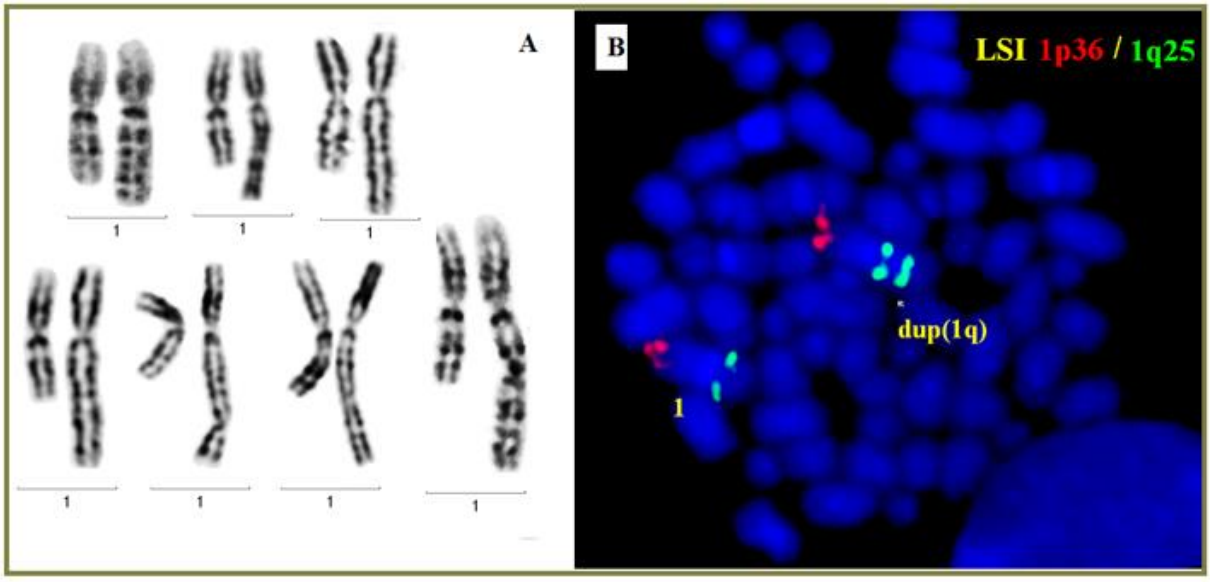

Figure 1. Partial karyotypes showing duplication of the long arm of chromosome 1 (A) and Fluorescence in situ hybridization with LSI 1p36/1q25 probe (Abott, Vysis, USA) showing duplication of 1q25 sequences (green signal) on der(1) chromosome (arrow).

The dup(1q) was apparently secondary anomaly-occurring in a subclone or together with the well-known primary abnormalities such as t(8;21)(q22;q22) in 2 (Tien et al.,1988; Lee et al.,

2004), t(9;22)(q34;q11) in 8 CML (Kohno and Sandberg., 1980; Smadja et al., 1984; Hild and Fonatsch 1990; Lee et al., 2003; 54. Schoch et al., 2003; Palandri et al., 2009; Fabarius et al., 2011; Sun et al., 2011) and 1 AML (de Souza et al., 2000). t(15;17)(q24;q21) was found in 2 cases (Berger et al., 1988; Cuneo et al., 1992), 21q22 abnormalities in 5 (Raimondi et al., 1999; Kim et al., 2009; Jekarl et al., 2010) and 11q23 rearrangements in 4 AML patients (Nacheva et al., 1982; Harrison et al., 1998; Taylor et al., 2000; Clavio et al., 2001).
Cytogenetic clonal variation with karyotypically unrelated clones was found in 5 MPN (Rege-Cambrin et al., 1987; Andrieux et al., 2003; Alfaro et al., 2008; Hild and Fonatsch 1990; Sun et al., 2011) and 2 AML patients (Alter et al., 2000; Strehl et al., 2001).

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

Duplication of all or part of the long arm of chromosome 1 is one of the most frequent chromosomal abnormalities in human neoplasia. In hematologic malignancies of the myeloid lineages, it has been reported in patients with various myeloproliferative conditions, including chronic myeloproliferative neoplasms and acute myelogenous leukemia. Although cytogenetically heterogeneous, duplication of the 1q21-1q32 segment is most commonly observed, indicating that certain chromosome 1 regions might harbor genes that are casually implicated in oncogenesis. The unbalanced nature of this rearrangement indicates, that the sequential duplication of 1q leading to chromosomal imbalances is likely implicated in neoplastic processes by a gene dosage effect. Whether appearance of 1q duplication marks only preleukemic cells participating indirectly, or it may be sufficient as the sole anomaly to promote leukemogenesis is unclear, as well as the role of cytotoxic therapy in some patients.

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