Case Report

Myeloid Antigen-positive T Cell Acute Lymphocytic Leukemia with t(14;18) and Trisomy 10: Report of a Case and Literature Review

Guoqiang Lin MD[#1], Limin Liu MD[#1], Guangsheng Zhao MD[1], Yejun Si MD[1], Xingxia Zhang MD[1], Yumei Sun MD[1], Shuhua Lu MD[1], Yanming Zhang MD•[1]

Abstract

The chromosomal translocation t(14;18)(q32;q21) is commonly associated with neoplasms of follicular center cell origin and has also been reported in cases of chronic lymphocytic leukemia. However, T cell acute lymphoblastic (or lymphocytic) leukemia (T-ALL) with t(14;18)(q32;q21) has been rarely reported. Here, we report a case of myeloid antigen-positive T-ALL (My+T-ALL) with t(14;18)(q32;q21) and trisomy 10. This is the first reported case of My+T-ALL (L2) with such chromosomal abnormalities. Other published *de novo* ALL cases, with t(14;18)(q32;q21) and without a documented history of lymphoma, are summarized and reviewed in this report. The patient in this study was treated with remission induction therapy and intensive chemotherapy, followed by maintenance therapy. As of this writing, he has remained in remission for more than 3 years and has presented a better clinical outcome compared with other reported adult ALL patients with t(14;18)(q32;q21).

Keywords: Chromosomal translocation, chromosomal abnormalities, lymphoma, remission induction therapy

Cite this article as: Lin G, Liu L, Zhao G, Si Y, Zhang X, Sun Y, Lu S, Zhang Y. Myeloid Antigen-positive T Cell Acute Lymphocytic Leukemia with t(14;18) and Trisomy 10: Report of a Case and Literature Review. *Arch Iran Med*. 2015; 18(8): 537 – 541.

Introduction

The translocation t(14;18)(q32;q21), involving the *immunoglobulin heavy* locus (*IGH*) on 14q32 and the *B cell CLL/lymphoma 2* (*BCL2*) locus on 18q21, is often used to identify follicular lymphoma and is sometimes found in diffuse large B cell lymphoma[1-3]. Cases of acute and chronic B cell lymphocytic leukemia carrying this translocation have been reported [1-3]. Due to the translocation, the oncogene *BCL2* is activated by its proximity to the *IGH* enhancer, which promotes the formation of B cell neoplasm; most of the cases carrying this translocation show poor prognosis[4]. However, this translocation has been rarely reported in acute T cell acute lymphoblastic (or lymphocytic) leukemia (T-ALL).

In addition to translocation, aneuploidy (change in chromosomal number) is another common type of chromosomal abnormality among various cytogenetic anomalies described in leukemia. Many kinds of aneuploidy are thought to influence disease progression by contributing growth advantages to cancer cells, and some rare forms are specific for certain morphologic types and clinical features [5]. Trisomy 10 (+10) is often detected in acute myeloid leukemia (AML) in adults and B cell acute lymphocytic leukemia (B ALL) in children; AML and B ALL are considered to be associated with the expression of the molecules CD7 and CD33 in leukemic blasts [6].

T-ALL with +10 is a rare phenomenon. Inoue, *et al*.[7] reported an adult case of biphenotypic leukemia with +10 as the sole abnormality. Here, we describe a morphologically and immunologically proven myeloid antigen-positive T-ALL (My+T-ALL) case in whom t(14;18)(q32;q21) and +10 were identified.

Authors' affiliation: [1]Department of Hematology, Huai'an Hospital Affiliated to Xuzhou Medical College and Huai'an Second People's Hospital, 62 Huaihai South Road, Huai'an, Jiangsu Province, China, 22902.
•Corresponding author and reprints: Yanming Zhang MD, Department of Hematology, Huai'an Hospital Affiliated to Xuzhou Medical College and Huai'an Second People's Hospital, 62 Huaihai South Road, Huai'an, Jiangsu Province, China, 223002
Telefax: +86-0517-80871786; E-mail: zhangyanming2005@126.com
[#]Contributed equally to this article
Accepted for publication: 22 June 2015

Case Report

The patient is a 58-year-old Chinese man who had splenectomy because of trauma. Otherwise, he was free of significant medical history including lymphadenectasis. The patient had fever and fatigue for 10 days in October 2009 and the symptoms persisted after initial antibiotic treatments. Physical examination did not reveal other abnormalities except for an 8-cm scar on the left upper abdomen due to previous splenectomy. Complete blood count on admission showed a white blood cell count of 20.15×10^9/L with 13.8% neutrophils, 77.0% lymphocytes, and some abnormal blasts. Hemoglobin, platelet, and red blood cell counts were 126 g/L, 176×10^9/L, and 4.00×10^{12}/L, respectively.

The bone marrow aspirate revealed that 86.4% of blasts had lymphoblastic morphology, consistent with the criteria for French, American, and British (FAB) sub-type L2. Cytochemically, all stains were negative except for periodic acid-Schiff (medium/fine granular-positive blastic forms 86.4%; Figure 1). The blasts were myeloid antigen-positive T-ALL. Lab results of cell surface markers by Fluorescence Activated Cell Sorting analyses for the patient's bone marrow sample are presented as follows: CD34(44.9%), CD7(81.1%), cCD3(29.9%), CD13(41.1%), CD33 (72.2%), CD2 (10.6%), CD10 (14.1%), CD79 (2.1%), CD19 (2.0%), CD20 (1.4%), HLA-DR (11.1%), CD14 (0.5%), CD15 (2.2%), and CD117 (1.2%). A direct cytogenetic analysis of a bone marrow sample was performed at diagnosis, and showed a normal male karyotype, 46, XY, and an abnormal karyotype, 47, XY, +10, t(14;18)(q32;21), in 40% and 60% of metaphase cells analyzed, respectively (Figure 2). Fusion transcripts were not de-

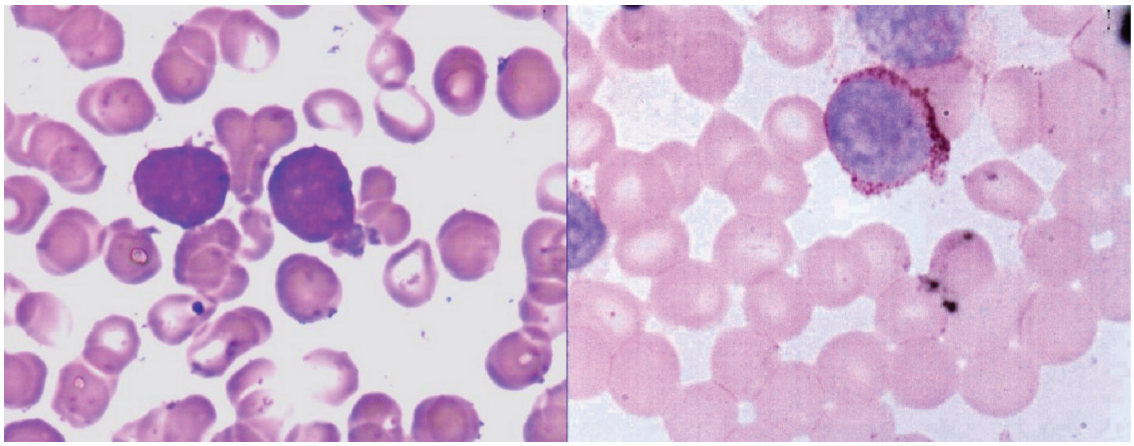

Figure 1. The patient's bone marrow smear. **Left)** Blast cells are negative for peroxidase stain. **Right)** Large blasts and immature cells are positive for periodic acid-Schiff stain. These cells have irregular nuclei, loose chromatin with heterogeneous structures, more cytoplasm with mild basophilia, and one or more prominent nucleoli.

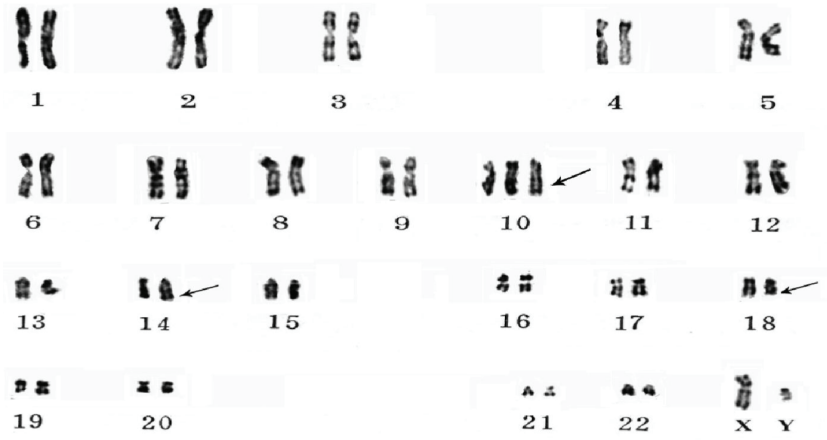

Figure 2. The patient's karyotype. ISCN (International System for Human Cytogenetic Nomenclature) for this abnormal karyotype: 47, XY,+10,t(14;18)(q32;21). The abnormal trisomy chromosome 10 and the abnormal chromosome 14 and 18 involving the t(14;18)(q32;21) translocation are marked with arrow lines.

tected by a multiplex nested RT-PCR for 29 types of leukemia-associated genes. Although the blast cells had both T cell markers (cCD3 and CD7) and myeloid markers (CD13 and CD33), diagnosis of My+T-ALL (L2) was confirmed in accordance with criteria of the FAB and the European Group for the Immunological Characterization of Leukemias (EGIL). The patient achieved morphological remission after the first course of induction chemotherapy with vindesine, daunorubicin, L-asparaginase, and dexamethasone. Thereafter, the patient continued intensive chemotherapy and maintenance therapy with various combinations of anthracyclines, vindesine, dexamethasone, L-asparaginase, etoposide, cyclophosphamide, high-dose methotrexate, cytarabine, and mercaptopurine. The patient did not have central nervous system infiltration of tumor and further received prophylactic treatment with methotrexate, cytarabine, and dexamethasone for four rounds. Regretfully, the patient refused the post-treatment cytogenetic study due to financial considerations. Relapse occurred after a 3-year remission. Fortunately, morphological remission was achieved again after prompt re-induction therapy. The patient has remained in remission since then.

Discussion

The case in our study had morphological features of ALL-L2, based on the criteria of the FAB classification. However, in addition to T cell markers such as cCD3 and CD7, the myeloid antigens CD13 and CD33 were also detected in this case. This suggested a diagnosis of My+T-ALL (L2) according to the FAB and the EGIL criteria. The cytogenetic and chromosomal analyses revealed the coexistence of t(14;18)(q32;q21) and +10. To the best of our knowledge, this is the first report of a case of My+T-ALL(L2) with such chromosomal abnormalities.

The translocation t(14;18)(q32;q21) is commonly associated with malignant lymphoma, especially follicular lymphoma. However, it has also been described in some cases of *de novo* acute B cell lymphoblastic leukemia. The first case of t(14;18)(q32;q21) in ALL was reported in 1983 by Mufti, *et al.*[8] Subsequently, many ALL cases with t(14;18)(q32;q21) have been reported, but none of them were T-ALL. Here, we present a summary of these reports (Tables 1 and 2).

The information summarized in Table 1 shows that most reported ALL cases with t(14;18)(q32;q21) were positive for B cell sur-

Table 1. Reported cases of *de novo* ALL with t(14;18)(q32;q21) and without a documented history of lymphoma.

Ref.	Gender	Age, Y	LNE	BM blast, %	Immunophenotype	Molecular genetics	Diagnosis	Therapy	Follow up (m)
[12]	Male	62	Yes	91	CD19+, CD10+	NA	ALL(L2)	Yes	NA
[8]	Male	21	Yes	91	Ig-k+Anti-u+Anti-J5+Tdt+HLA-DR+	NA	Pre-B ALL(L2)	Yes	2.5
[13]	Female	50	Yes	NA	SigM+,k+, CD3-,CD10+,CD19+, CD20+, HLA-DR+, p53-	*BCL2, BCL6, c-MYC*	B ALL	Yes	NA
[4]	Female	69	No	90.4	CD10,CD19, CD20, HLA-DR, SmIgκ,CD16, CD7, CD2,	*BCL2*, *BCL6 Ig-JH*, *c-MYC*	B ALL (L2)	Yes	10
[14]	Male	37	NA	38	SmIgMI+,CD19+,CD20+,HLA-DR+	NA	ALL (L2)	Yes	12
[14]	Male	73	NA	87	SmIgMk+,CD19+,CD20+,CD10+, HLA-DR+	NA	ALL(L3)	Yes	8
[15]	Female	73	Yes	48 (PB)	CD19+CD22+HLA-DR+CD10+Igλ±	*BCL2*, *Ig-JH*, *c-MYC*	B ALL (L3)	NA	0.25
[16]	Male	67	No	NA	HLA-DR+CD19+CD20+SIg+ IgGk+	NA	ALL(L3)	No	NA
[17]	Female	57	Yes	95(PB)	Anti-J5+HLA-DR+BA-1+	NA	ALL(L3)	Yes	7
[18]	Female	71	No	NA	CD10+CD19+CD20+CD22+HLA-DR+CD45+TdT+IgM+Igλ+	*BCL2*, *Ig-JH*, *c-MYC*	ALL(L3)	No	NA
[18]	Female	60	Yes	NA	CD10+CD19+CD20+HLA-DR+ CD45+TdT+ IgM+Igλ+	*BCL2*, *Ig-JH*, *c-MYC*	ALL(L2)	Yes	NA
[19]	Female	41	NA	98	HLA-DR+,CDl9+,CDl0+,CD20+, CD22+,CD25+.	*BCL-2*, *Ig-JH*, *c-MYC*	ALL(L3)	Yes	10
[20]	Female	64	Yes	21(PB)	CD10+CD19+CD20+SIgκ+	NA	ALL (L1)	Yes	NA
[21]	Female	62	Yes	□70	HLADR+,CD10+,CD19+,CD20+,CD22+,CD38+,CD71+	*BCL2*, *Ig-JH*, *c-MYC*	B-ALL	Yes	NA
[22]	Male	51	Yes	80	CD19+,CD20+,CD22+,SIgk+	*E2A/PBX1*, *BCL2*	ALL(L1)	Yes	5
[22]	Male	40	Yes	95	CD10+CD19+CD20+HLA-DR+	NA	ALL(L2)	Yes	NA
[23]	Male	35	No	100	CD10+CD19+CD24+HLA-DR+	*BCL2*, *c-MYC*	ALL(L3)	Yes	NA
[23]	Male	59	No	85(PB)	CD10+CD19+CD25+HLA-DR+	*BCL3*	ALL(L2)	Yes	1
[24]	Male	26	No	NA	CD10+CD19+CD22+	*IGH/BCL2*	Pre-B	Yes	36
[24]	Male	66	No	NA	NA	*IGH/BCL2*	ALL	Yes	6
[24]	Female	70	NA	NA	CD10+CD19+CD20+CD22+SIg+	*IGH/BCL2*	ALL(L3)	Yes	7
[24]	Male	70	Yes	NA	CD10+CD19+	*IGH/BCL2*	B ALL	Yes	3
[24]	Male	65	Yes	NA	CD20+	NA	ALL(L2)	Yes	10
[24]	Female	57	No	NA	CD10+CD19+	*IGH/BCL2*	Pre-B	Yes	2.5
[24]	Male	60	No	NA	CD10+CD19+CD20+SIg+	*IGH/BCL2 c-MYC*	ALL(L3)	Yes	3
[24]	Male	63	Yes	NA	CD10+CD19+CD20+	*IGH/BCL2 c-MYC*	ALL(L3)	Yes	6
[24]	Female	76	No	NA	CD19+CD22+SIg+	*IGH/BCL2 c-MYC*	ALL(L3)	Yes	6
[24]	Female	59	No	NA	CD10+	*IGH/BCL2 c-MYC*	ALL(L2)	Yes	1.5
[24]	Male	69	Yes	NA	CD10+CD19+CD20+CD22+SIg+	*IGH/BCL2 c-MYC*	ALL(L3)	Yes	1.5
[24]	Female	86	No	NA	CD10+CD19+CD20+CD22+	*IGH/BCL2 c-MYC*	Pre-B	Yes	0.5

BM = bone marrow, LNE = lymph node enlargement and organomegaly, PB = peripheral blood, NA = not available.

face markers such as CD10, CD19, CD20, and CD22. Moreover, most of these cases showed t(14;18)(q32; q21) in association with other cytogenetic abnormalities (Table 2). The median age of these patients is 56.5 years (range 15–86 years). In contrast, the median age of the adult ALL without t(14;18)(q32; q21) ranged from 33 to 37 years. The longest survival time for the patients listed in Table 2 was 36 months. However, the 5-year survival rate of the adult ALL patients (age > 20 years) without t(14;18) was 51.11%. [9]

It is well known that t(14;18)(q32; q21) leads to overexpression of *BCL2* chimeric mRNA and BCL2 protein which promotes B cell survival by blocking apoptosis. Furthermore, BCL2 protein can cooperate with c-Myc, which contributes to tumorigenesis. However, the effect of t(14;18)(q32; q21) on T cell leukemia is still unknown.

Trisomy 10, as a sole cytogenetic abnormality, has rarely been

Table 2. Karyotype results of previous cases of *de novo* ALL with t(14;18)(q32;q21) and without a documented history of lymphoma.

Ref.	Karyotype
[12]	46,XY/47,XY,+X,dup(7)(q22q10),t(14;18)(q32;q21)
[8]	46,XY[2]/46,XY,t(8;14)(q23;q32),t(14;18)(q32;q21)[3]/45,XY,-17,t(8;14)(q23;q32),t(13;17)(q12;p11),t(14;18)(q32;q21)[7]/46,XY,17,+t(13;13)(p11;q13),t(8;14)(q23;q32),t(13:17)(q12;p11),t(14;18)(q32;q21)[2]
[13]	49,XX,dup(2)(p12;p15),t(3.4)(q27;p13),t(8;14;18)q(24;q32;q21),+8,+ider(8),t(8;14;18) (q24;q32;q21),+der (12),t(1;12)(q24;q22)
[4]	45,X,X,t(3;19)(q21;p13),del(6)(q?),der(10),add(10)(p11),add(10)(q22),add(11)(q23),dup(12)(q13q22),t(14;18)(q32;q21),add(16)(q24),der(18)t(14;18)
[14]	46,XY(40%)/47,XY,+8,t(8;22) (q24;q11),t(14;18)(q32;q21)(60%)
[14]	46,XY,t(8;22)(q24;q11),t(14;18)(q32;q21)
[15]	47,XX,+20,t(8;14)(q24;q32),t(14;18)(q32;q21)/46XX,t(14;18)(q32; q21)
[16]	46,XY(10%);47,XY,+ t(8;22) (q24;q1?),12,t(14;18)(q32;q21),+ace(90%).
[17]	46,XX,+X,del(4)(q11),t(4;9)(q?;q?),+8,t(8;22)(q24;q12),del(9)(q11),t(14;18)(q32;q21),+18,-19,+20
[18]	51, XX,t(l;3;11)(q42.3;q27.1;q23.1),+7,t(8;9)(q24.2;q13.3),+12,del(13)(q12.3q21.2),t(14;18)(q32.3;q21.3),add(17)(p13),+20
[18]	47,XX,t(1;3;7)(p32.1;q21.1;q22.l),t(8;9)(q24.2; pl3.3),+12,t(l4;18)(q32.3;q21.3)
[19]	46,XX,t(2;3)(pl2;q27),del(8)(q24),t(l4;18)(q32;q21)
[20]	46,XX,+3,del(3)(p21),der(9)t(9;15)(p11;q15),-10,der(11)t(8;11)(p11;p13),t(14;18)(q32;q21),-16,+der(19)t(1;19)(q12;p13.3)
[21]	47,XY,+1,add(2)(p25),t(8;14)(q24;q32)de1(13)(q2l;q31),t(14;18)(q32;q21),del(17)(p12),t(20;22)(ql3;q11)/47, idem,del(19)(p12)/47,idem, der(19)t(1;19)(q23;p13)
[22]	47,XY,add(1)(p36),add(3)(q23),+7,t(14;18)(q32;q21),t(15;22)(q26;q21),der(19)t(1;19)(q23;p13)
[22]	45,XY,der(1)t(1;13)(q21; q1?)
[23]	46,XY,t(8;14)(q24.1;q32),t(14;18)(q32;q21)/46,XY,1q+,t(8;14)t(14;18)/47,XY,1q+,t(8;14)t(14;18),+mar/46,XY
[23]	46,X,-Y,-9,del(12)(p12),-13,-13,-14,t(14;18)(q32;q21),+mar1,+mar2,+mar3,+mar4,+mar5
	46,XY,t(14;18)(q32;q21)[7]/47,idem,+21[3]/46,XY[27]
[24]	46,XY,add(3)(q28),t(14;18)(q32;q21),add(22)(q12)[15]/46,XY[5]
[24]	50,XX,+5,+8,del(9)(p22),+12,t(14;18)(q32;q21),+mar1[10]/46,XX[17]
[24]	64~71,XY,add(1)(p11),der(1)?t(1;15)(p12;q12),-2,-3,add(4)(p11),add(4)(q12),add(5)(q11),add(7)(p13),-9,add(9)(q34),-10,-11,del(11)(q13q?14),ins(11;?)(q13;?),+13,add(14)(q32),t(14;18)(q32;q21),-15,der(16)t(9;16)(q23;q22),add(17)(p11),der(18)t(14;18),+der(18)t(14;18),-19,der(19)del(19)(p?13)add(19)(q?13),+22,+1~3r,+mar1,+mar2,+mar3,+mar4,+mar5,+mar6[cp20]/46,XY[6]
[24]	45,X,-Y[26]/48~50,XY,hsr(1)(q?21)2,+8,+12,t(14;18)(q32;q21),del(17)(p11)[cp3]/46,XY[22]
[24]	46,XX,add(1)(q41),der(3)t(3;14;?)(q27;q32;?),t(8;14)(q24;q32),der(18)t(14;18)(q32;q21)[28]/92,idem2[3]/46,XX[1]
[24]	50~52,X,-Y,+2,t(2;8)(p12;q24),del(6)(q14),+7,+8,+der(8)t(2;8),add(10)(q24),+11,+13,t(14;18)(q32;q21),?i(17)(q10),+20,-21[cp16]/101~104,idem×2[cp4]
[24]	48,XY,der(3)t(1;3)(q23;q27),del(4)(p12),+i(6)(p10),7,t(8;22)(q24;q11),t(14;18)(q32;q21)[21]/46,XY[1]
[24]	49,XX,+X,+1,i(1)(q10),2,t(8;22)(q24;q11),t(14;18)(q32;q21)[5]/53~57,idem,+X,+5,+7,+der(8)t(8;22),+11,+der(14)t(14;18),+20,+22[cp5]/46,XX[10]
[24]	46,XX,del(6)(q15q21),t(8;9)(q24;p13),ins(12;?)(q13;?),t(14;18)(q32;q21)[21]/46,XX[5]
[24]	48,XY,+7,+11,add(11)(q23)2,t(14;18)(q32;q21)[21]/46,XY[2].isht(8;14)(MYC+,IGH+,BCL2+;IGH+,MYC+)
[24]	47~48,XX,-4,add(9)(p13),+10,+12,-13,t(14;18)(q32;q21),+20,+mar[cp16]/46,XX[32].ishmar(30MYC+)

seen in *de novo* leukemia. It has been mainly detected in AML with an incidence ranging from 0.2% to 0.5% in *de novo* AML,[10] but has been more rarely observed in ALL. One child and one adult pre-B cell leukemia cases with trisomy 10 have been reported. Trisomy 10 was suggested as a good prognostic indicator in childhood and adult ALL and a poor prognostic indicator in childhood AML.[10] In summary, the prognosis of trisomy 10 has been reported to be poor or good for AML, being more favorable in pediatric patients than in adults.

As reported, trisomy 10 may be associated with specific expression of CD7 and CD33 in leukemic blasts.[6,11] In accord with these reports, our case was also positive for CD7 and CD33. There are several genes on trisomy 10 that are associated with tumorigenesis, including BMI1, EST RET, HOXI1 and MX1. The gain of these genes may contribute to the association of trisomy 10 with malignant tumors.[7] Further research is needed to investigate the underlying mechanisms directing the clinical and pathological significance of trisomy 10 in acute leukemia.

The patient in our study experienced a favorable clinical outcome. However, the t(14;18)(q32; q21) translocation has been associated with a poor prognosis for ALL, as discussed above. It is unclear whether the favorable outcome in our case was due to the unique nature of T cell leukemia or the trisomy 10. Nevertheless, the details of our case suggest that prognosis of a known cytogenetic feature varies when associated with other genetic or pathological features, and reminds us of the importance of com-

prehensively considering all case-specific conditions when evaluating prognosis.

In short, we present a rare case of myeloid antigen-positive T-ALL (L2) with the coexistence of t(14;18)(q32;q21) and trisomy 10. A relatively favorable clinical outcome was observed in this case.

Clinical practice points

The chromosomal translocation t(14;18)(q32;q21) is commonly associated with neoplasms of follicular center cell origin and chronic lymphocytic leukemia. Trisomy 10 is often detected in AML in adults and B ALL in children. Here, we report a case of My+T-ALL with t(14;18)(q32;q21) and +10. This is the first reported case of My+T-ALL (L2) with such chromosomal abnormalities. The patient in this study has presented a better clinical outcome compared with other reported adult ALL patients with t(14;18)(q32;q21). It might follow that the t(14;18)(q32;q21) with +10 in My+T-ALL are good prognostic factors, although further research is needed.

Conflict of interest

All authors declare that there is no conflict interests involved

Acknowledgment

The authors thank the technologists of the Cytogenetics Department and Molecular Genetics and Flow Cytometry Department at the First People's Hospital Affiliated to Suzhou University.

This work was supported by the Network Hospital of Clinical Medicine, Science and Technology Project, Jiangsu Science and Technology Department (No. BL2012005), Hematology Academician Workstation of Huaian City (No. HAP201321).

References

1. Greer WL, Lee CL, Callanan MB, Zayed E, Sadek I. Case of acute lymphoblastic leukemia presenting with t(14;18)/BCL2, t(8;14)/cMYC, and t(1;2)/FCGR2B. *Am J Hematol*. 2003; 74(2): 112-8.
2. Hardianti MS, Tatsumi E, Syampurnawati M, Furuta K, Suzuki A, Saigo K, et al. Presence of somatic hypermutation and activation-induced cytidine deaminase in acute lymphoblastic leukemia L2 with t(14;18)(q32;q21). *Eur J Haematol*. 2005; 74(1): 11-9.
3. Kern W, Haferlach T, Schnittger S, Schoch C. Detection of t(14;18)(q32;q21) in B-cell chronic lymphocytic leukemia. *Arch Pathol Lab Med*. 2005; 129(3): 410-1.
4. Saigo K, Kumagai S, Kogi M, Imoto S, Hamano K, Morita S, et al. Acute B-cell lymphoblastic leukemia with t(14;18): establishment of a new cell line. *Cancer Genet Cytogenet*. 2003; 140(2): 174-5.
5. Suzuki A, Kimura Y, Ohyashiki K, Kitano K, Kageyama S, Kasai M, et al. Trisomy 10 in acute myeloid leukemia. Three additional cases from the database of the Japan Adult Leukemia Study Group (JALSG) AML-92 and AML-95. *Cancer Genet Cytogenet*. 2000; 120(2): 141-3.
6. Czepulkowski B, Powell AR, Pagliuca A, Mufti GJ. Trisomy 10 and acute myeloid leukemia. *Cancer Genet Cytogenet*. 2002; 134(1): 81-3.
7. Inoue T, Fujiyama Y, Kitamura S, Andoh A, Hodohara K, Bamba T. Trisomy 10 as the sole abnormality in biphenotypic leukemia. *Leuk Lymphoma*. 2000; 39(3-4): 405-9.
8. Mufti GJ, Hamblin TJ, Oscier DG, Johnson S. Common ALL with pre-B-cell features showing (8;14) and (14;18) chromosome translocations. *Blood*. 1983; 62(5): 1142-6.
9. Xie Y, Davies SM, Xiang Y, Robison LL, Ross JA. Trends in leukemia incidence and survival in the United States (1973-1998). *Cancer*. 2003; 97(9): 2229-35.
10. Sakai Y, Nakayama H, Matsuzaki A, Nagatoshi Y, Suminoe A, Honda K, et al. Trisomy 10 in a child with acute nonlymphocytic leukemia followed by relapse with a different clone. *Cancer Genet Cytogenet*. 1999; 115(1): 47-51.
11. Morgan R, Chen Z, Stone JF, Cohen J, Gustafson E, Jolly PC, et al. Trisomy 10: age and leukemic lineage associations. *Cancer Genet Cytogenet*. 1996; 89(2): 173-4.
12. Borrego S, Antinolo G, Parody R, Jimenez F. Translocation (14;18) in a patient with common acute lymphoblastic leukemia (FAB l2). *Cancer Genet Cytogenet*. 1993; 65(2): 177-8.
13. Stamatoullas A, Buchonnet G, Lepretre S, Lenain P, Lenormand B, Duval C, et al. De novo acute B cell leukemia/lymphoma with t(14;18). *Leukemia*. 2000; 14(11): 1960-6.
14. Thangavelu M, Olopade O, Beckman E, Vardiman JW, Larson RA, McKeithan TW, et al. Clinical, morphologic, and cytogenetic characteristics of patients with lymphoid malignancies characterized by both t(14;18)(q32;q21) and t(8;14)(q24;q32) or t(8;22)(q24;q11). *Genes Chromosomes Cancer*. 1990; 2(2): 147-58.
15. Brito-Babapulle V, Crawford A, Khokhar T, Laffan M, Matutes E, Fairhead S, et al. Translocations t(14;18) and t(8;14) with rearranged bcl-2 and c-myc in a case presenting as B-ALL (L3). *Leukemia*. 1991; 5(1): 83-7.
16. Smith SR, Bown N, Wallis JP. Acute lymphoblastic leukemia of Burkitt type (L3) with a (14;18) and an atypical (8;22) translocation. *Cancer Genet Cytogenet*. 1992; 62(2): 197-9.
17. Gluck WL, Bigner SH, Borowitz MJ, Brenckman WD, Jr. Acute lymphoblastic leukemia of Burkitt's type (L3 ALL) with 8;22 and 14;18 translocations and absent surface immunoglobulins. *Am J Clin Pathol*. 1986; 85(5): 636-40.
18. Nacheva E, Dyer MJ, Fischer P, Stranks G, Heward JM, Marcus RE, et al. C-MYC translocations in de novo B-cell lineage acute leukemias with t(14;18)(cell lines Karpas 231 and 353). *Blood*. 1993; 82(1): 231-40.
19. Berger R, Flexor M, Le Coniat M, Larsen C-J. Presence of three recurrent chromosomal rearrangements, t (2; 3)(p12; q37), del (8)(q24), and t (14; 18), in an acute lymphoblastic leukemia. *Cancer Genet Cytogenet*. 1996; 86(1): 76-9.
20. Kouides PA, Phatak PD, Wang N, Bennett JM. B-cell acute lymphoblastic leukemia with L1 morphology and coexistence of t(1;19) and t(14;18) chromosome translocations. *Cancer Genet Cytogenet*. 1994; 78(1): 23-7.
21. Wlodarska I, Stul M, De Wolf-Peeters C, Verhoef G, Mecucci C, Cassiman JJ, et al. t(1;19) without detectable E2A rearrangements in two t(14;18)-positive lymphoma/leukemia cases. *Genes Chromosomes Cancer.* 1994; 10(3): 171-6.
22. Rowe D, Devaraj PE, Irving JA, Hogarth L, Hall AG, Turner GE. A case of mature B-cell ALL with coexistence of t(1;19) and t(14;18) and expression of the E2A/PBX1 fusion gene. *Br J Haematol*. 1996; 94(1): 133-5.
23. Kramer MH, Raghoebier S, Beverstock GC, de Jong D, Kluin PM, Kluin-Nelemans JC. De novo acute B-cell leukemia with translocation t(14;18): an entity with a poor prognosis. *Leukemia*. 1991; 5(6): 473-8.
24. D'Achille P, Seymour JF, Campbell LJ. Translocation (14;18)(q32;q21) in acute lymphoblastic leukemia: a study of 12 cases and review of the literature. *Cancer Genet Cytogenet*. 2006; 171(1): 52-6.