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CASE REPORT

SIL-TAL1-Positive Adult T-ALL with t(11;14)(p15;q11.2): A Rare Case Report Highlighting Prognostic Challenges and Treatment Implications

Siyu Chen[1,2,*], Shengwang Wu[1,2,*], Nan Li[1,2], Xing Qiang[1,2], Yongjie Tang[1,2], Yimei Feng[1,2], Cheng Zhang[1,2], Xiangui Peng[1,2], Shuiqing Liu[1,2], Xi Zhang[1–3]

[1]Medical Center of Hematology, Xinqiao Hospital of Army Medical University, Chongqing, 400037, People's Republic of China; [2]Chongqing Key Laboratory of Hematology and Microenvironment, Chongqing, 400037, People's Republic of China; [3]Jinfeng Laboratory, Chongqing, 401329, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xiangui Peng, Email pxgpxg1964@tmmu.edu.cn; Shuiqing Liu, Email liushuiqing062@tmmu.edu.cn

Abstract: T-cell acute lymphoblastic leukemia (T-ALL) with coexisting SIL-TAL1 fusion and t(11;14)(p15;q11.2) is exceedingly rare. There are limited data on the clinical course and outcomes of such patients. We report a case of a 19-year-old male presenting with aggressive T-ALL harboring these abnormalities, along with NOTCH1 and PTEN mutations. SIL-TAL1 positivity is associated with poor prognosis in T-ALL. Despite achieving remission with intensive chemotherapy, the patient experienced rapid relapse and poor overall survival, reflecting the ineffectiveness of conventional treatments. The findings highlight the synergistic role of SIL-TAL1 and t(11;14) in disease progression and underscore the urgent need for targeted therapies and immunotherapies to improve outcomes in such high-risk cases.

Keywords: T-cell acute lymphoblastic leukemia, SIL-TAL1, t(11,14)(p15,q11.2), NOTCH1, PTEN, targeted therapy

Introduction

T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive hematological malignancy derived from the malignant transformation of T-cell precursors that accounts for 25% of adult ALL case.[1] Various studies reported that adult patients with T-ALL had low rates of complete remission (CR) and short median overall survival, which can range from 11 to 17 months.[2] SIL-TAL1 fusion gene, which is formed by the 90 kb-interstitial-deletion TAL1 gene fusing with the 5' region of its neighboring gene SIL, is one of the common chromosomal abnormalities in T-ALL with an incidence rate of 16% to 29%, but with low incidence in adult patients.[3,4] The formation of SIL-TAL1can cause the aberrant expression of TAL1 (also known as SCL, TCL5) protein, which is a member of the class II basic helix-loop-helix (bHLH) family transcription factor. Normally, TAL1 regulates myeloid cell differentiation and proliferation, and determines the direction of hematopoietic stem cell differentiation, while the ectopic expression of TAL1 are considered strong drivers in T-ALL and can engage with a large number of protein partners to regulate activating and repressive transcriptional activities.[5,6] Studies have shown that T-ALL patients with SIL-TAL1-positive usually show high levels of leukocytes, higher risk of tumor lysis syndrome and disseminated intravascular coagulation, and have a higher rate of early deaths.[7] However, the clinical characteristics of T-ALL patients with SIL-TAL1-positive are similar to those of adult T-ALL patients with SIL-TAL1-negative, and it is difficult to distinguish through clinical symptoms, bone marrow morphological examination, chromosome karyotype analysis, and immunophenotyping. Therefore, molecular genetics assays are important to distinguish them.

Moreover, about 50%–70% of T-ALL patients possess abnormal karyotypes, such as t(1;14)(p32;q11), t(7;11)(q34;p15), t(10;14)(q24;q11), t(7;10)(q35;q24), and t(11;14)(p13;q11). Among them, t(11;14)(p15;q11.2) is a relatively common

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chromosomal abnormality that can lead to high-level expression of LIM domain only 1 (LMO1, also called Ttg1 and Rbtn1).[8,9] LMO1, a member of LMO proteins (LMO1–4) which contain two tandem LIM domains that mediate protein–protein interactions, is frequently overexpressed in T-ALL.[10–12] Herein, we report a rare case of adult T-ALL patient with both SIL-TAL1-positive and t(11;14)(p15;q11.2). Next-generation sequencing revealed that this also patient carried NOTCH1 and PTEN gene mutations. Unfortunately, the patient passed away due to the poor therapeutic effects of chemotherapy. Targeted therapy, immunotherapy, novel adjuvant chemotherapeutics, and their combinations hold promise to improve clinical prognosis for such patients.

Case

A 19-year-old man patient who presented with cough for more than 1 month and fever for 20 days visited our hospital in September, 2022. Neck ultrasound revealed bilaterally enlarged lymph nodes with abnormal structures in the region IV and V. Then, the patient was initially admitted to the Respiratory Department of our hospital. Subsequently, bone marrow puncture and other examinations were performed for further diagnosis. The results of routine blood examination were as follows: leukocytes 100.69×10^9/L (reference range $3.5–9.5\times10^9$/L), neutrophil percentage 11.3% (reference range 50–70%), lymphocyte percentage 78.3% (reference range 20–50%), platelets 59×10^9/L (reference range $100–300\times10^9$/L). Bone marrow smears revealed active bone marrow hyperplasia with many primitive and immature lymphocytes (94.5%) (Figure 1A and B). The lymphocytes were medium in size, with little cytoplasm, granular chromatin, and visible nucleoli. Chemical staining results showed that peroxidase (POX) stain is negative and periodic acid-Schiff (PAS) stain is positive (Figure 1C and D). These findings implied that the patient might be diagnosed with ALL. Bone marrow biopsy displayed abnormal proliferation of lymphocytes with diffuse distribution

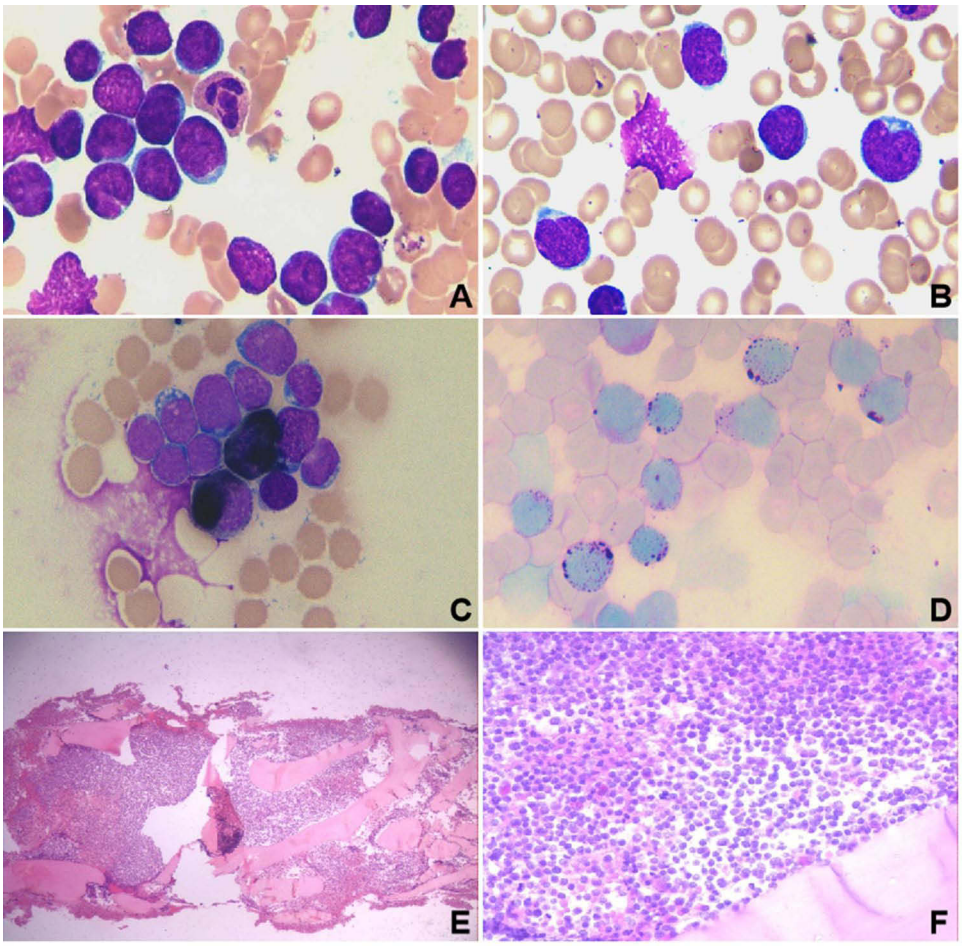

Figure 1 Bone marrow biopsy and bone marrow cytomorphological examination of the patient. (**A-D**) Bone marrow smear images. (**A** and **B**) Wright's staining, 100×; (**C**) POX staining, 100×; (**D**) PAS staining, 100×. (**E** and **F**) Bone marrow biopsy images (Hematoxylin and eosin staining, (**E**) 4×; (**F**) 40×).

(Figure 1E and F). FISH analysis showed that the patient had T cell receptor alpha/delta (TCRAD) gene breakage, implying a diagnosis of ALL again (Figure 2A and B). Immunohistochemical staining results were as follows: CD34(-), TdT(-), CD20(-), PAX5(-), CD5(-), CD7(+), BCL2(-), suggesting a diagnosis of T-ALL. Subsequently, flow cytometry analysis showed that bone marrow precursors accounted for 80.7% of the nuclear cells characterized by $CD34dim^{+}$ $CD117^{-}$ $CD38^{+}$ HLA-DR^{-} $CD7^{+}$ $CD13^{-}$ $CD33^{-}$ $CD123^{-}$ $CD15^{-}$ $CD16^{-}$ $CD3p^{+}$ $CD5^{+}$ $CD4^{+}$ $CD8^{-}$ $CD2^{+}$ $cCD3^{+}$ $CD56^{-}$ $CD99^{+}$, indicating a diagnosis of T-ALL again. Chromosome analysis exhibited a 46, XY, t(11;14)(15;q11.2)[15]/46,XY[5] karyotype (Figure 3). Leukemia fusion gene analysis by real-time quantitative PCR showed that SIL-TAL1 fusion gene was positive. Next-generation sequencing testing revealed that the patient harbored NOTCH1 (mutation frequency: 36.14%, mutation type: terminator acquisition, mutation site: exon34, nucleotide alteration: c.7560G>A, amino acid alteration: p.Trp2520*) and PTEN (mutation frequency: 24.00%, mutation type: frameshift deletion, mutation site: exon6, nucleotide alteration: c.595 597deIATG, amino acid alteration: p. Met199del; mutation frequency: 21.90%, mutation type: non-frameshift insertion, mutation site: exon5, nucleotide alteration: c.296 297insGAG, amino acid alteration: p.Glu99 Leu10 0insArg) mutations (Table 1).

Subsequently, he was transferred to the Hematology Department of our hospital and was given hormonal therapy to reduce tumor load. Hydration, urinary alkalization, and urate-lowering therapy were also selected to improve the patient outcome. Due to abnormal coagulation, the patient was transfused plasma. Under the guidance of ultrasound, thoracentesis was conducted to drain the pleural effusion. Then the patient received 4 weeks of chemotherapy with PCIOD

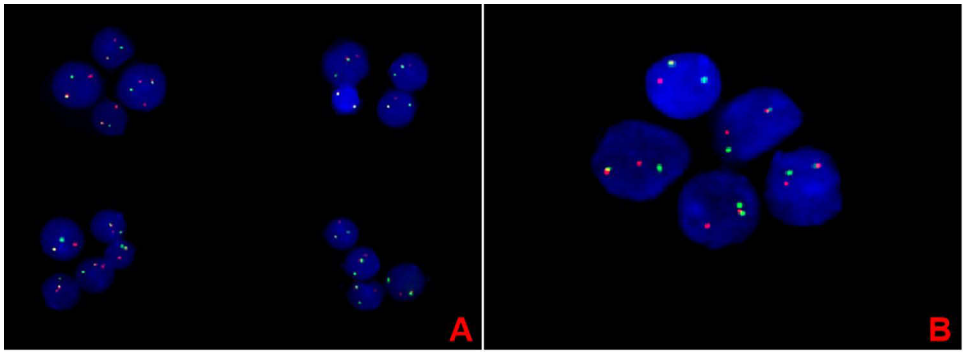

Figure 2 FISH results. (**A**) Positive TCRAD gene breakage; (**B**) Positive TCRAD gene breakage. Red: 5'-end probe signals; green: split 3'-end probe signals; yellow: normal unsplit red-green signal pair.

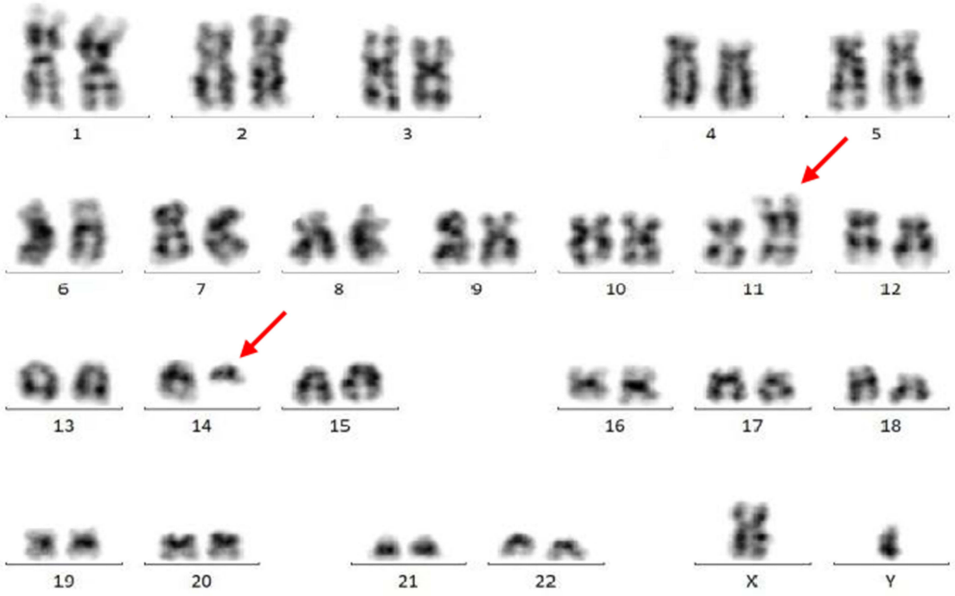

Figure 3 Chromosome analysis exhibited a 46, XY, t(11;14)(15;q11.2)[15]/46,XY[5] karyotype of the patient. Abnormal chromosome 11 and chromosome 14 were shown by red arrows.

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Table 1 Mutated Genes in This T-ALL Patient with Both SIL-TAL1 Positive and T(11;14)(p15;q11.2)

Mutated Gene	Transcript ID	Mutation site	Nucleotide Alteration	Amino Acid Alteration	dbSNP	Mutation type	Mutation Frequency
NOTCH1	NM 017617.4	exon34	c.7560G>A	p.Trp2520*	–	Terminator acquisition	36.14%
PTEN	NM 000314.6	exon6	c.595 597delATG	p. Met199del	rs1064793244	Frameshift deletion	24.00%
PTEN	NM 000314.6	exon5	c.296 297insGAG	p.Glu99 Leu10 0insArg	–	Non-frameshift insertion	21.90%

Table 2 Details of the PCIOD and CAML Regimens

Regimen	Recommended Dosage	Dosage	Date
PCIOD Regimen			
Vindesine	2 mg/m^2	3 mg	d1, d10
Cyclophosphamide	800 mg/m^2	340 mg	d2-d3
		600 mg	d5
Idarubicin	8–12 mg/m^2	10 mg	d5-d7
Pegaspargase	2000–2500 U/m^2	3750 U	d8
Dexamethasone	12–20 mg/m^2	20 mg	d4-d10
CAML Regimen			
Cyclophosphamide	750~1000 mg/m^2	1200 mg	d1, d6
Cytarabine	75~100 mg/m^2	160 mg	d1-d3, d8-d10
6-Mercaptopurine	50~75 mg/m^2	100 mg	d1-d7
Pegaspargase	2000~2500 U/m^2	3750 U	d4

Notes: The PCIOD Regimen is the recommended dosage for adults, and the CAML Regimen is the recommended dosage for children. Adults should refer to the children guidelines and then calculate the dosage based on their weight in kilograms.

regimen (pegaspargase + cyclophosphamide + idarubicin + vindesine + dexamethasone) (Table 2). To prevent central nervous system (CNS) involvement, intrathecal chemotherapy (5mL physiological saline + 10mg methotrexate + 5mg dexamethasone, and 5mL physiological saline + 50mg cytarabine) was given during the treatment. However, he presented with myelosuppression and infection after chemotherapy, and an emergency blood transfusion was given to increase the number of erythrocytes and platelets. Afterwards, bone marrow cell morphology analysis and flow cytometry minimal residual disease (MRD) detection showed no abnormality, indicating that the patient had achieved remission and was discharged to his home in October 2022. Then the patient returned to the outpatient department for review after 5 days. MRD assays revealed that CD45dim cCD3$^+$ mCD3$^-$ CD7$^+$ CD5$^+$ CD99$^+$ CD4$^-$ CD8$^-$ cells accounted for less than 1.0×10^{-4} of CD45$^+$ white blood cells (WBCs), suggesting MRD-negative. Morphologic examination of bone marrow displayed active hyperplasia, but peripheral blood smear showed immature granulocytes, with 4% neutrophilic myelocytes (normal range, 0%) and 24% neutrophilic metamyelocytes (normal range, 0–1%), possibly due to compensatory hyperplasia of bone marrow cells caused by chemotherapy. Then the patient was readmitted and given chemotherapy with CAML regimen (cyclophosphamide + cytarabine + 6-mercaptopurine + pegaspargase) (Table 2). Intrathecal chemotherapy was also performed (the dosage and regimen are the same as those in the previous therapy). After treatment, his condition gradually improved and he was discharged in November, 2022 after stabilization. However, the patient was readmitted to hospital one month later and received methotrexate (2 g, d1), cytarabine (2 g, d2-d3, q12h) and pegaspargase (3750 U, d4) combination chemotherapy, as well as intrathecal chemotherapy (the dosage and regimen were the same as those in the previous therapy). Then the patient was discharged in January, 2023 after his condition gradually improved. But the patient was readmitted to our hospital for the fourth time after 21 days due to persistent pain in the upper abdomen accompanied by non-projectile vomiting without obvious inducement. On examination, white blood cell count was elevated to 292.63×10^9/L (reference range 4–10×10^9/L), the neutrophils ratio was reduced to 11.5% (reference range 50–70%), lymphocytes accounted for 69.1% (reference range 20–50%), monocytes accounted for 19.1%

(reference range 3–10%), and the absolute value of monocytes was elevated to 55.93×10^9/L (reference range $0.1–0.6\times10^9$/L). Bone marrow smears showed active marrow hyperplasia, with 71% of cells being primitive and immature lymphocytes, indicating T-ALL relapse. Immunophenotyping revealed a population of CD45dim $cCD3^{few+}$ $mCD3^-$ $CD7^+$ $CD5^+$ $CD99^+$ $CD4^+$ $CD8^-$ cells, constituting 51.94% of $CD45^+$ WBCs. These findings indicated that a large number of aberrant precursor T-cell phenotype existed, strongly suggesting a risk of relapse of T-ALL. After receiving two days of induction therapy with dexamethasone and cyclophosphamide, he suddenly lost consciousness, exhibited no verbal response to stimuli without bilateral pupil dilation, suspecting of intracranial hemorrhage caused by T-ALL relapse. Regretfully, the patient deceased due to progressive disease with no CT was done. Given the fact that a majority of reports showed intracranial hemorrhage was closely associated with T-cell acute lymphoblastic leukemia, we thought this patient died primarily from T-ALL relapse.[13,14]

Discussion

T-ALL is an intractable hematological malignancy and patients with T-ALL typically present with elevated leukocytes, anemia, and thrombocytopenia, mediastinal thymic masses, and central nervous system infiltration.[15] With advancements in chemo-therapy regimens, the complete remission rate of T-ALL patients has significantly improved in recent years.[16] However, the overall survival rate still remains low. Relapse remains a major challenge in T-ALL treatment and indicates a poor prognosis.[17,18] Due to lack of established clinical relevance, the WHO has not defined genetic subtypes of T-ALL. Recently, T-ALL is classified into two subtypes based on gene expression data, including TAL1/TAL2/LMO1/LMO2/LYL1 driven leukemia, and TLX1-, TLX3-, NKX2-1- and HOXA9 activated subtypes. Additional genetic alterations have also been identified, such as STAG2/LMO2 driven leukemia, HOXA13-activated, BCL11B-activated, and SPI1-rearranged. With the advent of recent technical developments, T-ALL classification has been improved by whole genome (WGS), whole exome (WES), whole transcriptome sequencing (WTS) and divided into 15 subtypes or even more. It is noted that other genetic alterations are mostly sequence mutations and DNA copy number changes affecting important pathways in T-ALL, such as NOTCH pathway (NOTCH1 and FBXW7), PI3K-AKT pathway (PIK3CA, PIK3CD, PIK3R1, PTEN, and MTOR), JAK-STAT pathway (IL7R, JAK1, JAK3, STAT5B, FLT3, PTPN2, SH2B3, and PRLR), and key transcription factors (PHF6, EZH2, MYC,WT1,TCF7). These molecular classification and alterations are crucial for prognosis prediction and personalized treatment. For example, NOTCH1, FBXW7, PHF6, and EP300 are commonly associated with favorable prognosis, while NRAS, KRAS, PI3K pathway related genes, TP53, DNMT3A, IDH1, IDH2, and IKZF1 are associated with a higher relapse risk.[19] Targeting these genes can provide a new perspective for the treatment of T-ALL patients.

In this case, the clinical characteristics, including clinical manifestations, bone marrow cell morphology, chromosome abnormalities, and immunophenotype, are similar to those of adult T-ALL patients with SIL-TAL1 negative. Molecular assays are needed to distinguish between the above two types. At present, the effect of the STL-TAL1 fusion gene on the prognosis of patients with T-ALL is still controversial and we present the related studies in Table 3. For example, Kikuchi et al suggested that patients with SIL-TAL1-positive had better prognosis.[20] While most researchers deemed that SIL-TAL1-positive was closely associated with worse prognosis, high white blood cell (WBC) count, high prevalence of tumor lysis syndrome and disseminated intravascular coagulation, and high early mortality.[21–23] Moreover, SIL-TAL1 can be an indicator of minimal residual disease (MRD) monitoring. For example, Zhao et al analyzed 29 cases of T-ALL patients who undergo HSCT and found that the expression level of the SIL-TAL1 gene can serve as a reliable indicator for MRD in T-ALL patients after transplantation.[24]

Moreover, the patient experienced rapid relapse and poor overall survival despite achieving remission with intensive chemotherapy possibly due to SIL-TAL1 and t(11;14) together result in such aggressive disease. Studies have shown that SIL-TAL1 can cause the aberrant expression of TAL1 protein, which is a member of the class II basic helix-loop-helix (bHLH) family transcription factors and can engage with protein partners to regulate downstream transcriptional activities.[5,6] t(11;14)(p15;q11.2) is a relatively common chromosomal abnormality that can lead to high-level expression of LIM domain only 1 (LMO1).[8,9] Aplan et al have shown that the abnormal expression of TAL1 protein driven by SIL regulatory elements can conjunct with the misexpressed LMO1 protein and collaboratively induce aggressive T-cell malignancies.[26] In transgenic mouse models, the synergistic action of TAL1 and LMO1 display thymocyte

Table 3 Comparison of the Prognosis Between SIL-TAL1 Positive and Negative Patients in T-ALL

Reference	Year	Number of Reported Cases	WBC ($\times 10^9$/L)	CR	RFS/EFS	OS	MRD
[25]	2023	SIL-TAL1 positive (n=19)	88.6(38.3–498.6)**	89.5%(16/19)	3-years RFS:49.2%*	3-years:60.9%	
		SIL-TAL1 negative (n=196)	30.25(0.7–494.0)	70.3%(111/158)	3-years RFS:70.6%	3-years:74.4%	
[22]	2016	SIL-TAL1 positive (n=21)	≥50(n=18)**; <50(n=3)	71.4%(15/21)			MRD level at month 12 $<10^{-4}$(n=9); MRD level at month 12 ≥ 10^{-4}(n=2);
		SIL-TAL1 negative (n=79)	≥50(n=44); <50 (n=35)	64.6%(51/79)			MRD level at month 12 $<10^{-4}$(n=20); MRD level at month 12 ≥ 10^{-4}(n=1)
[23]	2015	SIL-TAL1 positive (n=56)	<20(n=7)**; ≥20 and <100 (n=11); ≥100(n=38)	96.4%(53/55)	5-years EFS:64.1%	5-years:78.6%	SR (n=5) IR (n=28) HR (n=10)
		SIL-TAL1 negative (n=303)	<20(n=67); ≥20<100(n=115); ≥100(n=116)	92.6%(274/296)	5-years EFS:71.2%	5-years:76.3%	SR (n=34) IR (n=131) HR (n=48)
[21]	2014	SIL-TAL1 positive (n=12)	238.3(48.8–819.8)*				
		SIL-TAL1 negative (n=56)	101.1(1.1–593)				
[7]	2013	SIL-TAL1 positive (n=15)	184(21–559)*	90.9%(10/11)	Median 2 months**	Median 4 months**	
		SIL-TAL1 negative (n=47)	47.2(0.77–597)	91.1%(41/45)	Median 12 months	Median 25 months	

Notes: *P<0.05; ** p<0.01.
Abbreviations: WBC, white blood cell; CR, complete response; RFS, recurrence-free survival; EFS, Event-Free Survival; OS, overall survival; MRD, minimal residual disease; SR, stratified as standard risk; IR, intermediate risk; HR, high risk.

developmental abnormalities, resulting in the generation of T-ALL.[27] Tremblay et al have showed that oncogenes TAL1 and LMO1 synergize to expand primitive thymic progenitors and inhibit late differentiation stages, fostering a favorable environment for the emergence of self-renewing leukemia-initiating cells in T-ALL.[28] Furthermore, Oram et al have revealed that the activation of TAL1, coupled with the disruption of epigenetic repression of LMO1 regulatory elements, induces ectopic expression of LMO1, which aided in the development and maintenance of T-ALL.[12] Additionally, Sanda et al have identified that TAL1 can recruit its regulatory partners HEB, E2A, LMO1/2, GATA3, and RUNX1 to form a core TAL1 complex, thus mediating carcinogenic effects by activating downstream genes, like MYB proto-oncogene in T-ALL.[29] According to the literature, we reasoned that SIL-TAL1 and t(11;14) together resulted in such aggressive disease. But the underlying mechanisms still need to be further explored.

Currently, treatment options for T-ALL remain relatively limited. Traditional chemotherapy regimens for newly diagnosed T-ALL patients include induction therapy, consolidation therapy, maintenance therapy, and central nervous system leukemia prophylaxis. Anthracyclines (such as doxorubicin or idarubicin), glucocorticoids (such as dexamethasone or prednisone), vinca alkaloids (such as vincristine or vindesine) are common chemotherapy drugs for induction therapy. Cyclophosphamide, cytarabine, methotrexate are common drugs for intensive chemotherapy. Unfortunately, early relapse (within 2 years of diagnosis) accounts for 80% of total relapses in T-ALL, leading to a poor prognosis for patients. Five-year OS rates for T-ALL patients with chemotherapy are only 10% and the number of patients who achieved a complete response is significantly reduced after recurrence.[30] Herein, two cases (including the present case) about T-ALL patients with both SIL-TAL1-positive and t(11;14)(p15;q11.2) are compared in the Table 4. Due to financial reasons, the patient declined allogeneic hematopoietic stem cell transplantation (allo-HSCT) at the early stage of treatment, and chose traditional chemotherapy regimen. Considering the complexity of the disease, allo-HSCT was planned after the third phase of chemotherapy. Unfortunately, the patient's condition rapidly deteriorated and he passed away. Another case reported by Wang et al showed that after one course of chemotherapy, the symptoms were in remission, but the patient still died after hematopoietic stem cell transplantation (HSCT).[25] The two studies suggest that SIL-TAL1-positive T-ALL patients with the t(11;14)(p15;q11.2) exhibit poor clinical prognosis even after HSCT. However, retrospective study of more cases are needed to further assess the conclusion.

It is noted that the patient also carried mutations in NOTCH1 and PTEN. NOTCH1 is an important transmembrane receptor and its intracellular fragment can be cleaved by γ-secretase complex to release NICD (an activated form of

Table 4 The Clinical Feature of Two T-ALL Patients with Both SIL-TAL1 Positive and t(11;14)(p15;q11.2)

Number	Gender	Age	Chromosome Karyotype	Mutated Gene	Response After Treatment	HSCT or not	Outcome
1	Male	14	46,XY,der(4),inv(8)(p23q23), der(11)t(11;14)(p15;q21), der(14)add(14)(p11)t(11;14) [8]/46,XY[2]	PTEN	Complete remission after 1 course of treatment	HSCT	Death
2	Male	19	46,XY,t(11;14)(15;q11.2) [15]/46,XY[5]	NOTCH1, PTEN	Complete remission after 1 course of treatment	Not HSCT	Death

NOTCH1) in the cytoplasm, then NICD can translocate into the nucleus where activate the transcription of target genes.[31] There is growing evidence that NOTCH signaling pathway mediates tumor cell growth, angiogenesis, metastasis and drug resistance. PTEN gene is a recognized tumor suppressor gene that negatively regulates the PI3K/Akt signaling pathway and is often mutated in several tumor types.[32] Studies have shown that the mutation rate of NOTCH1 is approximately 60% in T-ALL, whereas PTEN is approximately 13%.[33,34] Furness et al and Zuurbier et al found that PTEN tend to have a higher incidence of mutation, while NOTCH1 have a lower incidence of mutation in T-ALL patients with SIL-TAL1-positive group.[34,35] However, the relationship and the underlying regulation mechanisms among SIL-TAL1, t(11;14)(p15;q11.2), NOTCH1, and PTEN still need further investigation. In this report, the patient was positive for NOTCH1 and PTEN mutations, which can cause continuous NOTCH1 and PI3K-AKT signaling in T-ALL. Targeting these signaling pathways could provide novel treatment strategies for T-ALL. Currently, small molecule γ-secretase inhibitors (GSIs) which can prevent the release of active NICD and inhibit NOTCH1 signaling have been designed specifically for T-ALL treatment.[36,37] PI3K inhibitor ZSTK-474 co-administrated with nelarabine (a novel nucleoside analog indicated for the treatment of refractory or relapsed T-ALL) can also significantly inhibit T-ALL cell growth and induce nelarabine-resistant T-ALL primary cell apoptosis.[38] Additionally, immunotherapy is also an optional for T-ALL treatment, such as CAR-T cell therapy, antibody drug conjugates, and bispecific antibodies.[39–41]

In summary, adult T-ALL patients with both SIL-TAL1-positive and t(11;14)(p15;q11.2) are rarely reported, and they exhibit a poor prognosis when treated with traditional chemotherapy and HSCT. This highlights the inadequacy of current treatment strategies and the urgent need for future research to collect comprehensive data on similar rare cases, including clinical features, treatment responses, molecular genetic profiles, and long-term follow-up outcomes, to further investigate new therapeutic targets, optimize treatment protocols, and improve the prognosis of patients. Moreover, establishing specialized repositories for these rare T-ALL subtypes can improve the utilization rate increase our understanding of these rare cases.

Ethics Approval

This study was approved by the Medical Ethics Committee of Second Affiliated Hospital of Army Medical University, written informed consent was obtained from the parents of the patient for publication of all the data and accompanying images contained in this study. Institutional approval was required to publish the case details (institution name: the Medical Ethics Committee of Second Affiliated Hospital of Army Medical University). Institutional approval was granted to publish the case details (institution name: the Medical Ethics Committee of Second Affiliated Hospital of Army Medical University).

Consent to Participate

Written informed consent was obtained from the parents of the patient for publication of all the data and accompanying images contained in this study.

Consent for Publication

All authors/participants have seen and approved the final version of the manuscript being submitted.

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Disclosure

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