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Case Report

Rare *NUP98::SETBP1* fusion transcript in a refractory adult T-cell lymphoblastic lymphoma

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Abstract: T-lymphoblastic lymphoma (T-LBL) is an aggressive malignancy of T-lymphoid precursors, rarely co-occurring with consequent rearrangement of nucleoporin 98 (*NUP98*) gene. The *NUP98::SETBP1* fusion gene was first reported in a de novo T-cell acute lymphoblastic leukemia (T-ALL) patient. However, it has not yet been reported in patients with T-LBL. We report a case of T-LBL with the *NUP98::SETBP1* fusion gene. The *NUP98::SETBP1* fusion gene was discovered by RNA sequencing (RNA-seq) in the patient's bone marrow cells and verified in various tissues through direct sequencing and reverse transcriptase polymerase chain reaction (RT-PCR). Unfortunately, despite the use of various treatment methods, including traditional chemotherapy, anti-CD7 CAR-T cell therapy, and CD38-targeted therapy, the patient showed no improvement and ultimately died from disease progression. To our knowledge, this is the first report of the translocation involving *NUP98* and *SETBP1* genes in T-LBL. It is necessary to collect additional cases and conduct carefully designed experiments to establish the recurrence of this fusion in other T-LBL cohorts and confirm its role as a novel oncogenic rearrangement in T-LBL, providing a basis for managing such patients.

Keywords: T-lymphoblastic lymphoma, nucleoporin 98, set binding protein 1, RNA sequencing, gene fusion

Introduction

The nucleoporin 98 (*NUP98*) gene localized to chromosome band 11p15, which was first described in 1996, when Borrow et al. and Nakamura et al. independently reported that *NUP98* was fused to *HOXA9* in de novo acute myeloid leukemia (AML) patient bearing t(7;11)(p15;p15) [1, 2]. The gene is known to be fused to nearly 40 different genes in hematological malignancies, including *HOXA9/11/13*, *HOXC11/13*, *HOXD11/13*, *PRPPX1/2*, *GSX2*, *LEDGF*, *NSD1/3*, *KMT2A*, *RARG*, *KDM5A*, *PMX1*, *RAP1GDS1*, *TOP1* and among others [3]. Based on whether there is a homeodomain moiety, *NUP98* fusion partners can be classified into two major categories. Seven clustered "class I" HOX genes (*HOXA9*, *HOXA11*, *HOXA13*, *HOXC11*, *HOXC13*, *HOXD11* and *HOXD13*) and 5 non-clustered "class II" HOX genes (*HHEX*, *GSX2*, *PRRX1*, *PRRX2* and *POU1F1*) are among the partners with retained homeodomains [4].

NUP98-rearranged can occur in almost all hematological malignancies, with higher incidence in pediatric cases [5]. Previous literature reports that the detection rate of *NUP98*-rearranged in childhood AML is approximately 5% to 10%, and approximately 2% to 4% in adult AML [6-9]. *NUP98*-rearranged occur in approximately 12% of pediatric T-cell acute lymphoblastic leukemia (T-ALL) cases, notably higher than in B-cell acute lymphoblastic leukemia (B-ALL) or other B-cell malignancies [10]. In addition, the *NUP98*-rearranged also has been detected in therapy-related AML (t-AML), myelodysplastic syndrome (MDS), chronic myeloid leukemia (CML) and mixed-phenotype acute leukemia (MPAL) [10-12].

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So far, five *NUP98* chimaeras have previously been reported in T-ALL: *NUP98::RAP1GDS1*, *NUP98::VRK1*, *NUP98::ADD3*, *NUP98::CCDC-28A*, and *NUP98::SETBP1* [3]. The *NUP98::SETBP1* fusion gene is exceptionally rare, with only one case reports identified through an extensive search in the PubMed database. In 2006, the *NUP98* gene at 11p15 was first shown to be fused to the *SETBP1* gene at 18q12 in t(11;18)(p15;q12)-positive T-ALL [13]. Here, we report an adult T-cell lymphoblastic lymphoma (T-LBL) case with primary refractory disease. Comprehensive genetic testing showed that many molecular variations (including *TP53* mutations, and *NUP98::SETBP1* fusion) were highly linked to his serious and complex medical history.

Case presentation

A 33-year-old male, who presented with cough and fever for five days, was admitted to our hospital on July 28, 2024. Peripheral blood analysis revealed normal white blood cells (WBC, 7.45 × 10^9/L) and hemoglobin (Hb, 125 g/L), with slightly elevated platelets (PLT, 353 × 10^9/L). The routine biochemical indicators and tumour biomarker (CA19-9, CA125, AFP and CEA) were within normal limits. Serological evaluations were negative, including HIV by enzyme-linked immunosorbent assay (ELISA), hepatitis B virus (HBV) by serology or polymerase chain reaction (PCR), and Epstein-Barr virus (EBV) and cytomegalovirus (CMV) by PCR. Autoantibodies associated with rheumatic diseases (ANA, ENA, anticardiolipin) were negative. Bone marrow (BM) examination revealed a small number of lymphoblast (3.5%), phagocytes, and hemophagocytosis, with negative Periodic Acid-Schiff (PAS) staining (**Figure 1A**). Flow cytometry (FCM) of the BM identified a 1.2% abnormal cell population of nuclear cells expressing CD7, CD45RA, CD117, and CD38. Chromosomal analysis revealed a karyotype of 46, XY [20] (**Figure 1B**). The next-generation sequencing (NGS) examination of BM sample identified specific mutations, namely *TP53* R175H (1.57%) and *KDM6A* V1113* (2.0%). RNA sequencing (RNA-seq) analysis of BM sample identified a gene fusion between exon 12 of *NUP98* and exon 6 of *SETBP1*, which was validated by agarose gel electrophoresis analysis of polymerase chain reaction (PCR) amplification products and Sanger sequencing (**Figure 1C-E**). FCM of the pleural effusion sample identified a 37% abnormal cell population of nuclear cells expressing CD7, CD45RA, CD117, and CD38. In addition, the presence of the *NUP98::SETBP1* fusion gene was also confirmed by reverse transcriptase PCR (RT-PCR) in pleural effusion sample. PET-CT imaging revealed multiple-area lymph node enlargement, pleural thickening, mediastinal masses, and pericardial invasion, with SUVmax values ranging 5.1 to 36.4 (**Figure 2A**). Histologic examination of the left pleura revealed diffuse infiltration by tumor cells, accounting for approximately 90% of the tissue cellularity, with no evidence of vascular or perineural invasion. The tumor cells exhibiting scant to moderate cytoplasm and predominantly round nuclei, some displaying irregular nuclear contours; the chromatin appeared relatively fine, and mitotic figures were frequent. Immunohistochemical studies revealed the tumor cells were diffusely positive for CD3, CD7, CD117, CD99, and BCL-2, with a Ki-67 proliferation index of 95%. The tumor cells also showed focal positivity for CD5 (approximately 30% of cells) and were negative for TDT (<1%), CD34, CD1a, CD20, CD4, CD8, BCL6, and EBER (**Figure 2B**). The immunohistochemical results were further confirmed by pathological examination of the left supraclavicular lymph nodes. T-LBL is supported by the tissue morphology and immunohistochemical findings. Integrating these findings, the patient was definitively diagnosed with T-LBL (stage IV-B, IPI: 2 points). From 12 August 2024 to 2 September 2024, one courses of VDCLP chemotherapy regimen [cyclophosphamide (CTX) 375 mg/m^2 d1-2, daunorubicin (DNR) 50 mg/m^2 d1-3, vincristine (VCR) 2 mg/d d1/8/15/22, pegaspargase (PEG-ASP) 3750 U d1/15, prednisone (PDN) 60 mg/m^2 d1-14] were administered.

On 11 September 2024, the patient returned to our hospital for regular chemotherapy. BM smear showed a reduced proportion of lymphoblasts, with 1.5%. FCM of BM and pleural effusion still revealed that 0.3% and 6.5% of nucleated cells expressed CD7, CD45RA, CD117 and CD38, respectively. Chest CT scan showed that the irregular mass shadow in the left anterior superior mediastinum was unchanged, and the left pleural effusion had increased. Considering the poor therapeutic effect, one course of a highly intensive induction chemotherapy regi-

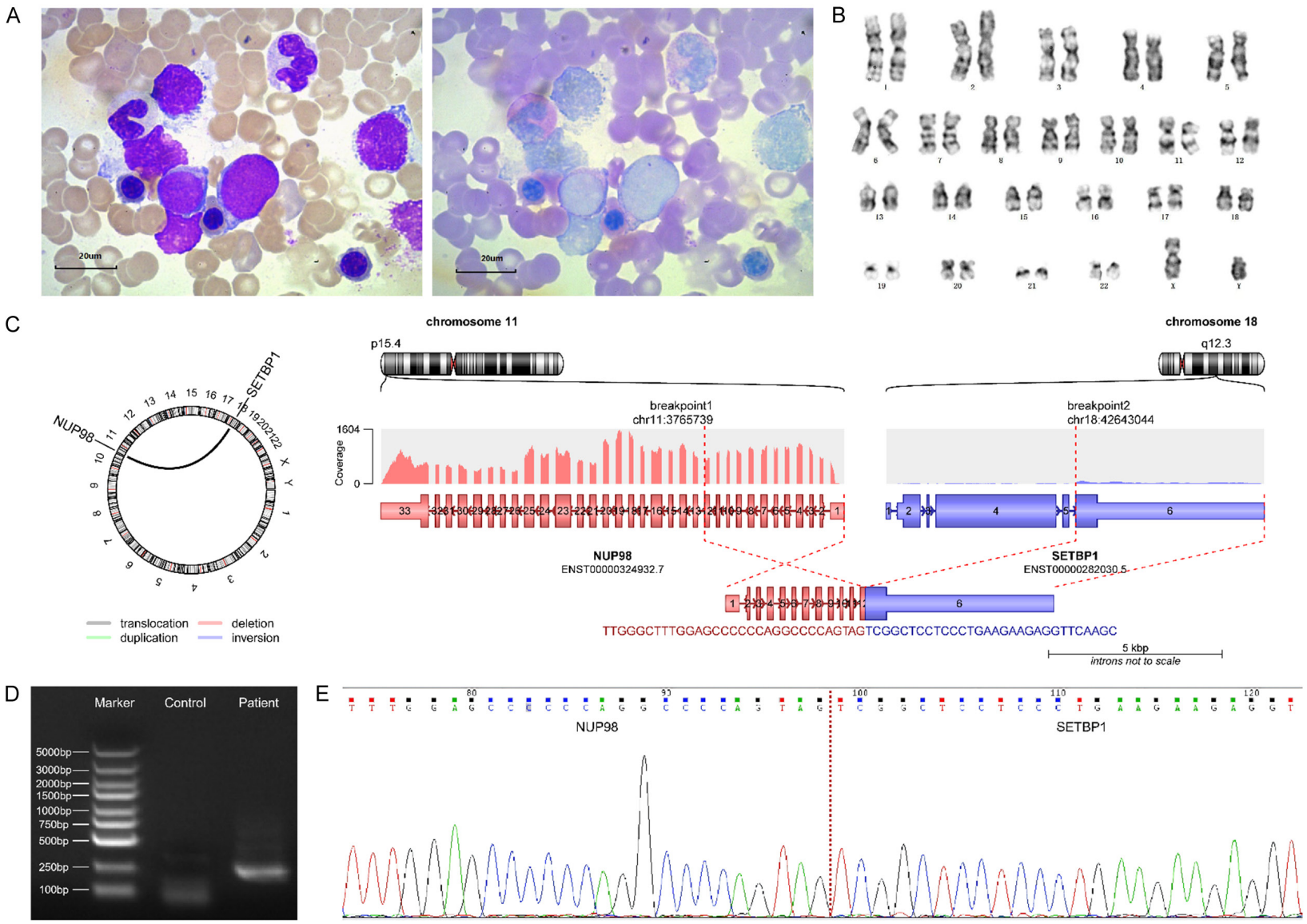

Figure 1. Morphologic, cytogenetic and molecular characterization of BM samples from the T-LBL patient with *NUP98::SETBP1* fusion. A. Bone marrow smear showing lymphoblast infiltration of 3.5% at diagnosis (×400). B. Karyotype analysis at primary diagnosis. C. The predicted structure of *NUP98::SETBP1* in-frame gene fusion transcript from RNA-sequencing was visualized using Arriba (*NUP98* breakpoint chr11:3765739; *SETBP1* breakpoint chr18:42643044). D. Agarose gel electrophoresis analysis of PCR products. E. Sanger sequencing confirmed an in-frame fusion between exon 12 of *NUP98* and exon 6 of *SETBP1* (Forward primer: 5'-CTGGACAGGCATCTTTGTTTG-3', Reverse primer: 3'-GTGGACAGGATCTTGGTGTAG-5').

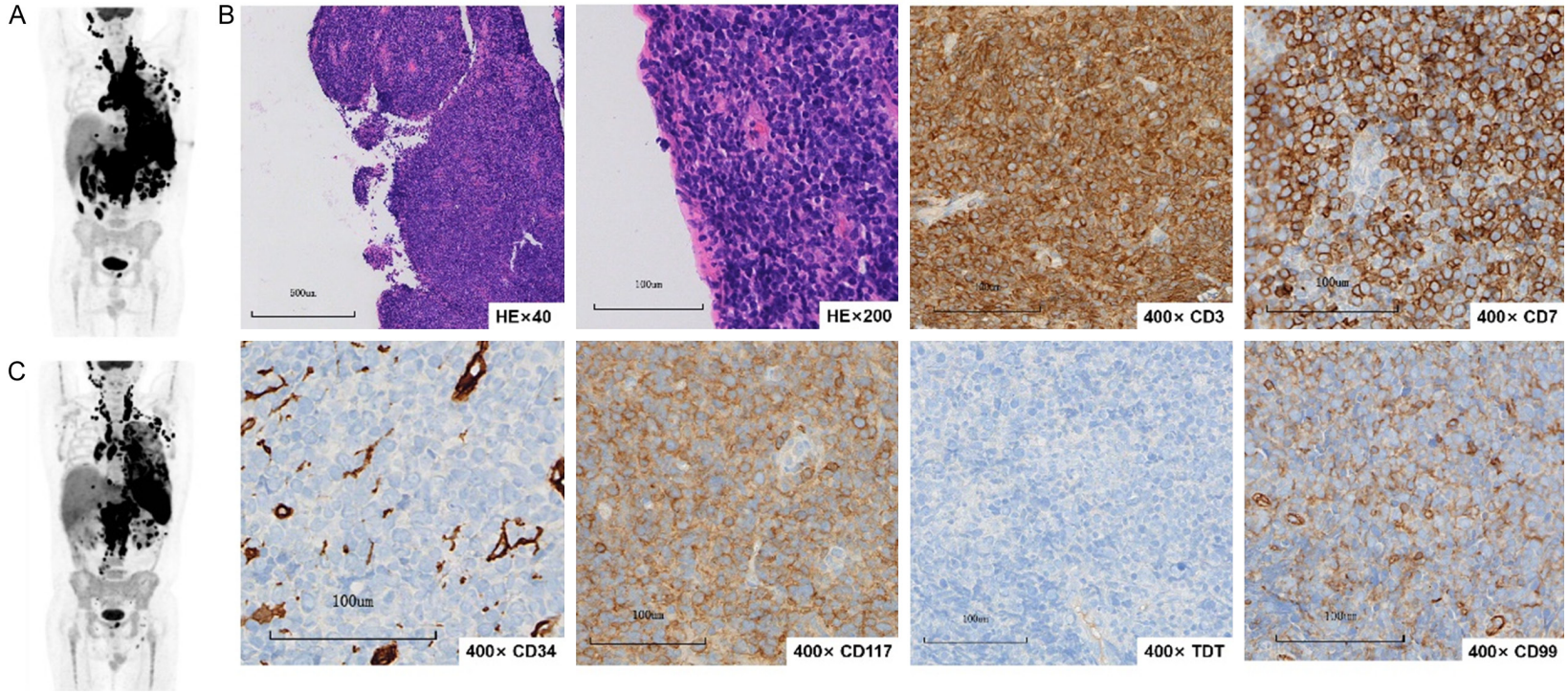

Figure 2. The imaging and pathological characteristics of the patient. A. PET/CT findings at initial diagnosis. B. Histological examination of the left pleura revealed diffuse infiltration by tumor cells, characterized by scant to moderate cytoplasm and predominantly round nuclei, some displaying irregular nuclear contours, with relatively fine chromatin and frequent mitotic figures. Immunophenotypic analysis (EnVision method) demonstrated positivity for CD3, CD7, CD117, and CD99. C. PET/CT findings following two cycles of induction chemotherapy.

men with MA [methotrexate (MTX) 2 g/m^2 d1, cytarabine (Ara-C) 2 g/m^2 d2-3 twice daily] was administered from 11 September 2024 to 13 September 2024. The patient experienced extremely severe gastrointestinal bleeding, with the maximum daily bleeding volume of 1500 mL, from 21 September 2024 to 6 October 2024. After endoscopic hemostasis (gastroenterology), embolization of a superior mesenteric artery branch and the gastroduodenal artery (interventional radiology), and advanced life support (ICU), the patient's gastrointestinal bleeding gradually improved.

On 23 October 2024, the patient's disease status was re-evaluated. BM morphology indicated a significant increase in the proportion of lymphoblasts, accounting for 21%. BM FCM examination showed that the proportion of abnormal phenotypic nucleated cells was increased (9.6%), and the phenotype was consistent with previous results. PET/CT scans demonstrated suppressed activity at the original tumor site post-treatment, but persistent metabolic activity (Deauville score 5). Multiple nodules with increased glucose metabolism have emerged in the bilateral preauricular regions, neck, liver capsule, iliac vessels, and inguinal regions. These findings suggest lymphoma progression (Deauville score 5) (**Figure 2C**). In addition, the patient presented with moderate pericardial effusion on this admission. FCM of pericardial effusion detected 41% abnormal cells, phenotypically identical to those in the BM. The *NUP98::SETBP1* fusion gene was further confirmed by RT-PCR of pericardial effusion samples. 25 October 2024 to 27 October 2024, one course of a highly intensive chemotherapy regimen with MEO [liposome-entrapped mitoxantrone 20 mg/m^2 d3, etoposide (VP-16) 100 mg/m^2 d1-3, oxaliplatin (OXA) 100 mg/m^2 d1] was administered.

On 28 November 2024, peripheral blood (PB) morphology revealed 33% lymphoblasts. FCM of PB revealed that 35.6% of nucleated cells abnormally expressed CD7, CD45RA, CD117 and CD38, respectively. Due to disease progression during treatment, with both BM and PB FCM demonstrating high CD7 expression in tumor cells, the patient received anti-CD7 CAR-T cell (2 × 10^6/kg) therapy on December 15, 2024. Grade 2 cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) developed post-infusion. Disease status of the patient be reassessed on January 26, 2025. CT scan demonstrated a left anterior superior mediastinal mass, left pleural thickening, left pleural effusion, mediastinal/retroperitoneal lymphadenopathy, and splenomegaly, all no obvious changes compared to prior imaging. BM morphology demonstrated a significantly increased proportion of lymphoblasts (70.5%). FCM of BM revealed that 74.5% of nucleated cells abnormally expressed CD33, CD117, CD45RA and CD38. Based on these examination results, the patient was assessed to be in a state of non-remission following anti-CD7 CAR-T cell therapy. Despite poor clinical status, the patient and family requested treatment continuation. Given high CD38 expression in tumor cells, anti-CD38 monoclonal antibody (daratumumab) monotherapy was initiated on January 27. On 18 February 2025, PB morphology demonstrated that lymphoblasts remained elevated at 88%. Unfortunately, the patient eventually died secondary to disease progression on 19 February 2025. Data on CD7 CAR-T cell expansion, persistence, cytokine levels, and tumor cell immunophenotype following CD7 CAR-T infusion are provided in the Figure S1 and Table S1.

Discussion

NUP98 fusion was first described in AML patients with recurrent t(7;11)(p15;p15) translocation that resulted in a *NUP98::HOXA9* fusion gene [1, 2]. Since then, this fusion has been detected in a spectrum of hematologic malignancies, including AML, T-ALL, MDS, t-AML/MDS, CMML, and MPAL, with a low incidence (<5%) [4]. *NUP98* fusion oncoproteins induce abnormal gene expression programs, and rewired protein interactions, ultimately causing cell cycle alterations and structural cellular changes, collectively driving leukemogenesis [14].

Currently, nearly 40 *NUP98* fusion partners have been identified in hematological malignancies. At least five different fusion partners of the *NUP98* gene have been cloned in T-ALL, such as *SETBP1* in t(11;18)(p15;q12) in patients with T-ALL [13]; *RAP1GDS1* in t(4;11)(q21;p15) seen in patients with T-ALL and AML [15, 23, 25]; *VRK1* in t(14;11)(q32;p15) in patients with T-ALL [16]; *CCDC28A* in t(6;11)(q24;p15) in patients with T-ALL and AML [17]; and *ADD3* in t (10;11)(q25;p15) in patients with

T-ALL and AML [18, 24]. The first case with t(11;18)(p15;q12) was reported in a 9-year-old T-ALL patient in 2006. In this study, Panagopoulos et al. demonstrated that *SETBP1* on 18q12 was the partner of *NUP98* gene in this translocation. This translocation resulted in the *NUP98::SETBP1* fusion gene, a significant molecular alteration in hematological malignancies [13]. To provide a comprehensive understanding of this disease entity, we systematically summarized the clinical features, MICM (morphology, immunology, cytogenetics and molecular biology) characteristics, treatment response, and survival outcomes of reported T-ALL/LBL with *NUP98* rearrangements in the literature (**Table 1**).

T-LBL is a rare and aggressive subtype of T-cell non-Hodgkin's lymphoma (T-NHL) that mainly affects children and young adults, involving the thymus, lymph nodes, and BM. T-LBL accounts for approximately 85% of all immature lymphoblastic lymphomas, while immature B-cell lymphomas make up the remaining portion [19]. Previous studies have shown that T-LBL and T-ALL differ not merely in bone marrow blast proportion. The unique fusion transcript landscape in T-LBL is quite different from that detected in T-ALL, and the *NUP98* fusion gene is extremely rare [16, 20, 21].

Here we first presented one T-LBL patient with *NUP98::SETBP1* fusion gene. The fusion gene was discovered by RNA-seq and verified in various tissues through direct sequencing and RT-PCR. Our case is the second case of a T-cell malignancies with *NUP98::SETBP1* fusion gene. However, only the fusion gene was detected in our case, without t(11;18)(p15;q12) chromosomal abnormalities. We speculate this occurred because tumor cell burden was too low at initial diagnosis to yield sufficient metaphases for cytogenetic analysis. Furthermore, *NUP98*-rearranged is likely an underrepresentation given the cryptic nature of these fusions frequently not detected by conventional karyotyping. Therefore, RNA-seq, RT-PCR and/or FISH may be more reliable methods to detect this abnormality [22].

The *NUP98::SETBP1* fusion resulting from t(11;18)(p15;q12) translocation is extremely rare in T-cell malignancies, and even in all hematological malignancies. Thus, no conclusions can be drawn with regard to its clinical implications. As summarized in **Table 1**, *NUP98* fusions in T-ALL/LBL exhibit variable outcomes, with some cases achieving durable remission, while others show primary resistance. Notably, the survival time (>2 years) in previously reported pediatric T-ALL cases with *NUP98::SETBP1* exceeded that of our adult T-LBL patient (206 days) [13]. This discrepancy may suggest heterogeneity in the clinical behavior of this fusion across different T-cell malignancies. Furthermore, our patient's concomitant TP53 mutation, a well-established marker of chemoresistance and poor prognosis in lymphoid malignancies, likely contributed to the aggressive course and limited response to chemotherapy, CAR-T therapy, and targeted agents. In T-LBL, a disease with distinct molecular and clinical features, the presence of *NUP98::SETBP1* may signal high-risk behavior, particularly when co-occurring with other high-risk lesions such as *TP53* mutations. However, larger cohort studies are needed to determine whether *NUP98::SETBP1* is an independent prognostic marker in T-LBL.

Notably, our patient had relapsed or refractory T cell acute lymphoblastic leukemia and was refractory to multiple prior therapies, including anti-CD7 CAR-T therapy. Previous studies have shown that the poor therapeutic effect of CAR-T therapy may be related to the following factors, including antigen escape, an immunosuppressive tumor microenvironment, T-cell dysfunction or exhaustion related to prior therapies, and clonal heterogeneity [26-29]. Review of our patient's treatment course revealed loss of the CD7 antigen on tumor cells following anti-CD7 CAR-T therapy, suggesting that treatment failure may be attributed to antigen escape. Furthermore, the inadequate expansion of CD7-targeted CAR-T cells may be another factor. It is well known that there is no standard treatment plan for patients with relapsed/refractory T-LBL. Although anti-CD7 CAR-T cell therapy failed in our patient, it remains a significant treatment modality for this population [27]. Furthermore, combination regimens based on nelarabine [30], venetoclax [31, 32], HDAC inhibitors [33], or CD3/CD38 bispecific antibodies [34] also have demonstrated promising efficacy. Finally, given the role of *NUP98* fusions in altering transcriptional programs, small molecule inhibitors targeting downstream effectors such as CDK4/6 and Menin could be considered in the context of clinical trials [35]. In the future, it is necessary to accumulate more

Table 1. Clinical features of T-ALL/LBL patients with NUP98 fusion reported in the literature and the present study

Fusion partner	Age	Gender	WBC (10^9/L)	PB blast	BM blast	Immunophenotype	Karyotype	Induction regimen	Response	Relapse/ Refractory	Transplantation	Survival	References
ADD3	23	Male	4.8	12%	68%	T/Myeloid blasts: CD34+(dim), CD45+(dim), CD13+(dim), CD11b+(dim), CD33+, HLA-DR+, cMPO+, cCD3+, CD7+; T-Lympho-blasts: CD34+, CD45+, TdT+, cCD3+, CD7+, CD5+, CD11b+, CD33+	46, XY, add(11)(p15) [27/35]	IAE	CR	No	Autologous	8+ months	[18]
ADD3	31	Male	46.53	68%	81%	CD3+, CD5+, CD117+, CD38+, cCD3+, CD7 (partial+), CD200 (partial+)	46, XY [20]	V-HAG	NR	Yes	No	4+ months	[24]
RAP1GDS1	50	Female	14.4	NA	NA	pro-T-ALL	NA	NA	CR	No	NA	NA	[16]
RAP1GDS1	21	Male	438.3	NA	NA	pro-T-ALL	46, XY, del(4)(q23), der(7), add(9)(q34), add(11)(p25)	NA	CR	No	NA	NA	[16]
RAP1GDS1	21	Male	423	99%	NA	CD2+, CD3+(30%), CD4-, CD5+, CD7+, CD8-, CD10+, CD11b+(14%), CD14-, CD19-, CD20-, CD33+(34%), CD34+, CD71+, HLA-DR+	46, XY, t(4;11)(q21p15), +2mar	NA	CR	Yes	Allogeneic*2	43 months	[25]
RAP1GDS1	25	Female	1.8	87%	NA	CD2+(13%), CD3-, CD4-, CD5+(13%), CD7+, CD8-, CD10+(14%), CD13+(5%), CD14-, CD19-, CD33+(18%), CD34+(5%)	46, XX, t(4;11)(q21p14-15), del(12)(p13), +del(13)(q12q14)	NA	NR	Yes	No	34 days	[25]
RAP1GDS1	49	Male	169	99%	NA	CD2-, CD4-, CD5+, CD7+, CD8-, CD10+(9%), CD19-, CD34+	46, XY, t(4;11)(q21p15), del(5)(q13q31)	NA	CR	Yes	No	14 months	[25]
RAP1GDS1	16	Female	34.1	90%	NA	CD19-, CD10-, CD20-, CD7+, CD5+, CD2+, CD1a+, sCD3-, CyCD3+, CD13+, CD33-, CD34-, HLA-DR-, TdT+	47, XX, t(4;11)(q21;p15), +8	VDLP	CR	No	Allogeneic	NA	[15]
RAP1GDS1	38	Female	35.1	74%	95%	CD19-, CD10+, CD20-, CD7+, CD5+, CD2-, CD1a-, sCD3+, CyCD3+, CD4+, CD13-, CD33-, CD34-, HLA-DR-, TdT+	46-47, XX, t(4;11)(q21;p15), +mar	VDLP	CR	Yes	Allogeneic	NA	[15]
RAP1GDS1	25	Male	45.9	80%	96%	CD2+, CD3+, CD5+ CD7+, CD33+	46, XY, t(1;4;11)(p32;q21;p15)	VDP	CR	No	Allogeneic	38+ months	[23]
CCDC28A	26	Male	NA	NA	NA	NA	46, XY, t(6;11)(q21-22;p15), add(12)(p13) [17]/46, XY [3]	NA	NA	NA	NA	NA	[17]
DDX10	43	Female	10.67	NA	NA	ETP-ALL	47, XX, t(4;11)(q12;p15), +M [8]/46, XX [2]	NA	CR	No	NA	NA	[16]
VRK1	13	Female	3.8	NA	NA	ETP-ALL	46, XX, del(6)(q13q23) [6]/46, XX [14]	NA	CR	No	NA	NA	[16]
SETBP1	9	Male	4.7	NA	33%	CD34+, TdT+, CD7+, CD5+	46, XY, t(11;18)(p15;q12), del(12)(p11)(15)	Nordic ALL-2000 protocol	CR	No	NA	2+ years	[13]
SETBP1	33	Male	7.45	0	3.5%	CD7+, CD45RA+, CD117+, CD38+	46, XY [20]	VDCLP	NR	YES	No	206 days	Our case

Abbreviations: WBC, white blood cell; PB, peripheral blood; BM, bone marrow; NR, no remission; CR, complete remission; NA, not available.

cases and conduct carefully designed experiments to further establish the recurrence of the fusion in other sample series of T-LBL and to confirm its role as a novel cancer-driver rearrangement of the *NUP98::SETBP1* fusion gene in different diseases, providing a basis for managing such patients.

Besides the *NUP98::SETBP1* fusion, our patient harbored a *TP53* hotspot missense variant (R175H) and a truncating *KDM6A* variant (V1113*), both detected at low VAF in bone marrow. TP53 mutations are strongly associated with genomic instability, chemoresistance, and poor prognosis, while KDM6A loss may have altered epigenetic regulation and glucocorticoid sensitivity in the context of a NUP98-rearranged T-cell malignancy [36, 37]. Taken together, these concurrent abnormalities may contribute to an aggressive and treatment-refractory phenotype, but their functional cooperation cannot be established from a single case and requires validation in larger cohorts and experimental systems.

Conclusions

To our knowledge, this is the first report of *NUP98::SETBP1* fusion in T-LBL and the second report in T-cell malignancies. This fusion gene was discovered through RNA-seq and confirmed in multiple tissues, including BM, pleural effusion, and pericardial effusion, using direct sequencing and RT-PCR. However, the BM chromosome examination showed a normal karyotype. These results suggest that comprehensive approaches should be utilized in clinical practice to assess genetic and molecular abnormalities of patients. Furthermore, in our study, multiple treatment regimens were ineffective for this patient, including traditional chemotherapy, anti-CD7 CAR-T cell therapy, and CD38-targeted (Daratumumab) therapy. However, the number of T-LBL cases with *NUP98::SETBP1* fusion gene reported so far is too small to draw firm conclusions about the prognosis of the diseases with this translocation. Thus, it is necessary to accumulate more data on patients with *NUP98::SETBP1* fusion gene.

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Disclosure of conflict of interest

None.

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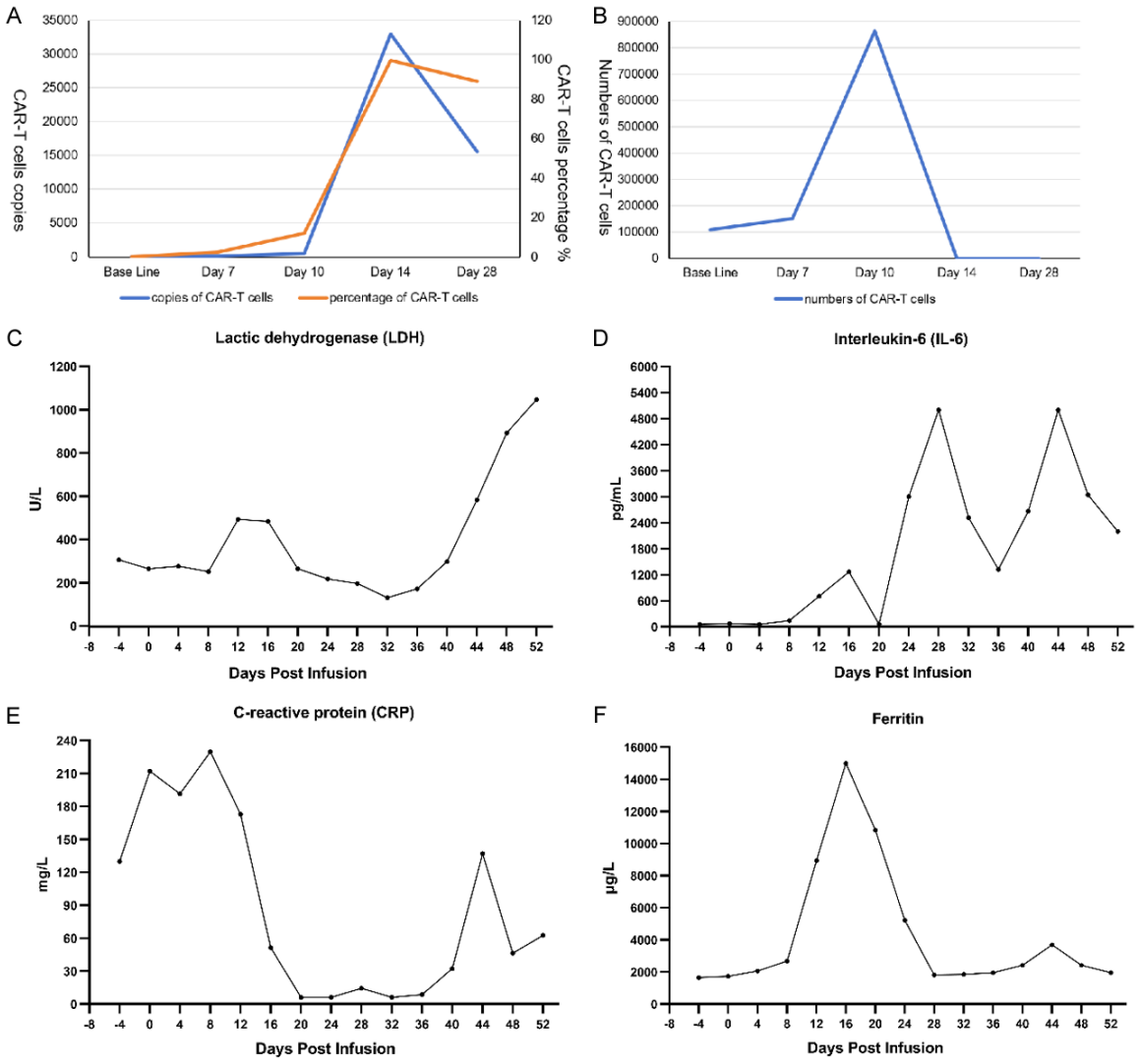

Figure S1. Cell expansion, persistence and cytokine levels in peripheral blood following anti-CD7 CAR-T cell infusion. A. Copies and percentage of CAR-T cells. B. Numbers of CAR-T cells. C. Levels of LDH during CAR-T cell therapy. D. Levels of IL-6 during CAR-T cell therapy. E. Levels of CRP during CAR-T cell therapy. F. Levels of Ferritin during CAR-T cell therapy.

Table S1. Flow cytometry test results of the patient at different time points

Time	Sample	CD7		CD38		CD33	
		Positivity rate (%)	MFI	Positivity rate (%)	MFI	Positivity rate (%)	MFI
2024-09-11	BM	100	4496	98.4	12661	NA	NA
2024-10-23	BM	100	4416	100	38085	4.3	275
2024-11-28	PB	74.2	3796	99.4	26443	NA	NA
2024/12/15 (anti-CD7 CAR-T therapy)							
2024-12-25	PB	51.1	985	99.2	29527	29.8	1447
2025-01-26	BM	3.5	385	98.1	35541	88.4	1344
2025/01/27 (anti-CD38 monoclonal antibody therapy)							

Abbreviations: PB, peripheral blood; BM, bone marrow; MFI, mean fluorescence intensity; NA, not available.