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ORIGINAL ARTICLE

Clinical, Immunologic, and Genetic Characteristics of T-lymphoblastic Leukemia with *STIL-TAL1* Fusion

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SUMMARY

Background: **T-lymphoblastic leukemia (T-ALL) is an aggressive hematologic malignancy with a less favorable prognosis. The genetic background of T-ALL is widely heterogeneous, with the co-occurrence of multiple genetic abnormalities. The *STIL-TAL1* rearrangement results from a submicroscopic deletion on chromosome 1p33 and is present in 15 - 25% of T-ALL cases. Submicroscopic deletions are not detected by conventional cytogenetic analyses but can be identified through array comparative genomic hybridization and/or high-throughput RNA sequencing. Patients with the *STIL-TAL1* fusion exhibit distinct characteristics, such as a young age, high white blood cell count, typical immunophenotype, and specific genetic abnormalities. However, the clinical, laboratory, and prognostic significance of this rearrangement remains unclear. This study was performed to identify *STIL-TAL1* rearrangement resulting from submicroscopic 1p33 deletion in T-ALL and to investigate the clinical, immunologic, and genetic characteristics of T-ALL patients with *STIL-TAL1* fusion.**
Methods: **A total of 15 T-ALL patients were enrolled over a 6-year period (2018 - 2023). We evaluated clinical features and laboratory findings, including immunophenotyping, multiplex reverse transcriptase-polymerase chain reaction (RT-PCR), karyotype analysis, next-generation sequencing (NGS), and chromosomal microarray analysis (CMA), on bone marrow or peripheral blood specimens at the diagnostic stage.**
Results: **Multiplex RT-PCR was performed on 15 cases of T-ALL, and *STIL-TAL1* fusion was detected in 3 cases (20.0%, 3/15). *STIL-TAL1*-positive patients were all male, under 40 years of age, and presented with lymph node enlargement, hepatosplenomegaly, and mediastinal mass. They showed relatively higher leukocyte counts and hemoglobin levels, but lower platelet counts compared to *STIL-TAL1* negative cases. Immunophenotyping analysis revealed higher sCD3 and lower CD34 expression with no aberrant myeloid lineage expression. CMA demonstrated a 63-kb heterozygous deletion at 1p33, along with additional copy number abnormalities such as *TCR* rearrangements and a biallelic *CDKN2A* deletion.**
Conclusions: **The T-ALL with *STIL-TAL1* fusion exhibits unique clinical, immunologic, and genetic characteristics. Further multi-center studies, incorporating cytogenetic and molecular analyses, are needed to elucidate the detailed pathophysiology, characteristics, and clinical significance of this gene rearrangement.**
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KEYWORDS

T-lymphoblastic leukemia, *STIL-TAL1*, submicroscopic deletion, microarray, *TCR*, *CDKN2A*

INTRODUCTION

T-lymphoblastic leukemia (T-ALL) is an aggressive hematologic malignancy characterized by the proliferation of immature T-cells. It accounts for 10 - 15% of pediat-

ric and 25% of adult acute lymphoblastic leukemia (ALL) cases, with a less favorable prognosis compared to B-lymphoblastic leukemia (B-ALL) [1,2]. T-ALL exhibits a heterogeneous genetic background, with multiple genetic abnormalities occurring simultaneously [2]. Therefore, there is currently no integrated test available for clinical use, and molecular cytogenetic tests are not commonly used in the diagnostic evaluation of T-ALL, with only a limited number of studies addressing this issue [2].

TAL1 is a classical oncogenic transcription factor that is abnormally expressed in acute leukemia, representing about 30 - 35% of T-ALL cases [3,4]. The *STIL-TAL1* rearrangement, which results from a submicroscopic deletion on chromosome 1p33, is one of the mechanisms causing the inappropriate overexpression of *TAL1* [1,5]. Patients with the *STIL-TAL1* rearrangement exhibit distinct clinical and biological characteristics, such as a relatively younger age, higher white blood cell (WBC) count, cortical T-cell immunophenotype, and specific genetic abnormalities [2,6]. However, the clinical and prognostic significance of this rearrangement remains controversial, with only a few studies available [1,6,7]. In this study, we demonstrated the presence of *STIL-TAL1* fusion resulting from a 1p33 deletion using reverse transcriptase-polymerase chain reaction (RT-PCR) and chromosomal microarray analysis (CMA). We also investigated the clinical, immunologic, and genetic characteristics of T-ALL patients with *STIL-TAL1* fusion.

MATERIALS AND METHODS

Patients

This study included 15 patients diagnosed with T-ALL based on a bone marrow study at Kyungpook National University Hospital from September 2018 to May 2023. During this period, a total of 17 patients were diagnosed with T-ALL, but 2 of them were excluded because RT-PCR for the *STIL-TAL1* fusion was not performed.

Demographics and laboratory tests

Demographics and clinical features were collected from electronic medical records. Laboratory tests, including a complete blood count (CBC), immunophenotyping, multiplex RT-PCR with HemaVision (DNA Diagnostic A/S, Risskov, Denmark), karyotyping, and next-generation sequencing (NGS), were conducted on bone marrow or peripheral blood specimens at the diagnostic stage. CMA using the CytoScan DX Assay (Thermo Fisher Scientific Inc, Frederick, USA) based on the GRCh37/hg19 assembly was performed to confirm a submicroscopic 1p33 deletion in the presence of *STIL-TAL1* fusion detected using multiplex RT-PCR (Figure 1).

Statistical analysis

Statistical analysis was not performed due to the small number of cases.

RESULTS

Demographics and clinical features in *STIL-TAL1*-positive and -negative T-lymphoblastic leukemia

Table 1 shows demographics and clinical features of the patients. Males were more prevalent at 73.3% (11/15) in T-ALL. All three *STIL-TAL1*-positive patients were male and under 40 years of age. On the other hand, *STIL-TAL1*-negative patients had a male to female ratio of 2:1 and were mostly over 20 years old. All T-ALL patients showed lymph node enlargement. Hepatosplenomegaly and mediastinal masses were found in all three *STIL-TAL1*-positive patients, but only in some of the *STIL-TAL1*-negative patients. CNS involvement was observed in one *STIL-TAL1*-positive patient. All *STIL-TAL1*-positive patients achieved complete remission (CR) with induction chemotherapy, and two of them remain alive without relapse. Conversely, 60.0% (6/10) of *STIL-TAL1*-negative patients achieved CR, with a recurrence rate of 50.0% (5/10) and a mortality rate of 50.0% (5/10).

Laboratory findings in *STIL-TAL1*-positive and -negative T-lymphoblastic leukemia

Table 2 presents the laboratory findings of the patients. All *STIL-TAL1*-positive patients had a WBC count of over 100 x 10^9/L, an Hb concentration of at least 10 g/dL, and a platelet count of less than 100 x 10^9/L. In contrast, most *STIL-TAL1*-negative patients had a WBC count of less than 100 x 10^9/L, an Hb concentration exceeding 10 g/dL, and a platelet count of at least 100 x 10^9/L. In immunophenotyping analysis, *STIL-TAL1*-positive patients showed higher levels of sCD3 and lower levels of CD34 expression compared to *STIL-TAL1*-negative patients. In particular, aberrant myeloid expression was only observed in *STIL-TAL1*-negative patients, not in *STIL-TAL1*-positive patients. Multiplex RT-PCR analysis indicated that *STIL-TAL1* was present in 20.0% (3/15) of all T-ALL patients, while *KMT2A-ELL* was detected in one case of *STIL-TAL1*-negative patients. Abnormal karyotypes were found in all *STIL-TAL1*-positive patients and 66.7% (8/12) of *STIL-TAL1*-negative patients.

Immunologic and genetic findings in *STIL-TAL1*-positive T-lymphoblastic leukemia

Table 3 presents detailed immunologic and genetic findings in *STIL-TAL1*-positive cases. A 63-kb 1p33 deletion leading to *STIL-TAL1* fusion was detected in all three patients through CMA, along with additional copy number abnormalities. Deletions in 7p14.1, 7q34, and 14q11.2 were considered *TCR* rearrangements, and a deletion in 9p21.3 was considered a biallelic *CDKN2A* deletion.

Table 1. Demographics and clinical features in *STIL-TAL1*-positive and -negative T-lymphoblastic leukemia.

	Total (n = 15)	STIL-TAL1-positive (n = 3)	STIL-TAL1-negative (n = 12)
Gender			
Male	11 (73.3)	3 (100)	8 (66.7)
Female	4 (26.7)	0 (0)	4 (33.3)
Age, years			
Median value (range)	32 (3 - 87)	14 (9 - 37)	49 (3 - 87)
1 - 9	2 (13.3)	1 (33.3)	1 (8.3)
10 - 19	2 (13.3)	1 (33.3)	1 (8.3)
20 - 39	4 (26.7)	1 (33.3)	3 (25.0)
≥ 40	7 (46.7)	0 (0)	7 (58.3)
Lymph node enlargement	15 (100)	3 (100)	12 (100)
Hepatosplenomegaly	11 (73.3)	3 (100)	8 (66.7)
Mediastinal mass	6 (40.0)	3 (100)	3 (25.0)
CNS involvement	1 (6.7)	1 (33.3)	0 (0.0)
CR to induction (2 N/A)	9/13 (69.2)	3 (100)	6/10 (60.0)
Relapse (2 N/A)	6/13 (46.2)	1 (33.3)	5/10 (50.0)
Death (2 N/A)	4/13 (30.8)	1 (33.3)	5/10 (50.0)

Data are presented as numbers (percentages) or medians (ranges). CNS - central nervous system, CR - complete remission, N/A - not applicable.

Table 2. Laboratory findings in *STIL-TAL1*-positive and -negative T-lymphoblastic leukemia.

	Total (n = 15)	STIL-TAL1-positive (n = 3)	STIL-TAL1-negative (n = 12)
WBC count, x 10^9/L			
Median value (range)	80.7 (2.8 - 388.9)	205.6 (197.6 - 208.8)	46.8 (2.8 - 388.9)
< 100	9 (60.0)	0 (0)	9 (75.0)
≥ 100	6 (40.0)	3 (100)	3 (25.0)
Hb, g/dL			
Median value (range)	10.7 (8.4 - 14)	11.6 (11.1 - 12.7)	9.8 (8.4 - 14.0)
< 10	7 (46.7)	0 (0)	7 (58.3)
≥ 10	8 (53.3)	3 (100)	5 (41.7)
Platelet count, x 10^9/L			
Median value (range)	93 (16 - 212)	72 (16 - 77)	109 (30 - 212)
< 100	8 (53.3)	3 (100)	5 (41.7)
≥ 100	7 (46.7)	0 (0)	7 (58.3)
Immunophenotype			
cCD3	13 (86.7)	3 (100)	10 (83.3)
sCD3	4 (26.7)	2 (66.6)	2 (16.7)
CD5	14 (93.3)	3 (100)	11 (91.7)
CD7	15 (100)	3 (100)	12 (100)
CD34	9 (60.0)	1 (33.3)	8 (66.7)
TdT	6 (40.0)	1 (33.3)	5 (41.7)
Myeloid (CD13, 33, 117)	7 (46.7)	0 (0)	8 (66.7)

Table 2. Laboratory findings in *STIL-TAL1*-positive and -negative T-lymphoblastic leukemia (continued).

	Total (n = 15)	STIL-TAL1-positive (n = 3)	STIL-TAL1-negative (n = 12)
Multiplex RT-PCR			
Detected	4 (26.7)	3 (100) STIL-TAL1	1 (9.1) KMT2A-ELL
Not detected	11 (73.3)	0 (0)	11 (91.7)
Karyotype			
Normal	4 (26.7)	0 (0)	4 (33.3)
Abnormal	11 (73.3)	3 (100)	8 (66.7)

Data are presented as numbers (percentages) or medians (ranges). WBC - white blood cell, Hb - hemoglobin, RT-PCR - reverse transcriptase-polymerase chain reaction.

Table 3. Immunologic and genetic findings in *STIL-TAL1*-positive T-lymphoblastic leukemia.

No. case	Gender/ Age	Immuno-phenotype (positive results)	Karyo-type	NGS (Tier 1 or 2)	CMA results	CMA interpretation	Follow-up
1	M/37 years	cCD3, CD3, CD5, CD7, TdT	46, XY, del(6) (q21q23) [8]/46, XY [13]	No variant	1p33(47,700,108-47,763,403) x 1 6q14.1q16.1(77,124,674-95,680,018) x 1 7p14.1(38,293,949-38,395,208) x 0 7q34(142,016,358-142,493,639) x 1 9p21.3(21,828,109-21,989,103) x 0 9p24.3p13.1(192,128-40,087,758) x 2 hmz	Deletion in 1p33 (63 kb) Deletion in 6q14.1q16.1 (18.6 Mb) Deletion in 7p14.1 (101 kb) Deletion in 7q34 (477 kb) Deletion in 9p21.3 (161 kb) LOH in 9p24.3p13.1 (39.9 Mb)	Relapse to death after allogenic PBSCT
2	M/9 years	cCD3, CD5, CD7	46, XY, add(2) (q33) [5]/46, XY [15]	FBWX7, NOTCH1, NRAS	1p33 (47,700,108-47,763,403) x 1 7p14.1(38,293,949-38,335,520) x 0 7q34(142,099,012-142,343,464) x 1 7q34(142,349,238-142,493,639) x 0 9p21.3(20,731,90-21,701,432) x 1 9p21.3(21,701,673-22,341,594) x 0 9p21.3p21.2(22,342,372-27,090,109) x 1 9p24.2p24.1(4,353,553-5,024,532) x 1 14q11.2(22,479,811-22,917,593) x 0	Deletion in 1p33 (63 kb) Deletion in 7p14.1 (41 kb) Deletion in 7q34 (244 kb) Deletion in 7q34 (144 kb) Deletion in 9p21.3 (969 kb) Deletion in 9p21.3 (639 kb) Deletion in 9p21.3p21.2 (4.7 Mb) Deletion in 9p24.3p24.1 (671 kb) Deletion in 14q11.2 (437 kb)	CR
3	M/14 years	cCD3, CD3, CD5, CD7, CD34	46, XY, t(11;14) (p13;q11.2) [17]/46, XY [3]	PTEN	1p33(47,700,162-47,763,403) x 1 7p14.1(38,293,949-38,371,334) x 1 9p21.3(21,828,109-21,993,964) x 0 9p24.3p21.1(192,128-33,103,872) x 2 hmz 10q22.1q26.3(71,632,409-135,426,536) x 2 hmz	Deletion in 1p33 (63 kb) Deletion in 7p14.1 (77 kb) Deletion in 9p21.3 (165 kb) LOH in 9p24.3p24.1 (32.9 Mb) LOH in 10q22.1q26.3 (63.8 Mb)	CR

M - male, NGS - next generation sequencing, CMA - chromosomal microarray analysis, LOH - loss of heterozygosity, CR - complete remission, PBSCT - peripheral blood stem cell transplantation.

DISCUSSION

The *TAL1* gene on chromosome 1p33 is a frequent target for chromosomal translocation, interstitial deletion, or mutation in T-ALL [6]. A submicroscopic interstitial deletion on chromosome 1p33 results in the fusion of 5' untranslated region of *STIL* with the coding region of *TAL1*, which is located only 18 kb away from *STIL* [8]. This deletion places *TAL1* expression under the control of the *STIL* promoter, resulting in aberrant overexpression of *TAL1* and thus promoting T cell leukemogenesis [7,8]. This intrachromosomal rearrangement known as

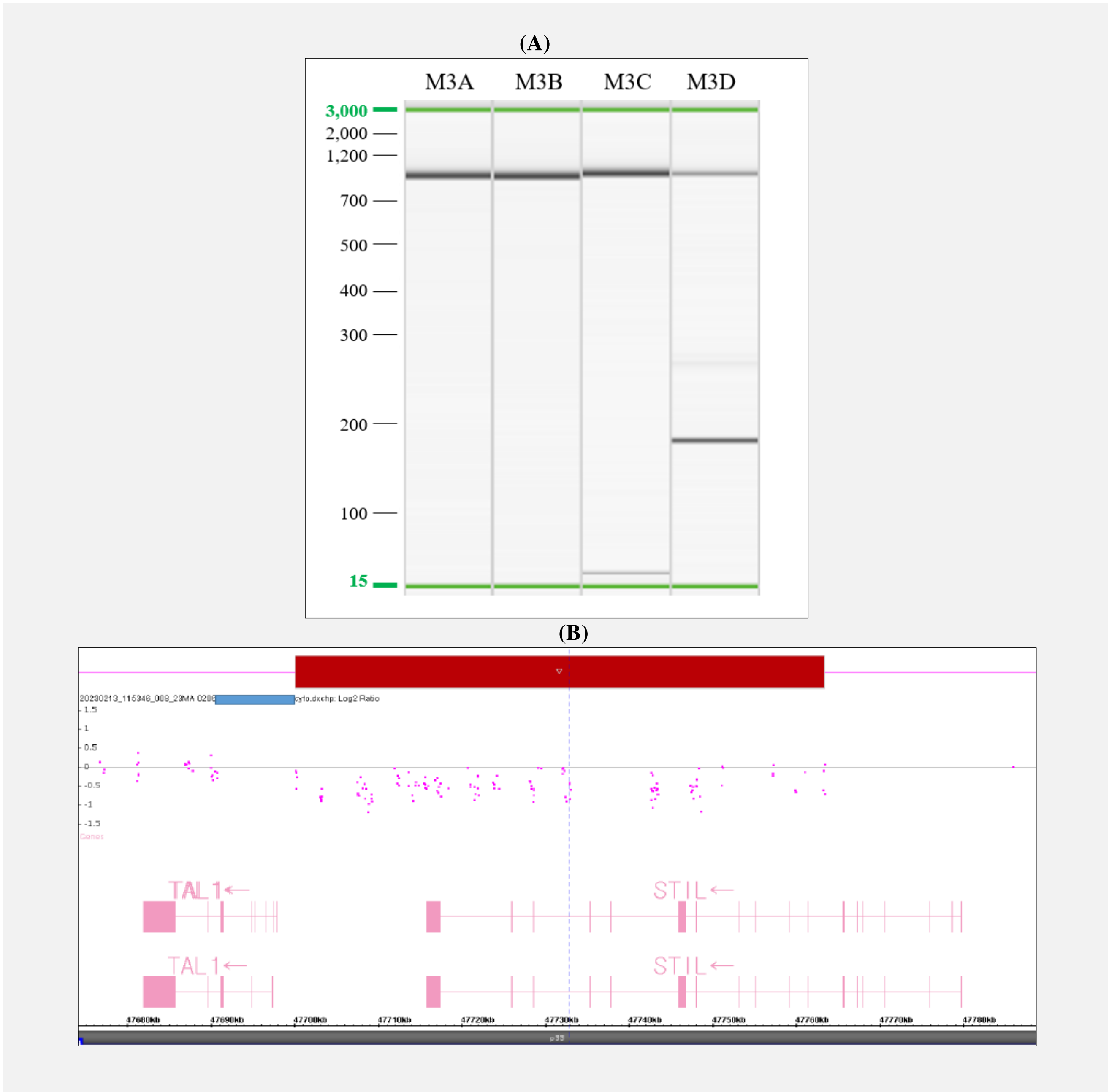

Figure 1. Detection of submicroscopic 1p33 deletion resulting in the *STIL-TAL1* fusion.

Multiplex RT-PCR revealed a single 186-base pair band representing the *STIL-TAL1* fusion transcript in the M3D lane (A). Chromosomal microarray analysis showing a 63-kb deletion on chromosome 1p33 resulting in the *STIL-TAL1* fusion (B).

'*STIL-TAL1* fusion' is observed in 15 - 25% of pediatric and young adult T-ALL cases, but much less frequently in older T-ALL patients [8]. A previous study in India has reported *STIL-TAL1* fusion in 15.4% (4/26) of pediatric cases and none of the seven adult T-ALL cases analyzed [9]. Two other studies have found *STIL-TAL1* fusion in 15.6% (56/359) and 15.4% (20/130) of T-ALL cases [7,10]. In our study, we detected *STIL-TAL1* fusion in 20.0% (3/15) of T-ALL cases, consistent with previous reports. In addition, *STIL-TAL1* fusion was found in 50% (2/4) in those under the age of 20, but only in 9.1% (1/11) of adults over the age of 20, and none of the seven adults over 40. These findings align with previous findings indicating a high prevalence of *STIL-TAL1* fusions in children and young adults.

T-ALL with *STIL-TAL1* fusion is associated with distinct laboratory characteristics, such as high WBC count and hemoglobin levels, as well as a mature cortical T-cell immunophenotype (sCD3 positive) [2,6,8]. This study found that all patients with *STIL-TAL1* fusion ex-

hibited higher WBC counts and hemoglobin levels, but lower platelet counts compared to *STIL-TAL1*-negative patients. The immunophenotype of *STIL-TAL1* positive cases showed more sCD3 expression and less CD34 expression compared to negative cases, indicating a more mature T-cell lineage. Notably, aberrant myeloid expression was only observed in *STIL-TAL1*-negative patients, not in *STIL-TAL1*-positive patients. Early T-cell precursor (ETP) ALL is a subtype of T-ALL, representing 5 - 15% of T-ALL cases, characterized by a distinct immature phenotype with the expression of stem cell and/or myeloid markers, and is associated with a poorer prognosis compared to other T-ALL subtypes [2, 4,5]. In the present study, ETP-ALL accounted for more than half of the *STIL-TAL1*-negative cases, suggesting that ETP-ALL is negative for the *STIL-TAL1* fusion and may have a significant impact on the overall prognosis of *STIL-TAL1*-negative cases.

In this study, all three patients with *STIL-TAL1* fusion showed lymph node enlargement, hepatosplenomegaly, and mediastinal mass, while *STIL-TAL1*-negative patients only partially showed these findings. In particular, central nervous system invasion was observed only in *STIL-TAL1*-positive cases. Despite high-risk factors such as a high WBC count, organomegaly, and CNS invasion, all three *STIL-TAL1*-positive patients responded well to induction chemotherapy, and two of them have survived without recurrence or death so far, suggesting a favorable prognosis. On the other hand, only some of the *STIL-TAL1*-negative patients responded to induction therapy, and half of them experienced relapse and death. A study based on European Organization for Research and Treatment of Cancer (EORTC) trials has reported that those with SIL-TAL fusion had low levels of residual disease at the end of induction and a favorable prognosis [11]. However, other studies have reported either unfavorable or no difference in prognosis for *STIL-TAL1*-positive patients [6,7,12]. The prognosis of this rearrangement remains controversial.

Chromosome deletions, as a mechanism for fusion gene formation, are mostly submicroscopic. Therefore, they are not detected by cytogenetic analyses but instead are identified through array comparative genomic hybridization and/or high-throughput RNA sequencing [8]. In 1990, two independent research groups studying T-ALL used southern analysis to detect an approximately 90-kb interstitial deletion in 1p33 that caused *STIL-TAL1* fusion, and this was the first description of a fusion gene resulting from an interstitial, submicroscopic deletion [8]. Yu and colleagues performed a microarray analysis and found a submicroscopic deletion of 77 - 86 kb in 1p33, leading to a *STIL-TAL1* fusion in 9 of 22 pediatric T-ALL patients [13]. In the present study, we first detected *STIL-TAL1* fusion using a multiplex RT-PCR test, included in the diagnostic workup for acute leukemia. Although all three cases with *STIL-TAL1* fusions showed abnormal karyotypes, chromosomal analysis did not reveal a 1p33 deletion. We then performed CMA and detected 63-kb deletions in 1p33.

In addition, CMA results revealed T cell receptor rearrangements (deletions at 7p14.1, 7q34, or 14q11.2) and biallelic deletion of *CDKN2A* (deletion at 9p21.3), which were not identified through karyotyping. The spectrum of genetic abnormalities in T-ALL is highly heterogeneous and diverse. Chromosomal translocations of transcription factor oncogenes to the regulatory regions of T-cell receptor (*TCR*) genes are characteristic of T-ALL, with about 50% of patients exhibiting chromosomal translocations involving 14q11 (*TCR* alpha and *TCR* delta) and 7q34 (*TCR* beta), leading to oncogene overexpression [1,4]. Deletion of the cyclin-dependent kinase inhibitor 2A (*CDKN2A*) locus on chromosome 9p21, which encompasses the *p16/INK4A* and *p14/ARF* suppressor genes, is present in up to 70% of T-ALL cases, leading to dysregulated proliferation [1,4, 14].

According to NGS results, this study found additional genetic alterations such as *NOTCH1*, *FBWX7*, and *PTEN* mutations. *NOTCH1* mutation is commonly found in 60 - 70% of T-ALL cases, while *FBWX7* and *PTEN* mutations are found in 15% and 10% of cases, respectively [1,4]. A previous study on single-cell genetics in *STIL-TAL1*-positive T-ALL has shown that *STIL-TAL1* fusion and *CDKN2A* loss are both early or truncal events, in contrast to other recurrent genetic changes including *NOTCH1* and *PTEN* mutations, which are secondary and subclonal [15]. In addition, the study concluded that there is a strong selective pressure between *PTEN* inactivation/loss, *STIL-TAL1* fusion, and *CDKN2A* loss. Therefore, these genetic alterations, including *STIL-TAL1* rearrangement, could play a significant role in minimal residual disease (MRD) monitoring and identifying new therapeutic targets [15,16].

In conclusion, the present study identified the *STIL-TAL1* rearrangement resulting from submicroscopic 1p33 deletion in T-ALL, as well as the unique clinical, immunologic, and genetic characteristics of T-ALL patients with the *STIL-TAL1* fusion. The results demonstrated that PCR, NGS, and microarray analyses allow for a more comprehensive evaluation of complex genetic abnormalities in T-ALL with the *STIL-TAL1* fusion. Further multi-center studies are needed, including extensive cytogenetic and molecular analyses, to determine the pathophysiology, characteristics, clinical significance, novel therapeutic targets, and MRD monitoring of this gene rearrangement.

Ethical Approval:
The protocol of this study was reviewed and approved by the Institutional Review Board of Kyungpook National University Hospital (approval no. 2024-02-017).

Declaration of Interest:
The authors declare that they have no competing interests.

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