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CASE REPORT

# Acute myelomonocytic leukemia and T-lymphoblastic lymphoma as simultaneous bilineage hematologic malignancy treated with decitabine: A case report

So-Yeon Jeon, Na-Ri Lee, Seungah Cha, Ho-Young Yhim, Jae-Yong Kwak, Kyu Yun Jang, Namsu Kim, Yong Gon Cho, Chang-Hoon Lee

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## Abstract

*BACKGROUND*

Simultaneous bilineage hematologic malignancies are rare; however, several cases of acute myeloid leukemia (AML) and T-lymphoblastic lymphoma (T-LBL) co-occurrence have been reported. A standard treatment for simultaneous AML and T-LBL has not yet been established, and its prognosis is very poor. Further studies to develop standard treatments are required to increase patient survival rates.

*CASE SUMMARY*

A 69-year-old man complaining of pleuritic chest pain visited the emergency room. Computed tomography revealed multiple enlarged lymph nodes (LNs) in the neck and groin and pulmonary thromboembolism with pulmonary infarction. Furthermore, a peripheral blood smear performed due to leukocytosis revealed circulating blasts. Acute myelomonocytic leukemia (AMML) was diagnosed after bone marrow examination, and T-LBL positivity for terminal deoxynucleotidyl transferase, cluster of differentiation (CD)34, and CD4 was confirmed by cervical LN biopsy. Decitabine and dexamethasone were administered because he could not receive intensive chemotherapy due to poor performance status. Complete remission of AMML and T-LBL was achieved after 4 cycles of decitabine plus dexamethasone.

*CONCLUSION*

We report the therapeutic effect of decitabine, a hypomethylating agent (HMA), in patients with concurrent bilineage hematologic malignancies and suggest that further studies are required to evaluate the therapeutic effect of HMAs on both lymphoid and bilineage hematologic malignancies.

**Key Words:** Simultaneous bilineage hematologic malignancies; Acute myelomonocytic leukemia; T-lymphoblastic lymphoma; Decitabine; Pulmonary thromboembolism; Case report



**Core Tip:** Simultaneous bilineage hematologic malignancies are rare. Here, we report an unusual case of concurrent acute myelomonocytic leukemia (AMML) and T-lymphoblastic lymphoma (T-LBL), treated with decitabine. After the fourth cycle of decitabine and dexamethasone administration, a patient achieved complete remission of AMML and T-LBL. Hypomethylating agents (HMAs) have been approved for the treatment of myelodysplastic syndrome and intensive induction-ineligible acute myeloid leukemia; however, their therapeutic effect on lymphoid malignancy has not yet been sufficiently investigated. This case suggests the need of further studies to evaluate the therapeutic effects of HMAs on lymphoid and bilineage hematologic malignancies.

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## INTRODUCTION

Simultaneous bilineage hematologic malignancies are extremely rare and their pathogenesis remains unclear. This disease has a poor prognosis, and a standard chemotherapy regimen has not yet been established. According to an analysis of several case reports, intensive chemotherapy followed by hematopoietic stem cell transplantation has shown better outcomes; however, treatment remains challenging[1].

DNA methylation is a mechanism of DNA modification that suppresses gene transcription, and methylation of tumor suppressor genes contributes to cancer cell growth and survival. Hypomethylating agents (HMAs) have been approved for the treatment of myelodysplastic syndrome and acute myeloid leukemia (AML), and a combination of HMAs and venetoclax, a B-cell lymphoma 2 (Bcl-2) inhibitor has been reported to improve survival outcome in patients with intensive chemotherapy-ineligible AML.

Here, we report a case of concurrent acute myelomonocytic leukemia (AMML) and T-lymphoblastic lymphoma (T-LBL), treated with decitabine.

## CASE PRESENTATION

### *Chief complaints*

A 69-year-old man with chest wall pain visited the emergency room.

### *History of present illness*

The patient presented with pain in the right chest wall that worsened with breathing. The pain suddenly occurred the day before the visit and gradually worsened thereafter. He was examined at a primary medical center before visiting our hospital and was suspected to have pneumonia.

### *History of past illness*

The patient had taken clopidogrel after being diagnosed with cerebral infarction.

### *Personal and family history*

The patient had no relevant family history.

### *Physical examination*

Physical examination revealed multiple enlarged lymph nodes (LNs) in the neck and groin with a body temperature of 38.5 °C.

### *Laboratory examinations*

Initial testing showed leukocytosis (white blood cell: 156900/μL, normal range: 4000-10000/μL), anemia (Hb: 8.3 g/dL, normal range: 13-16.5 g/dL), and thrombocytopenia (platelet: 67000/μL, normal range: 150000-450000/μL). Peripheral blood smear revealed leukocytosis with blast cells (95%). C-reactive protein (142.29 mg/L, normal range: 0-5.0 mg/L), lactate dehydrogenase (1193 U/L, normal range: 0-250 U/L), and d-dimer (2.2 mg/L FEU, normal range: 0-0.49 mg/L FEU) levels were elevated.

### *Imaging examinations*

Computed tomography (CT) revealed multiple enlarged LNs on both sides of the diaphragm (cervical, axillary, mediastinal, inguinal, and external iliac LNs) (Figure 1A and B). Acute pulmonary thromboembolism (PTE) with pulmonary infarction and pneumonia were detected in the right and left lungs, respectively. A thrombus was detected in the thoracic aorta (Figure 1C and D).

## FINAL DIAGNOSIS

Bone marrow examination after leukapheresis revealed AMML; positive for immunophenotypes of cluster of differentiation (CD)13, CD33, CD34, CD64, human leukocyte antigen-DR, and myeloperoxidase (MPO) and cytochemical staining of MPO, alpha-naphthyl butyrate esterase (ANBE), chloroacetate esterase, and acid phosphatase (Figure 2). Cervical lymph node biopsy confirmed T-LBL diagnosis; positive for CD4, CD34, and terminal deoxynucleotidyl transferase (TdT) and negative for CD5, CD7, CD8, CD19, CD20, CD56, and MPO (Figure 3). It was negative for T-cell markers such as CD5, CD7, and CD8; however, positive for immunophenotypes of T-LBL, TdT, and CD34 and another T-cell marker, CD4. Additionally, B-cell, natural killer cell, and myeloid markers were negative, so malignant cell infiltration caused by other lineages could be excluded. We performed next-generation sequencing (NGS) with bone marrow sample, and a RUNX1 (M466I) and TET2 (E135K) mutations were detected. We also performed fluorescence in situ hybridization analysis to evaluate cytogenetic abnormalities, including platelet-derived growth factor receptor alpha (PDGFRA) and platelet-derived growth factor receptor beta (PDGFRB) rearrangements; however, no abnormal cytogenetic abnormalities were detected. Chromosome analysis was performed with a bone marrow sample and revealed normal male karyotype; 46, XY.

## TREATMENT

Due to the poor performance status and comorbidities, the patient could not undergo intensive chemotherapy; instead, decitabine (20 mg/d for 5 d) and dexamethasone (40 mg for 4 d) were administered. He underwent anticoagulant therapy with dalteparin (200 IU/kg) followed by apixaban (10 mg twice daily for 7 d and then 5 mg twice daily) for PTE and thrombus treatment. Bone marrow examination was performed after four cycles, and complete remission (CR) was achieved. CT confirmed complete resolution of the PTE (Figure 1E and F), and positron emission tomography/CT revealed no remnants of lymphoma (Figure 1G).

## OUTCOME AND FOLLOW-UP

The patient received 13 cycles of decitabine without any complications; however, AMML recurred 14 mo after treatment initiation. He received chemotherapy with idarubicin (12 mg/m$^2$ for 2 d) and cytarabine (200 mg/m$^2$ for 5 d); however, he failed to respond and died due to disease progression and pneumonia. The progression-free survival and overall survival rates were 14 and 19 mo, respectively.

## DISCUSSION

Simultaneous bilineage hematologic malignancies have a poor prognosis, with a median survival time of 15 mo[1]. Only 24 cases have been reported since 1976, and concurrent AML and T-LBL and chronic myeloid leukemia and T-LBL were the most common[1]. Its pathogenesis remains unclear; however, Tsukasaki *et al*[2] reported two possible mechanisms: First, AML develops primarily due to compromised immunity in patients with T-cell lymphoma. Moreover, increased levels of growth factors, such as granulocyte-colony stimulating factor, macrophage-colony stimulating factor, and granulocyte-macrophage colony-stimulating factor, produced by lymphoma cells result in AML[2]. PDGFRA and PDGFRB rearrangements are associated with myeloid/lymphoid neoplasms. Chang *et al*[3] reported a case of concurrent AML and T-LBL with PDGFRB rearrangement. We performed a fluorescence in situ hybridization analysis; however, PDGFRA and PDGFRB rearrangements were not detected. In contrast, RUNX1 and TET2 mutations were detected by NGS conducted with bone marrow samples. Since RUNX1 plays a role in hematopoietic stem cell differentiation, somatic mutations and chromosomal rearrangements involving this gene contribute to hematologic malignancy development[4, 5]. RUNX1 mutations promote leukemogenesis by offering growth advantages and differentiation defects to hema-

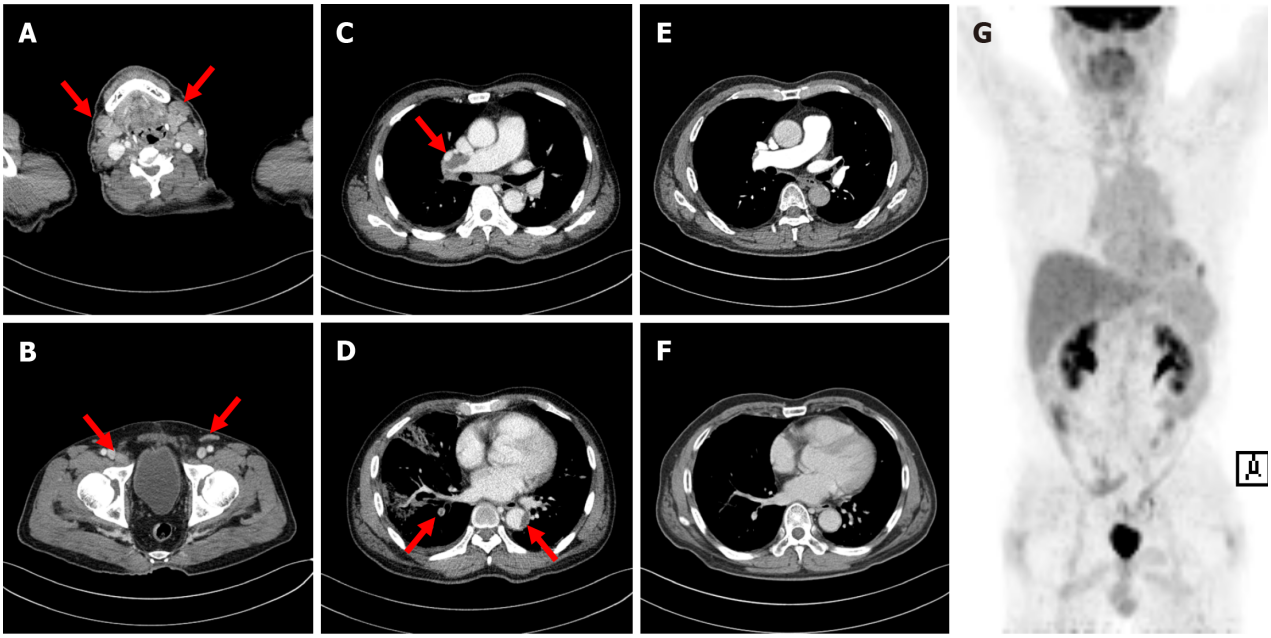


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**Figure 1 Imaging examination of the T-lymphoblastic lymphoma and pulmonary thromboembolism.** A-D: Multiple enlarged lymph nodes (A and B) and thromboembolism (C and D) visible on computed tomography (CT); E-G: CT (E and F) and positron emission tomography/CT (G) confirm the complete resolution of thromboembolism and complete response of T-lymphoblastic lymphoma, respectively.

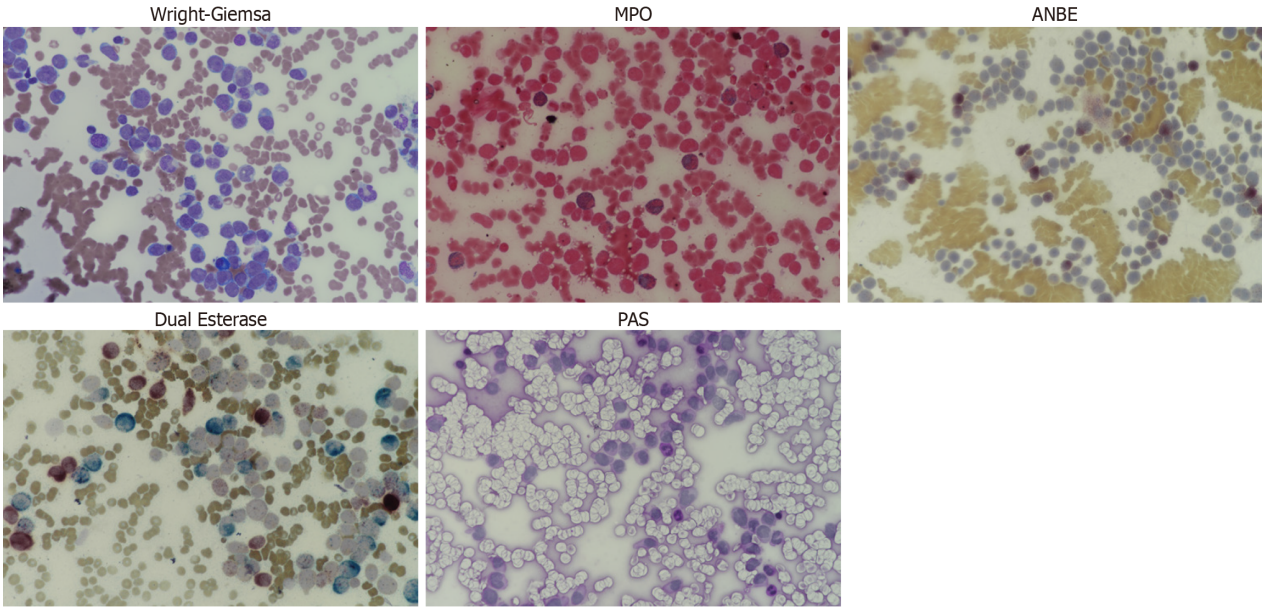


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**Figure 2 Bone marrow findings.** 87% of all nucleated cells in the bone marrow are leukemic blasts. Cytochemically, the leukemic cells are positive for myeloperoxidase, myeloid marker and alpha-naphthyl butyrate esterase, monocyte marker. Dual esterase stain including alpha-naphthyl acetate esterase, another nonspecific esterase and chloroacetate esterase reveals that leukemic cells are derived from monocytes. While leukemic cells are negative for Periodic acid-Schiff stain. MPO: myeloperoxidase; ANBE: alpha-naphthyl butyrate esterase; PAS: Periodic acid-Schiff.

topoietic progenitor cells. Somatic mutation of RUNX1 is detected in 15%-30% of adult de novo patients with AML, and recent studies reported that acute lymphoblastic leukemia (ALL) is accompanied by recurrent somatic mutation of RUNX1 as well[4]. Grossman *et al*[6] reported that 128 patients with T-ALL were analyzed for RUNX1 mutations, which were detected in 15 patients. Moreover, Della Gatta *et al*[7] reported that RUNX1 mutation is a key mediator of T Cell Leukemia Homeobox 1 and T Cell Leukemia Homeobox 3 induced T-ALL development. TET2 has anti-tumor activity *via* catalyzing DNA demethylation. Its mutation is well known epigenetic abnormality which contributes to the development of myeloid malignancies; however, it has been reported that TET2 mutation and other genetic abnormalities of epigenetic regulators such as DNMT3A, IDH2, and KMT2D are also associated with mature T-cell lymphoma. Among them, TET2

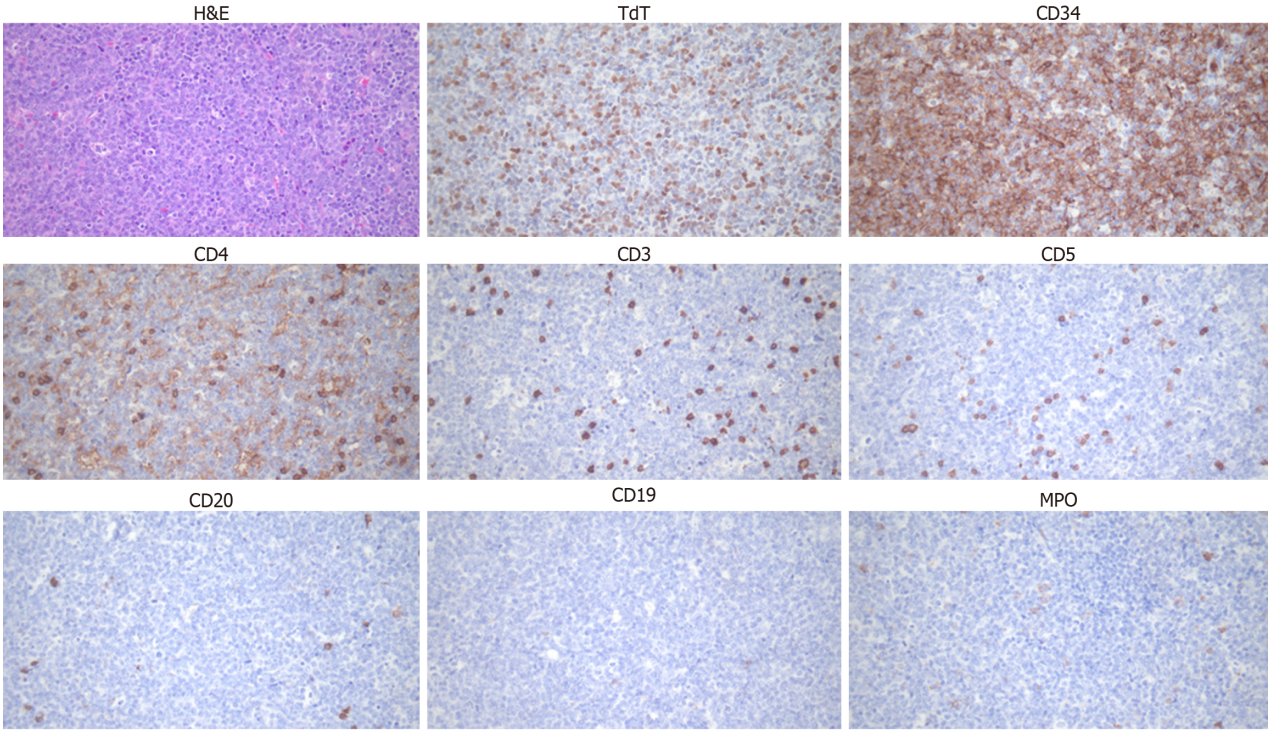


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**Figure 3 Histologic findings.** Tumor cells appear as a sheet and are medium-to-large-sized. Immunohistochemically, the tumor cells are moderately positive for terminal deoxynucleotidyl transferase, diffusely positive for cluster of differentiation (CD)34, weakly positive for CD4, and negative for CD3, CD5, CD20, CD19, and myeloperoxidase. H&E: Hematoxylin and eosin; TdT: Terminal deoxynucleotidyl transferase; CD: Cluster of differentiation; MPO: Myeloperoxidase.

and IDH2 mutations are the most frequent[8]. If identical mutations were detected in NGS analysis using an LN biopsy sample, it could be assumed that AMML and T-LBL were derived from the common leukemic stem cells. However, unfortunately, we could not perform NGS with the LN biopsy sample, so we have a limitation in determining whether the two diseases were derived from the same progenitor. Nevertheless, since T-LBL also showed a good response to decitabine, epigenetic abnormalities are expected to accompany it. Further studies are required to evaluate whether epigenetic abnormalities contribute to T-LBL development.

Since T-LBL cells usually invade the bone marrow, accurate lineage analysis in bone marrow specimen is required. Moreover, differential diagnosis of mixed-phenotype acute leukemia should be considered as well. Cytochemical staining results showed strong positivity for MPO, and monocyte marker, ANBE, while Periodic acid-Schiff stain, a marker of lymphoblast, was negative. Furthermore, immunofluorescence assay revealed that 78.06%, 99.68%, and 99.34% of bone marrow cells showed positivity for MPO, CD13, and CD33, respectively; 28% of cells were positive for CD64, a monocyte marker as well. These results imply that leukemic cells infiltrated in the bone marrow are derived from myeloid lineage, and the possibility that malignant T-cells invade the bone marrow was expected to be low.

Standard chemotherapy for simultaneous bilineage hematologic malignancies has not yet been established. However, a preliminary study showed that chemotherapy followed by transplantation was superior to chemotherapy without transplantation[1]. Methylation of gene promoters results in gene silencing, and aberrant methylation of tumor suppressor genes is associated with malignancy development such as AML. HMAs, including azacitidine and decitabine, have been approved for AML and myelodysplastic syndrome treatment; however, genetic methylation also plays an important role in lymphoid malignancy development[9,10]. Furthermore, decitabine and azacitidine have been reported to exert antileukemic effects on ALL[11-13]. The patient achieved CR after four cycles of decitabine and dexamethasone. Dexamethasone was administered in combination with decitabine to suppress T-lymphoma cells and was discontinued after the fourth cycle. The patient maintained the CR until the 13th cycle. Considering the therapeutic effects of HMA on both myeloid and lymphoid malignancies[9,10], it may be used to treat patients with concurrent AML and T-lymphoid malignancy as well as those with concurrent AML and B-lymphoid malignancy who cannot receive intensive chemotherapy. Particularly, if the epigenetic abnormality is present in both diseases, it is expected to respond well to HMA. However, further large-scale studies are required to provide a clear rationale for whether HMAs have anticancer effects in both lymphoid and bilineage hematologic malignancies. The combination of HMAs with venetoclax, a Bcl-2 inhibitor, has been approved as an induction therapy for elderly patients with AML who are ineligible for intensive chemotherapy. Patients who received venetoclax with either decitabine or azacitidine achieved 67% of CR or CR with incomplete count recovery rates, and the median overall survival was 17.5 mo[14]. Venetoclax has an anti-apoptotic effect, and its therapeutic efficacy has been reported in B-cell malignancies, such as chronic lymphocytic leukemia. Therefore, it is also expected to have anti-leukemic effects on T-lymphoid malignancy. The combination of venetoclax and HMAs is expected to have better efficacy than HMA monotherapy for bilineage hematologic malignancies; however, further research is required. Unfortunately, the patient was unable to receive venetoclax due to the medical reimbursement issue at the time.

## CONCLUSION

Here, we present an unusual case of concurrent AMML and T-LBL with thromboembolism. Due to the poor performance status and comorbidities, the patient received decitabine and dexamethasone instead of intensive chemotherapy, and strikingly, a CR was achieved. We suggest that decitabine may be a good treatment option for unfit patients diagnosed with bilineage hematologic malignancies. Further research is required to confirm the therapeutic efficacy of HMAs in both lymphoid and bilineage hematologic malignancies.

## FOOTNOTES

**Author contributions:** Jeon SY wrote the manuscript; Lee NR, Cha S, Jang KY, Kim N, and Cho YG collected and validated the data; Yhim HY and Kwak JY revised the manuscript; Lee CH was in charge of patient treatment and designed the manuscript; all authors have read and approved the final manuscript.

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