

Original Article

Cladribine Treatment Outcomes in Hairy Cell Leukemia:
A Single-center Experience

Seema Seçilmiş, Burcu Aslan Candır, Samet Yaman, Ersin Bozan, Bahar Uncu Ulu, Tuğçe Nur Yiğenoğlu, Dicle İskender, Merih Kızıl Çakar

University of Health Sciences Türkiye, Dr. Abdurrahman Yurtaslan Ankara Oncology Training and Research Hospital, Clinic of Hematology and Bone Marrow Transplantation Center, Ankara, Türkiye

ABSTRACT

Aim: Hairy cell leukemia (HCL) is a rare, indolent, chronic B-cell lymphoproliferative disorder. This study aimed to assess the clinical features, treatment responses, and outcomes of patients with HCL monitored at our institution.

Methods: We conducted a retrospective cohort study of 25 patients diagnosed with HCL who were followed at our center. Data on demographics, presenting symptoms, laboratory findings, immunophenotypic characteristics, treatment responses, and long-term follow-up were reviewed.

Results: The median age at diagnosis was 48 years, and most patients were male. Fatigue, abdominal fullness, and abdominal pain were the leading presenting symptoms. All patients had splenomegaly at diagnosis. All patients received cladribine as first-line therapy, achieving complete remission (CR) in 92.0% and partial remission in 8.0% of cases. During a median follow-up of 36.9 months after CR, three patients (12%) experienced relapse, but subsequently achieved CR following re-treatment with cladribine. No treatment-related deaths or secondary malignancies were recorded.

Conclusion: Cladribine continues to be a reliable and well-tolerated initial therapy for patients with HCL. In those who relapse, retreatment with cladribine is effective, reinforcing its established role in disease management.

Keywords: Hairy cell leukaemia, cladribine, prognosis, purine nucleoside analog

Introduction

Hairy cell leukemia (HCL) is a rare B-cell malignancy with an indolent course, first identified by Bouroncle et al. [1]. It is typically characterized by pancytopenia and splenomegaly and constitutes nearly 2% of all leukemia cases. The disease usually appears around the age of 55 and is 3-4 times more common in men than in women [2]. In many patients, long-lasting cytopenias are the first sign, but may remain undetected for years [2]. With more frequent peripheral blood testing, hairy cells can now be detected even in asymptomatic patients. In 5-10% of these cases, a watch-and-wait approach is used, with regular follow-up until treatment is needed [3,4].

Treatment becomes necessary when symptoms appear or blood counts continue to drop. While HCL usually progresses

slowly, most patients eventually need therapy because of worsening cytopenias or an enlarged, symptomatic spleen [5]. A major advance in HCL treatment occurred in the early 1990's with the introduction of purine analogs such as cladribine [2-chlorodeoxyadenosine, (2-CdA)] [6] and pentostatin (2'-deoxycoformycin) [7,8]. These drugs are now the first-line treatment for suitable patients, leading to durable complete remissions (CR) and longer survival in those without active infections and with good performance status [9-12].

This study aimed to evaluate the clinical features and treatment outcomes of patients with HCL managed at our center.

Address for Correspondence: Seema Seçilmiş MD, University of Health Sciences Türkiye, Dr. Abdurrahman Yurtaslan Ankara Oncology Training and Research Hospital, Clinic of Hematology and Bone Marrow Transplantation Center, Ankara, Türkiye

E-mail: secilmissema1@gmail.com **ORCID ID:** orcid.org/0000-0003-2957-2339

Received: 09.07.2025 **Accepted:** 08.11.2025 **Epub:** 14.11.2025 **Publication Date:** 12.12.2025

Cite this article as: Seçilmiş S, Aslan Candır B, Yaman S, et al. Cladribine treatment outcomes in hairy cell leukemia: a single-center experience. Acta Haematol Oncol Turc. 2025;58(3):212-217



Methods

The study was approved by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Dr. Abdurrahman Yurtarslan Ankara Oncology Health Application and Development Center of the (decision no: 2022-02/1651, date: 09.02.2022). All procedures were carried out in accordance with the ethical principles of the 1964 Helsinki Declaration.

This retrospective cohort study included 25 patients with classic HCL diagnosed between January 2010 and February 2022. Cases with variant HCL were excluded. Demographic characteristics, initial symptoms, physical examination findings, complete blood counts, biochemical test results, bone marrow biopsy reports, and peripheral blood smear evaluations were reviewed. Treatment responses, side effects, and relapses were also analyzed.

The diagnosis was made using peripheral blood smear, bone marrow morphology, and immunophenotypic and immunohistochemical findings [13,14].

Identifying typical “hairy” lymphoid cells under the microscope was key to the diagnosis. Imaging methods, such as computed tomography or ultrasound, were used to evaluate organ enlargement and lymph node involvement [5]. All patients underwent bone marrow aspiration and biopsy, and flow cytometry was used to confirm the diagnosis. This technique aided the identification of leukemic cells through detection of B-cell markers [Cluster of Differentiation 19 (CD19), CD20, CD22] and antigens such as CD11c, CD25, CD103, and CD123 [15,16].

Immunohistochemical staining of the bone marrow also supported the diagnosis [17,18]. Markers such as tartrate-resistant acid phosphatase (TRAP), annexin-1, and reticulin were used. Treatment was initiated in patients with cytopenias [Hemoglobin (Hb) <11 g/dL, white blood cell <1,000/ μ L, platelets <100,000/ μ L], symptomatic splenomegaly, systemic complaints, or frequent infections [5]. All patients had normal kidney and liver function and had no uncontrolled infections.

All patients were treated with the purine analog cladribine (2-CdA) as first-line therapy at a dose of 0.14 mg/kg/day subcutaneously for five days. Patients were monitored with daily temperature measurements and serial blood counts until the blood counts returned to normal.

Treatment response was assessed by physical examination, blood counts, and measurement of spleen size. Because bone marrow recovery after purine analog therapy can take several months, a repeat bone marrow biopsy was performed 4 to 6 months after treatment to confirm the clearance of leukemic cells [14]. CR was defined as normalized blood counts (Hb>11 g/dL without transfusion, platelets >100,000/ μ L, absolute neutrophil count>1500/ μ L), absence of organomegaly or lymphadenopathy, and absence of visible hairy cells in the blood or bone marrow. Partial remission (PR) was characterized by near-normalization of peripheral blood counts, accompanied by at least a 50% reduction in organomegaly and in bone marrow infiltration by hairy cells. Patients who did not meet the criteria for either

CR or PR were classified as non-responders. Hematologic relapse refers to the recurrence of cytopenias. Morphologic relapse was defined as the reappearance of leukemic cells on smears or in biopsies without accompanying peripheral blood cytopenias. While morphologic relapse alone did not always require treatment, hematologic relapse was assessed based on symptoms and lab findings to decide on further management [14,19].

Overall survival (OS) was measured from diagnosis to death from any cause or to last follow-up. After treatment, patients were monitored for neutropenic fever and possible treatment-related side effects.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous data were presented as medians with ranges, while categorical data were presented as percentages. OS and progression-free survival (PFS) were calculated using the Kaplan-Meier method.

Results

Patient Characteristics

This study analyzed 25 individuals diagnosed with HCL. Among them, 64% (n=16) were male and 36% (n=9) were female. The median age at diagnosis was 48 years, ranging from 27 to 68 years. The most frequent initial complaints included fatigue (84%), abdominal fullness (56%), and abdominal pain (48%). Only one patient (4%) had no symptoms at diagnosis and was referred to hematology following the detection of cytopenias during routine tests.

Splenomegaly was present in all patients at diagnosis, and 32% (n=8) had massive enlargement. Pancytopenia was observed in 72% (n=18) of patients. Hairy cells were identified in 88% (n=22) of peripheral blood smears, with a median infiltration rate of 30% (range: 0-80%). On bone marrow examination, the median infiltration by hairy cells was 70% (range, 5-99%).

All patients expressed CD19, CD20, and TRAP. CD103 was positive in 96% of cases, and CD11c and CD25 were positive in 92% of cases. Annexin A1 expression was detected in 88% of patients. The BRAF V600E mutation was tested for in four individuals, all of whom were positive. Due to technical constraints, this analysis could not be performed on the remainder of the cohort. A detailed overview of the demographic and clinical features is shown in Table 1.

Treatment Outcomes

All patients received cladribine as first-line therapy. Among them, 23 (92.0%) achieved CR, and 2 (8.0%) achieved PR. The median follow-up duration from CR was 36.9 months (range, 1.4-139.3 months). During follow-up, relapse occurred in three patients (12%), with a median time to relapse of 90 months (range 24-118 months) calculated from the date of CR. All relapsed cases were retreated with cladribine and achieved CR.

Table 1. Demographic and clinical characteristics of the patients (n=25)

Parameter	Value
Gender, n (%)	
Male	16 (64%)
Female	9 (36%)
Median age, years (range)	48 (27-68%)
Comorbidities, n (%)	11 (44%)
Type of HCL, n (%)	
<i>De novo</i>	24 (96%)
Therapy-related	1 (4%)
ECOG performance status, n (%)	
0	5 (20%)
1	18 (72%)
2	2 (8%)
Symptoms at diagnosis, n (%)	
Fatigue	21 (84%)
Weight loss	7 (28%)
Night sweats	2 (8%)
Fever	7 (28%)
Abdominal pain	12 (48%)
Abdominal fullness	14 (56%)
Infection	4 (16%)
Bone marrow fibrosis grade, n (%)	
Grade 1	10 (40%)
Grade 2	12 (48%)
Grade 3	3 (12%)
Bone marrow infiltration pattern	
Diffuse	17 (68%)
Interstitial	5 (20%)
Diffuse + interstitial	3 (12%)
Hairy cells in peripheral blood	22 (88%)
Peripheral LAP, n (%)	2 (8%)
Median hairy cells (%)	
In bone marrow	70% (5-99%)
In peripheral smear	30% (6-80%)
Splenic size by ultrasound (mm)	
Pre-treatment	170 (135-290)
Post-treatment	120 (106-160)
Laboratory findings at diagnosis	
Hemoglobin (g/dL)	9.6 (4.0-15.9)
WBC ($\times 10^3/\mu\text{L}$)	2.4 (0.7-18.1)
ANC ($\times 10^3/\mu\text{L}$)	0.51 (0.15-2.31)
Lymphocytes ($\times 10^3/\mu\text{L}$)	1.5 (0.1-13.6)
Monocytes ($\times 10^3/\mu\text{L}$)	0.09 (0-2.26)
Platelets ($\times 10^3/\mu\text{L}$)	58 (26-445)
Febrile neutropenia post-treatment	20 (80%)
HCL: Hairy cell leukemia, ECOG: Eastern Cooperative Oncology Group, LAP: Lymphadenopathy, WBC: White blood cell ANC: Absolute neutrophil count	

Median OS and PFS were not reached. At 12 years, the estimated OS and PFS rates were 100% and 80% (95% confidence interval: 53-95%), respectively. Details of treatment outcomes are provided in Table 2, and PFS is illustrated by the Kaplan-Meier curve in Figure 1.

Toxicity and Safety

The most frequent side effect observed was hematologic toxicity, followed by infections and gastrointestinal complaints. Anemia and thrombocytopenia improved after treatment, and leukocyte counts normalized within three months. Febrile neutropenia developed in 80% (n=20) of patients during the neutropenic phase, representing grade 3 hematologic toxicity according to Common Terminology Criteria for Adverse Events v5.0. Due to the persistent risk of infection and potential treatment-related mortality, all patients received prophylactic measures and infection management as recommended in current guidelines [20,21]. Mild gastrointestinal symptoms (grade 1-2 nausea and vomiting) were reported by seven patients (28%). Transient elevations in liver enzymes (aspartate aminotransferase, alanine aminotransferase) or bilirubin (grade 1-2) occurred in 4 patients (16%). No grade 4 hematologic or grade ≥ 3 non-hematologic toxicities were recorded.

Discussion

Classic HCL is a rare and indolent B-cell malignancy, marked by the slow buildup of malignant lymphoid cells in the bone marrow and spleen. This process often leads to cytopenias and a higher risk of infections [22]. With improved treatment options, most patients diagnosed with HCL now have a life expectancy comparable to that of healthy individuals [23].

In line with previous reports [2], our study demonstrated a higher frequency of HCL in males. Only one patient (4%) was asymptomatic at diagnosis, but treatment was initiated because of significant cytopenias.

Fatigue and abdominal complaints—often associated with splenic enlargement—are frequent in HCL, occurring in up to 90% of cases [24-26]. Our findings were consistent with this pattern: fatigue was the leading symptom (84%), followed by abdominal fullness (56%), and abdominal pain (48%). Splenomegaly was identified in all patients and remained the most common physical sign.

Table 2. Treatment outcomes of patients with hairy cell leukemia

	n (%)
CR	23 (92%)
PR	2 (8%)
Relapsed disease	3 (12%)
Follow-up, months, median (min-max)	36.9 months (1.4-139.3)
PFS	Not reached
OS	Not reached

CR: Complete response, PR: Partial remission, PFS: Progression-free survival, OS: Overall survival, min-max: Minimum-maximum

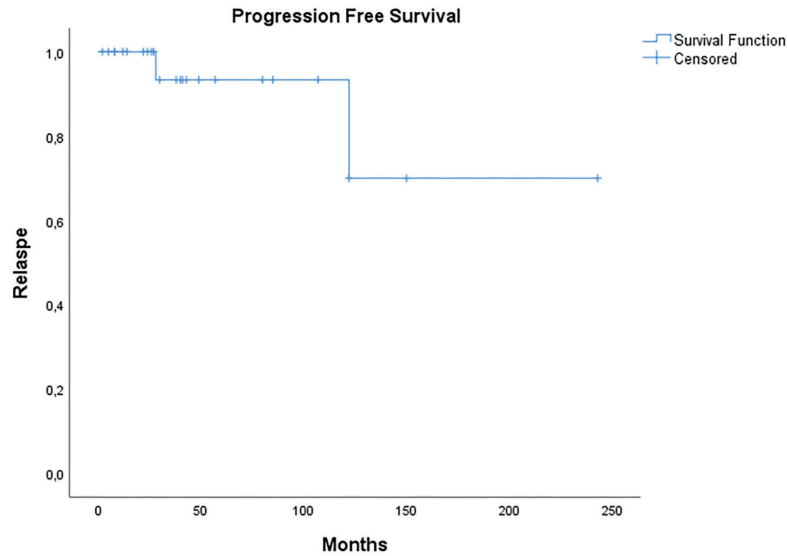


Figure 1. Progression-free survival (PFS)

Kaplan-Meier analysis showing PFS of the 25 patients. The median PFS was not reached. Censored observations are marked with a "+" symbol. At the 12-year follow-up, estimated PFS was 80% (95% confidence interval: 53-95%)

In our cohort, 72% of patients (n=18) had pancytopenia at diagnosis, reflecting baseline marrow suppression prior to therapy. Although no patient presented with febrile neutropenia at baseline, 80% developed it during cladribine treatment, which is consistent with the expected post-treatment cytopenic phase of purine analogs. This finding represents grade 3 hematologic toxicity, not an unexpected or life-threatening adverse event. Importantly, no grade 4 (life-threatening) hematologic toxicities were observed, and all episodes of febrile neutropenia resolved with appropriate antimicrobial and supportive management. Mild cytopenias and transient infections were observed; however, no grade ≥ 3 non-hematologic toxicities were observed in our cohort. Similar findings were reported in a Turkish series in which the rate of febrile neutropenia among patients treated with cladribine was 60% [27].

To minimize infection risk, prophylactic and supportive measures were systematically implemented. Patients with lymphopenia received prophylaxis with co-trimoxazole and aciclovir until lymphocyte recovery to $>1 \times 10^9/L$ [IV, B] [14]. Routine filgrastim use with cladribine is not recommended [IV, C] [20]; however, granulocyte colony-stimulating factor was administered selectively for severe infection-associated neutropenia. All patients were screened for hepatitis B, and those who tested positive received antiviral prophylaxis [14].

HCL typically causes suppression of at least two blood cell lineages, with leukopenia—particularly monocytopenia—being a hallmark feature. Monocyte depletion was observed in nearly all cases. Extranodal involvement, including lymphadenopathy and skeletal lesions, is uncommon at initial presentation but becomes more prominent in relapsed disease [28,29]. In our series, lymph node enlargement was detected in only two patients (8%).

Purine nucleoside analogs (PNAs), including cladribine and pentostatin, have achieved overall response rates exceeding 90%, with CR rates between 79% and 91%. No significant difference in efficacy has been noted between the two agents [3,9,30-32].

In our cohort, 23 patients (92%) achieved CR and 2 patients (8%) achieved PR following cladribine therapy. However, relapses can still occur despite the high efficacy of PNAs. A large cohort reported a relapse rate of only 3% after nearly 10 years of follow-up [30]. In another study, relapse rates for patients treated with pentostatin were 24% at 5 years and 42% at 10 years, whereas relapse rates for those receiving cladribine were 33% at 5 years and 48% at 10 years [33].

In our cohort, three patients (12%) experienced relapse after a median follow-up of 36.9 months. This relapse rate appears lower than those reported in large European series. In a study of 279 patients, Paillassa et al. [34] reported overall and CR rates of 97% and 78%, respectively, following first-line purine analog therapy. After a median follow-up of 10 years, the cumulative 10-year relapse incidence was 39%, and the median relapse-free survival was 11 years [34]. Despite the generally favorable long-term prognosis of HCL, relapses and secondary malignancies remain important concerns among long-term survivors.

The youngest individual in our cohort was 27 years old. Although prior studies have reported relapse rates as high as 58% among patients younger than 40 following cladribine therapy [35], both of our patients in this age group remained in long-term CR. Cladribine re-treatment has previously been associated with a 62% complete response rate and a 26% partial response rate [32]. In our series, all three patients who relapsed achieved CR again following a second course of cladribine.

Published data show OS rates of 96% at 4 years and 84% at 20 years for HCL patients [3,32]. By the fifth year after diagnosis, mortality risk becomes similar to that of peers in the general population [36]. In our cohort, no deaths were recorded over a median follow-up of 36.9 months.

Earlier reports indicate that 8-10 % of HCL survivors may develop secondary cancers over time [3,32]; nevertheless, no such events were detected in our series during follow-up.

In recent years, novel therapeutic strategies, including rituximab-based chemoimmunotherapy, targeted agents, such as BRAF and MEK inhibitors, and the BTK inhibitor ibrutinib, have demonstrated encouraging results in patients with relapsed or refractory HCL, providing new options beyond conventional purine-analog therapy [5].

The chief limitation of this work is the small number of patients, a consequence of the rarity of HCL. Moreover, assessment of the BRAF V600E mutation—found in nearly all classic HCL cases and viewed as a pivotal oncogenic driver [37]—was inconsistent because the study was retrospective and comprehensive molecular data were not always available.

In addition, molecular testing was limited to BRAF V600E and was performed in only four patients, while other potentially relevant mutations, such as MAP2K1 [38] and IGHV4-34 [24], were not assessed. These molecular aberrations are increasingly recognized as prognostic markers, and their absence restricts our ability to perform genotype-phenotype correlations and evaluate their impact on treatment response.

Unlike many other hematologic malignancies, HCL lacks a standardized risk stratification system. However, unfavorable outcomes have been linked to factors such as splenomegaly, elevated beta-2 microglobulin levels, leukocyte counts above $10 \times 10^9/L$, and levels of circulating hairy cells exceeding $5 \times 10^9/L$ [29]. In our analysis, minimal residual disease assessment and extended molecular profiling were not performed, further limiting the evaluation of long-term prognostic factors.

Conclusion

Cladribine continues to serve as a highly effective and well-tolerated first-line therapy for HCL, providing strong response rates and lasting remissions. Its manageable toxicity profile supports its continued use in clinical practice. In relapsed cases, cladribine re-administration remains a viable and effective strategy. However, prospective studies in larger populations are needed to optimize treatment approaches and enhance long-term outcomes in HCL.

Ethics

Ethics Committee Approval: The study was approved by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Dr. Abdurrahman Yurtarslan Ankara Oncology Health Application and Development Center of the (decision no: 2022-02/1651, date: 09.02.2022). All procedures were carried out in accordance with the ethical principles of the 1964 Helsinki Declaration.

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.S., B.A.C., D.İ., M.K.Ç., Concept: S.S., B.A.C., T.N.Y., Design: S.S., D.İ., M.K.Ç., Data Collection or Processing: S.S., B.A.C., S.Y., E.B., B.U.U., T.N.Y., Analysis or Interpretation: S.S., B.A.C., E.B., Literature Search: S.S., B.A.C., S.Y., E.B., B.U.U., T.N.Y., Writing: S.S., B.A.C.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

- Bouroncle BA, Wiseman BK, Doan CA. Leukemic reticuloendotheliosis. *Blood*. 1958;13:609-630.
- Tadmor T, Polliack A. Epidemiology and environmental risk in hairy cell leukemia. *Best Pract Res Clin Haematol*. 2015;28:175-179.
- Cornet E, Tomowiak C, Tanguy-Schmidt A, et al. Long-term follow-up and second malignancies in 487 patients with hairy cell leukaemia. *Br J Haematol*. 2014;166:390-400.
- Golomb HM. Hairy cell leukemia: lessons learned in twenty-five years. *J Clin Oncol*. 1983;1:652-656.
- Maitre E, Cornet E, Troussard X. Hairy cell leukemia: 2020 update on diagnosis, risk stratification, and treatment. *Am J Hematol*. 2019;94:1413-1422.
- Piro LD, Carrera CJ, Carson DA, Beutler E. Lasting remissions in hairy-cell leukemia induced by a single infusion of 2-chlorodeoxyadenosine. *N Engl J Med*. 1990;322:1117-1121.
- Kraut EH, Bouroncle BA, Grever MR. Pentostatin in the treatment of advanced hairy cell leukemia. *J Clin Oncol*. 1989;7:168-172.
- Spiers AS, Moore D, Cassileth PA, et al. Remissions in hairy-cell leukemia with pentostatin (2'-deoxycytosine). *N Engl J Med*. 1987;316:825-830.
- Else M, Dearden CE, Matutes E, et al. Long-term follow-up of 233 patients with hairy cell leukaemia, treated initially with pentostatin or cladribine, at a median of 16 years from diagnosis. *Br J Haematol*. 2009;145:733-740.
- Jehn U, Bartl R, Dietzfelbinger H, Haferlach T, Heinemann V. An update: 12-year follow-up of patients with hairy cell leukemia following treatment with 2-chlorodeoxyadenosine. *Leukemia*. 2004;18:1476-1481.
- Andrasiak I, Rybka J, Wrobel T. Response to the therapy in hairy cell leukemia: systematic review and meta-analysis. *Clin Lymphoma Myeloma Leuk*. 2018;18:392-399.e3.
- Goodman GR, Burian C, Koziol JA, Saven A. Extended follow-up of patients with hairy cell leukemia after treatment with cladribine. *J Clin Oncol*. 2003;21:891-896.
- Parry-Jones N, Joshi A, Forconi F, Dearden C; BSH guidelines committee. Guideline for diagnosis and management of hairy cell leukaemia (HCL) and hairy cell variant (HCL-V). *Br J Haematol*. 2020;191:730-737.
- Grever MR, Abdel-Wahab O, Andritsos LA, et al. Consensus guidelines for the diagnosis and management of patients with classic hairy cell leukemia. *Blood*. 2017;129:553-560.
- Grever MR, Blachly JS, Andritsos LA. Hairy cell leukemia: update on molecular profiling and therapeutic advances. *Blood Rev*. 2014;28:197-203.
- Venkataraman G, Aguhar C, Kreitman RJ, Yuan CM, Stetler-Stevenson M. Characteristic CD103 and CD123 expression pattern defines hairy cell leukemia: usefulness of CD123 and CD103 in the diagnosis of mature B-cell lymphoproliferative disorders. *Am J Clin Pathol*. 2011;136:625-630.
- Sherman MJ, Hanson CA, Hoyer JD. An assessment of the usefulness of immunohistochemical stains in the diagnosis of hairy cell leukemia. *Am J Clin Pathol*. 2011;136:390-399.

18. Falini B, Tiacci E, Liso A, et al. Simple diagnostic assay for hairy cell leukaemia by immunocytochemical detection of annexin A1 (ANXA1). *Lancet*. 2004;363:1869-1870. Erratum in: *Lancet*. 2004;363:2194.
19. Jones G, Parry-Jones N, Wilkins B, Else M, Catovsky D; British Committee for Standards in Haematology. Revised guidelines for the diagnosis and management of hairy cell leukaemia and hairy cell leukaemia variant*. *Br J Haematol*. 2012;156:186-195.
20. Robak T, Matutes E, Catovsky D, Zinzani PL, Buske C; ESMO Guidelines Committee. Hairy cell leukaemia: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. *Ann Oncol*. 2015;26:v100-v107.
21. Cornet E, Delmer A, Feugier P, et al. Recommendations of the SFH (French Society of Haematology) for the diagnosis, treatment and follow-up of hairy cell leukaemia. *Ann Hematol*. 2014;93:1977-1983.
22. Campo E, Swerdlow SH, Harris NL, Pileri S, Stein H, Jaffe ES. The 2008 WHO classification of lymphoid neoplasms and beyond: evolving concepts and practical applications. *Blood*. 2011;117:5019-5032.
23. Dinmohamed AG, Posthuma EFM, Visser O, Kater AP, Raymakers RAP, Doorduijn JK. Relative survival reaches a plateau in hairy cell leukemia: a population-based analysis in The Netherlands. *Blood*. 2018;131:1380-1383.
24. Troussard X, Maître E, Cornet E. Hairy cell leukemia 2022: update on diagnosis, risk-stratification, and treatment. *Am J Hematol*. 2022;97:226-236.
25. Quest GR, Johnston JB. Clinical features and diagnosis of hairy cell leukemia. *Best Pract Res Clin Haematol*. 2015;28:180-192.
26. Kreitman RJ, Arons E. Diagnosis and treatment of hairy cell leukemia as the COVID-19 pandemic continues. *Blood Rev*. 2022;51:100888.
27. Maral S, Albayrak M, Afacan Öztürk HB, et al. Cladribine Treatment in Hairy Cell Leukemia. *Medical Journal of Mugla Sitki Kocman University*. 2020;7:131-134.
28. De Propriis MS, Musiu P, Intoppa S, et al. Hairy cell leukaemia with low CD103 expression: a rare but important diagnostic pitfall. *Br J Haematol*. 2022;198:e28-e31.
29. Mendez-Hernandez A, Moturi K, Hanson V, Andritsos LA. Hairy cell leukemia: where are we in 2023? *Curr Oncol Rep*. 2023;25:833-840.
30. Chadha P, Rademaker AW, Mendiratta P, et al. Treatment of hairy cell leukemia with 2-chlorodeoxyadenosine (2-CdA): long-term follow-up of the Northwestern University experience. *Blood*. 2005;106:241-246.
31. Else M, Dearden CE, Matutes E, et al. Long-term follow-up of 233 patients with hairy cell leukaemia, treated initially with pentostatin or cladribine, at a median of 16 years from diagnosis. *Br J Haematol*. 2009;145:733-740.
32. Saven A, Burian C, Koziol JA, Piro LD. Long-term follow-up of patients with hairy cell leukemia after cladribine treatment. *Blood*. 1998;92:1918-1926.
33. Else M, Ruchlemer R, Osuji N, et al. Long remissions in hairy cell leukemia with purine analogs: a report of 219 patients with a median follow-up of 12.5 years. *Cancer*. 2005;104:2442-2248.
34. Paillasa J, Cornet E, Noel S, et al. Analysis of a cohort of 279 patients with hairy-cell leukemia (HCL): 10 years of follow-up. *Blood Cancer J*. 2020;10:62.
35. Rosenberg JD, Burian C, Waalen J, Saven A. Clinical characteristics and long-term outcome of young hairy cell leukemia patients treated with cladribine: a single-institution series. *Blood*. 2014;123:177-183.
36. Madanat YF, Rybicki L, Radivoyevitch T, et al. Long-term outcomes of hairy cell leukemia treated with purine analogs: a comparison with the general population. *Clin Lymphoma Myeloma Leuk*. 2017;17:857-862.
37. Tiacci E, Trifonov V, Schiavoni G, et al. BRAF mutations in hairy-cell leukemia. *N Engl J Med*. 2011;364:2305-2315.
38. Maitre E, Tomowiak C, Lebecque B, et al. Deciphering genetic alterations of hairy cell leukemia and hairy cell leukemia-like disorders in 98 patients. *Cancers (Basel)*. 2022;14:1904.