

Original Article

Retrospective Analysis of 17p Deletion Status and Treatment Modalities in Patients with Chronic Lymphocytic Leukemia

İ Mehmet Siddik Dilek¹, İ Abdullah Karakuş², İ Vehbi Demircan², İ Selahattin Tekeş³, İ Mehmet Orhan Ayyıldız²

¹Dicle University Faculty of Medicine, Department of Internal Medicine, Division of Medical Oncology, Diyarbakır, Türkiye

²Dicle University Faculty of Medicine, Department of Internal Medicine, Division of Hematology, Diyarbakır, Türkiye

³Dicle University Faculty of Medicine, Department of Medical Genetics, Diyarbakır, Türkiye

ABSTRACT

Aim: This study aimed to examine the demographic characteristics, disease stages, 17-deletion status, complications, treatment approaches, and survival data of 308 chronic lymphocytic leukemia (CLL) patients followed in the Department of Hematology at Dicle University.

Methods: A retrospective analysis was conducted on 308 patients diagnosed with CLL between January 2015 and December 2019. Data were compiled from patient records, including demographic information, complete blood counts, lactate dehydrogenase levels, lymphocyte counts, flow cytometry results, cytogenetic analysis (17p deletion), and imaging findings. Patients were classified according to the Rai and Binet staging systems, and their treatment protocols and disease complications were examined.

Results: In a retrospective analysis of 308 CLL patients, 61.7% were male, 38.3% were female, and the median age was 63 (28-86). Of the 170 patients studied for 17p deletion, 15.3% were found to be positive for 17p deletion. When patients were staged according to Rai, 8.4% were stage 0, 42.2% were stage I, 31.5% were stage II, 12.3% were stage III, and 5.5% were stage IV. According to the Binet staging, 29.5% of the patients were classified as stage A, 57.1% were classified as stage B, and 13.3% were classified as stage C. Secondary immunodeficiency was observed in 18.8% of the patients, autoimmune hemolytic anemia in 10.1%, and Richter transformation in 2.9%. One hundred ninety-nine patients (64.6%) received treatment, while 109 (35.4%) did not. During a median follow-up of 68.1 months, 38.3% of patients died. Patients with more advanced Rai and Binet stages had significantly shorter survival times. The median overall survival of patients with a positive 17p deletion was 83.4 months, while that of patients with a negative 17p deletion was 116 months ($p=0.015$).

Conclusion: The 17p deletion is associated with poor prognosis in CLL and is an important marker for guiding treatment and monitoring survival. The patient profile, stage, and treatment data from this regional study can contribute to the literature and guide the development of personalized approaches.

Keywords: Chronic lymphocytic leukemia, 17p deletion, leukemia, ibrutinib, venetoclax

Introduction

Chronic lymphocytic leukemia (CLL) is the most common leukemia in adults and is characterized by the clonal proliferation of mature B-lymphocytes. CLL is typically an indolent malignancy [1]. In Western countries, CLL constitutes a substantial proportion of adult leukemias; most patients are elderly, and the disease has a higher incidence in males. Genetic predisposition (such as 17p deletion and TP53 mutations) and environmental factors contribute to its etiology [1,2].

The clinical presentation of CLL is heterogeneous; most patients are asymptomatic and diagnosed incidentally via routine blood tests revealing lymphocytosis. Symptomatic cases may present with lymphadenopathy, splenomegaly, and infections [3]. Prognosis is guided by the Rai and Binet staging systems. Furthermore, genetic abnormalities including 17p deletion, 13q deletion, 11q deletion, and trisomy 12 hold prognostic and predictive significance [4-6]. The 17p deletion is a negative prognostic factor associated with shorter survival. Patients with 17p deletion have a worse response to treatment [7]. CLL

Address for Correspondence: Mehmet Siddik Dilek MD, Dicle University Faculty of Medicine, Department of Internal Medicine, Division of Medical Oncology, Diyarbakır, Türkiye

E-mail: dilekmehmet123@gmail.com **ORCID ID:** orcid.org/orcid.org/0000-0001-7730-6946

Received: 14.04.2026 **Accepted:** 20.07.2026 **Epub:** 29.07.2026 **Publication Date:** 21.08.2026

Cite this article as: Dilek MS, Karakuş A, Demircan V, Tekeş S, Ayyıldız MO. Retrospective analysis of 17p deletion status and treatment modalities in patients with chronic lymphocytic leukemia. Acta Haematol Oncol Turc. 2026;59(2):126-131



complications include Richter transformation, autoimmune hemolytic anemia (AIHA), and secondary immunodeficiency [8].

Treatment strategies vary based on disease stage and risk profile; a “watch and wait” approach is employed in early-stage disease, whereas advanced stages are managed with targeted therapies such as Bruton tyrosine kinase inhibitors (ibrutinib, acalabrutinib), B-cell lymphoma 2 inhibitors (venetoclax), and monoclonal antibodies (rituximab) or with chemotherapy-based regimens. In recent years, chemotherapy-free approaches have improved survival; however, outcomes remain poor in relapsed/refractory cases. Emerging therapies, including chimeric antigen receptor T-cell therapy, show promising potential [1,9].

In this single-center retrospective study, we analyzed the demographic characteristics, disease stages, 17p deletion status, complications, treatment modalities, and survival outcomes of 308 CLL patients to characterize the regional patient profile and contribute to the existing literature.

Methods

Patients

This study is a retrospective analysis of 308 patients diagnosed with CLL and/or followed up at the Department of Hematology, Dicle University, between January 2015 and December 2019. This study was conducted with the approval of the Non-Interventional Clinical Research Ethics Committee of Dicle University Faculty of Medicine (approval no: 338, date: 03.06.2021). Data were extracted from the hospital registry system and included the following: demographic characteristics (age, gender); complete blood count at initial presentation; peripheral blood smear examination; lactate dehydrogenase levels; absolute lymphocyte count; immunophenotypic evaluation by flow cytometry (including prognostic surface markers); and cytogenetic analysis results. Organomegaly and lymphadenopathy detected by imaging studies were also documented. Disease staging was performed according to the Rai and Binet systems. Treatment data, including chemotherapy regimens administered, were recorded. Among patients receiving multiple lines of therapy, the first-line treatment, the total number of treatment lines, and subsequent therapies were analyzed. Survival endpoints were defined as overall survival (OS), calculated from the date of diagnosis to the date of last follow-up or death, and treatment-free survival (TFS), calculated from the date of diagnosis to the initiation of first-line therapy.

Inclusion Criteria: Adult patients (≥ 18 years) diagnosed with CLL or small lymphocytic lymphoma between January 2015 and December 2019, with complete demographic, laboratory, and follow-up data available in the hospital registry system, were included.

Exclusion Criteria: Patients with a prior or subsequent diagnosis of another malignancy, incomplete staging or treatment information because of missing data, or a follow-up duration of less than 3 months were excluded from the study.

Statistical Analysis

Categorical variables were analyzed using the chi-square test. Continuous variables that did not follow a normal distribution were evaluated using the Mann-Whitney U and Kruskal-Wallis tests. Survival analyses were performed using the Kaplan-Meier method, and comparisons of survival curves were conducted with the log-rank test. A p value < 0.05 was considered statistically significant.

Results

A total of 308 patients were included in the study, of whom 190 (61.7%) were male and 118 (38.3%) were female. The median age was 63 years (range: 28-86 years). The 17p deletion status was evaluated in 170 patients; results were negative in 144 patients and positive in 26 patients (15.3%). The demographic and laboratory characteristics of the patients are presented in Table 1.

According to the Rai staging system, 26 (8.4%) patients were stage 0, 130 (42.2%) were stage I, 97 (31.5%) were stage II, 38 (12.3%) were stage III, and 17 (5.5%) were stage IV. Regarding Binet staging, 91 patients (29.5%) were stage A, 176 patients

Table 1. Demographic and laboratory characteristics

Characteristic	
Age	
Median (range)-years	63 (28-86)
Gender	
Male	190 (61.7%)
Female	118 (38.3%)
Leukocytes (/μL)	
Median (range)	28.7 (8.9-386)
Lymphocytes (/μL)	
Median (range)	21.2 (5-311)
Neutrophils (/μL)	
Median (range)	4.9 (0.6-16.9)
Hemoglobin (g/dL)	
Median (range)	13.1 (6.3-17.8)
Platelets (μL)	
Median (range)	195 (6.3-17.8)
Erythrocyte sedimentation rate (mm/h)	
Median (range)	11 (1-95)
Lactate dehydrogenase (IU/L)	
Median (range)	226 (95-1056)
Direct coombs	
Positive - n (%)	34 (11%)
Negative - n (%)	217 (70.5%)
Not tested - n (%)	57 (18.5)
Indirect coombs	
Positive - n (%)	8 (2.6%)
Negative - n (%)	217 (78.9%)
Not tested - n (%)	57 (18.5)
17p deletion - n (%)	
Tested	170
Positive	26 (15.3%)
Negative	144 (84.7%)

(57.1%) were stage B, and 41 patients (13.3%) were stage C. Evaluation of secondary complications revealed Richter transformation in 9 patients (2.9%), AIHA in 31 (10.1%), immune thrombocytopenic purpura in 6 (1.9%), and secondary immune deficiency in 58 (18.8%). AIHA was detected in 4 (4.4%) patients in Binet stage A, 22 (12.3%) in stage B, and 5 (13.2%) in stage C ($p=0.10$). Detailed information regarding patient staging and complications is provided in Table 2.

The median follow-up duration was 68.1 months (range: 3.1-186.6 months). Analysis of treatment status and disease stage at diagnosis showed that 109 patients (35.4%) remained untreated (observation), while 199 (64.6%) received treatment. As patients progressed through Binet stages, TFS time decreased significantly ($p<0.001$). Treatment modalities and distribution according to disease stage are summarized in Table 3.

For the 35 patients treated with ibrutinib, the median duration of therapy was 16 months (range: 2-41 months). When patients responses to ibrutinib treatment were examined, 7 showed a complete response, 22 showed a partial response, and 6 had refractory disease. Among the patients, 19 (54%) continued ibrutinib treatment; 10 (28.5%) died due to disease progression, and 6 (17.1%) were switched to venetoclax. Of the 7 patients treated with venetoclax, the median treatment duration was 7 months (range: 3-24 months); 2 (28.5%) continued treatment, while 5 (71.4%) died of disease progression during treatment. When responses to venetoclax treatment were examined, 4 patients showed a partial response and 3 were treatment-refractory. Data concerning patients receiving ibrutinib and venetoclax are shown in Table 4.

Over a median follow-up of 68.1 months (range: 3.4-186.6 months), 118 of the 308 patients (38.3%) died. Since less than 50% of the cohort reached the primary endpoint, the median OS for the total population was not reached. When analyzed by Rai stage, the median OS was 121.9 months [95% confidence interval (CI): 62.3-181.4] for stage 0, 128.7 months (95% CI: 115.5-141.8) for stage I, 105 months (95% CI: 83-127) for stage II, 83.6 months (95% CI: 46-121.1) for stage III, and 84.5 months (95% CI: 36.4-132.6) for stage IV ($p=0.001$). The OS curves by

Table 2. Staging and complications of patients

Characteristic	
Rai staging	
Stage 0 - n (%)	26 (8.4%)
Stage I - n (%)	130 (42.2%)
Stage II - n (%)	97 (31.5)
Stage III - n (%)	38 (12.3)
Stage IV - n (%)	17 (5.5)
Binet staging	
Stage A - n (%)	91 (29.5%)
Stage B - n (%)	176 (57.1%)
Stage C - n (%)	41 (13.3%)
Richter transformation - n (%)	9 (2.9%)
Autoimmune hemolytic anemia - n (%)	31 (10.1%)
Immune thrombocytopenic purpura - n (%)	6 (1.9%)
Secondary immunodeficiency - n (%)	58 (18.8%)

Table 3. Treatment patterns and outcomes

Characteristic	
Binet stage - treated/total patients (%)	
A	34/91 (37.4%)
B	133/179 (74.3%)
C	32/38 (84.2%)
Total	199/308 (64.6%)
Rai stage - treated/total patients (%)	
Stage 0	7/26 (26.9%)
Stage I	66/130 (50.8%)
Stage II	81/97 (83.5%)
Stage III	29/38 (76.3%)
Stage IV	16/17 (94.1%)
Total	199/308 (64.6%)
Number of treatment lines - n (%)	
Untreated	109 (35.4%)
1 line	94 (30.5%)
2 lines	64 (20.8%)
3 lines	22 (7.1%)
4 lines	17 (5.5%)
5 lines	2 (0.6%)
Treatment-free survival duration by Binet stage - median (95% confidence interval) - months	
Stage A	34.1 (22.6-45.6)
Stage B	22.1 (16.5-27.6)
Stage C	2.3 (0-5.5)
Treatment-free survival duration by Rai stage - median (95% confidence interval) - months	
Stage 0	29.4 (18.6-40.2)
Stage I	23.5 (13.3-30.8)
Stage II	27.2 (16.2-38.3)
Stage III	4.1 (0-14.6)
Stage IV	0.8 (0-2)
Treatments received by patients - n (%)	
Untreated	109 (35.4%)
Chlorambucil	95 (30.8%)
R-chlorambucil	6 (1.9%)
FC/R-FC	87 (28.2%)
R-bendamustine	87 (28.2%)
CVP/R-CVP	32 (10.4%)
CHOP/R-CHOP	16 (5.2%)
Ibrutinib	35 (11.4%)
Venetoclax	7 (2.3%)
First-line treatments - n (%)	
Untreated	109 (35.4%)
Chlorambucil	82 (26.6%)
Rituximab-chlorambucil	4 (1.3%)
FC/R-FC	54 (17.5%)
R-bendamustine	36 (11.7%)
CVP/R-CVP	18 (5.8%)
CHOP/R-CHOP	5 (1.6%)

R: Rituximab, FC: Fludarabine + cyclophosphamide, CVP: Cyclophosphamide + vincristine + prednisone, CHOP: Cyclophosphamide + doxorubicin + vincristine + prednisone

Table 4. Patients treated with ibrutinib and venetoclax	
Characteristic	
Patients treated with ibrutinib	
Treatment duration - median (range), months	16 (2-41)
Ongoing treatment - n (%)	19 (54)
Deceased - n (%)	10 (28.5%)
Switched to venetoclax - n (%)	6 (17.1%)
Total	35 (100%)
Patients treated with venetoclax	
Treatment duration - median (range), months	7 (3-24%)
Ongoing treatment - n (%)	2 (28.5%)
Deceased - n (%)	5 (71.4%)
Total	7 (100%)

Rai stage are shown in Figure 1. Regarding Binet staging, the median OS was 145.1 months (95% CI: 114.5-175.7) for stage A; 121.1 months (95% CI: 97.4-126.8) for stage B; and 83.6 months (95% CI: 104.5-129.9) for stage C (p=0.006). Figure 2 also shows the OS curve stratified by Binet stage. Patients with the 17p deletion had a median OS of 83.4 months (95% CI: 60.4-127), whereas patients without the 17p deletion had a median OS of 116 months (95% CI: 89.9-135.9) (p=0.015). The OS curve stratified by 17p deletion status is presented in Figure 3. Comprehensive OS data are summarized in Table 5.

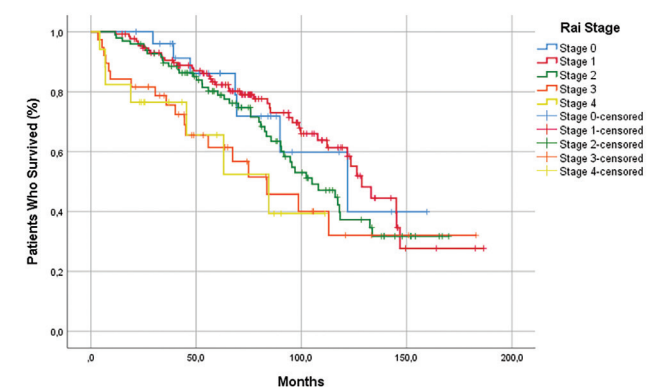


Figure 1. Rai Stage and overall survival

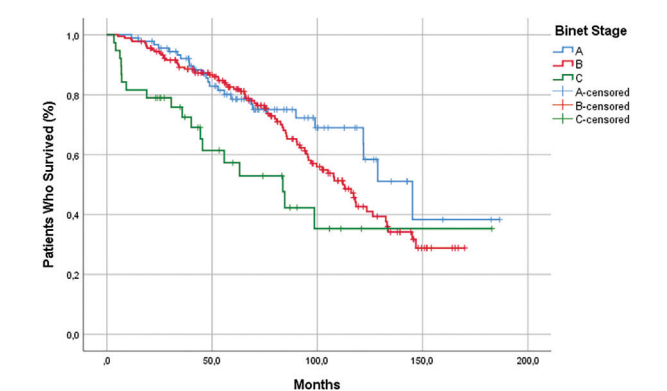


Figure 2. Binet staging and overall survival

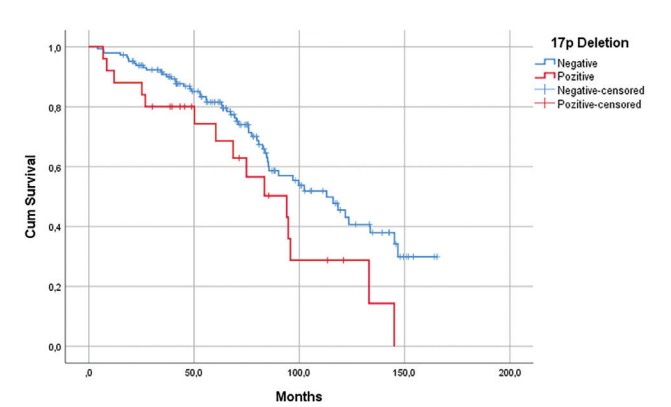


Figure 3. 17p deletion and overall survival

Table 5. Overall survival data	
Characteristic	p
Rai stage - median overall survival, months (95% confidence interval)	
Stage 0	121.9 (62.3-181.4)
Stage I	128.7 (115.5-141.8)
Stage II	105 (83-127)
Stage III	83.6 (46-121.1)
Stage IV	84.5 (36.4-132.6)
Binet stage - median overall survival, months (95% confidence interval)	
Stage A	145.1 (114.5-175.7)
Stage B	121.1 (97.4-126.8)
Stage C	83.6 (104.5-129.9)
17p deletion - median overall survival, months (95% confidence interval)	
Positive	83.4 (60.4-127)
Negative	116 (89.9-135.9)

Discussion

In our study, the male-to-female ratio was 1.6:1. According to 2025 United States statistics [10], this ratio is 1.53:1, while Aslan [11] reported 1.6 in 2013. The median age at diagnosis in our cohort was 63 years, compared with 66 years in the study by Küçük [12] in Aydın, 62 years in the study by Aslan [11] in Ankara, and 71.6 years in the study by Smith et al. [13] in the United Kingdom. The younger median age of CLL patients in Türkiye, compared with Western countries, may be attributed to geographic differences, a higher proportion of younger individuals in Türkiye’s population, and longer life expectancy in Western societies.

Analysis of Rai staging showed that 8.4% of patients were stage 0, 42.2% were stage I, 31.5% were stage II, 12.3% were stage III, and 5.5% were stage IV. In the original 1975 study by Rai et al. [4], the distribution was 17.6% stage 0, 23.2% stage I, 31.2% stage II, 16.8% stage III, and 11.2% stage IV. Aslan [11] reported in 2013 that 18% were stage 0, 21.6% stage I, 27.7% stage II, 12.7% stage III, and 9% stage IV. The lower proportion of stage 0 patients in our cohort may be explained by our center being

the sole hematology referral facility for several provinces for many years, resulting in patients presenting at symptomatic, treatment-requiring stages.

The incidence of AIHA in our study was 10.1%, compared with 11% in the study by Diehl and Ketchum [14] and 4.3% in the study by Mauro et al. [15]. Reported rates of AIHA in the literature range from 4% to 10% [8]. Variations in patient stage, follow-up duration, and demographic characteristics across studies likely account for this heterogeneity. In our study, Richter transformation occurred at a frequency of 2.9%, which is consistent with the 2-9% range reported in the literature [4]. Immune thrombocytopenia was observed in 1.9% of cases, compared with 2% in Diehl and Ketchum [14] and 1.1% in Barcellini et al. [16]. Overall, the frequencies of AIHA and immune thrombocytopenia in our study align closely with published data.

The 17p deletion was detected in 15.3% of tested patients. Hallek et al. [17] reported a rate of 10%, whereas Stilgenbauer et al. [18] found 30%. Sellner et al. [19] noted 3-8% in treatment-naïve patients and up to 30% in treatment-refractory cases. Differences in 17p deletion prevalence across studies may reflect variations in disease stage and prior treatment exposure.

TFS shortened significantly with advancing disease stage, consistent with findings from Rai et al. [4] and Shanafelt et al. [20]. Our results reinforce this established pattern.

OS also decreased significantly with higher Rai stage, mirroring the original observations of Rai et al. [4] in 1975. Shanafelt et al. [20] in 2010 similarly reported stage-dependent reductions in OS, but noted longer survival across all stages compared to the 1975 cohort. Likewise, OS in our study was prolonged compared with historical data, independent of stage. This improvement, seen in contemporary studies including ours and Shanafelt et al. [20], may be attributable to the advent of novel therapies and enhanced access to healthcare.

Patients with 17p deletion exhibited shorter OS, in agreement with the results of Tausch et al. [6] and Stilgenbauer et al. [7]. Thus, our findings are consistent with the literature and validate 17p deletion as a poor prognostic factor for OS. Due to national reimbursement policies during the study period, our patients could not access novel targeted agents such as ibrutinib and venetoclax as first-line therapies; instead, they received these agents in later lines of therapy after chemotherapy failure. The inability to initiate these highly effective targeted therapies in the frontline setting is considered a key factor that prevents their overall efficacy from reaching the high levels reported in the literature.

OS also decreased significantly with higher Rai stage, mirroring the original observations of Rai et al. [4] in 1975. Shanafelt et al. [20] in 2010 similarly reported stage-dependent reductions in OS, but noted longer survival across all stages compared to the 1975 cohort. Likewise, OS in our study was prolonged compared to historical data, independent of stage. This improvement, seen in contemporary studies, including ours and that of Shanafelt et al. [20], may be attributable

to the advent of novel therapies and enhanced access to healthcare.

Patients with 17p deletion exhibited shorter OS, in agreement with Tausch et al. [6] and Stilgenbauer et al. [7]. Thus, our findings confirm the literature and validate 17p deletion as a poor prognostic factor for OS.

Study Limitations

Several limitations of our study should be acknowledged. First, because our study was single-center and retrospective, our findings may primarily reflect the characteristics of a specific geographic region rather than those of a multi-center cohort. Second, the 17p deletion status was not evaluated in all patients; instead, our analysis was based on results available for 55.2% of the study population. Additionally, the median follow-up duration for patients receiving novel therapies, such as ibrutinib and venetoclax, was relatively short, thereby limiting the assessment of long-term treatment response and therapy-related complications. Finally, the absence of data regarding other genetic markers, including immunoglobulin heavy chain variable region mutation status, 11q deletion, and trisomy 12, precluded the establishment of a comprehensive genetic profile for all patients.

Conclusion

In this retrospective analysis of 308 patients with CLL, a significant association was identified between 17p deletion and poor prognosis. The presence of a 17p deletion at any point should prompt consideration of an unfavorable prognosis. As a critical prognostic marker in treatment and survival monitoring, 17p deletion must be carefully considered in the long-term management of CLL. Furthermore, evaluating survival outcomes and treatment efficacy according to disease stage may contribute to the optimization of future therapeutic strategies. These findings will guide the understanding of regional patient characteristics and the development of personalized treatment approaches.

Ethics

Ethics Committee Approval: This study was conducted with the approval of the Non-Interventional Clinical Research Ethics Committee of Dicle University Faculty of Medicine (approval no: 338, date: 03.06.2021).

Informed Consent: The data collection program for this study was approved by ethics committee with a waiver for individual informed consent for this retrospective study.

Footnotes

Authorship Contributions

Concept: M.D.F., A.K., V.D., S.T., M.O.A., Design: M.D.F., A.K., V.D., S.T., M.O.A., Data Collection or Processing: M.D.F., A.K., V.D., S.T., Analysis or Interpretation: M.D.F., A.K., V.D., S.T., M.O.A., Literature Search: M.D.F., A.K., V.D., S.T., M.O.A., Writing: M.D.F., A.K., S.T., M.O.A.

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

1. Hallek M. Chronic lymphocytic leukemia: 2025 update on the epidemiology, pathogenesis, diagnosis, and therapy. *Am J Hematol*. 2025;100:450-480.
2. NIH. Chronic lymphocytic leukemia - cancer stat facts. SEER. 2016-2022. Available from: <https://seer.cancer.gov/statfacts/html/clyl.html>
3. Longo DL, Fauci AS, Kasper DL, et al., editors. *Harrison's principles of internal medicine*. 22nd edition. New York: McGraw Hill; 2025. Available from: <https://accessmedicine.mhmedical.com/book.aspx?bookid=3541>
4. Rai KR, Sawitsky A, Cronkite EP, Chanana AD, Levy RN, Pasternack BS. Clinical staging of chronic lymphocytic leukemia. *Blood*. 1975;46:219-234.
5. Alrawashdh N, Sweasy J, Erstad B, McBride A, Persky DO, Abraham I. Survival trends in chronic lymphocytic leukemia across treatment eras: US SEER database analysis (1985-2017). *Ann Hematol*. 2021;100:2501-2512.
6. Tausch E, Schneider C, Robrecht S, et al. Prognostic and predictive impact of genetic markers in patients with CLL treated with obinutuzumab and venetoclax. *Blood*. 2020;135:2402-2412.
7. Stilgenbauer S, Schnaiter A, Paschka P, et al. Gene mutations and treatment outcome in chronic lymphocytic leukemia: results from the CLL8 trial. *Blood*. 2014;123:3247-3254.
8. Stilgenbauer S. Overview of the complications of chronic lymphocytic leukemia. UpToDate. 2026. Available from: https://www.uptodate.com/contents/overview-of-the-complications-of-chronic-lymphocytic-leukemia?search=cll&topicRef=4545&source=see_link#H13
9. NIH. Chronic lymphocytic leukemia treatment (PDQ®)-health professional version. 2025. Available from: <https://www.cancer.gov/types/leukemia/hp/ll-treatment-pdq>
10. Siegel RL, Kratzer TB, Giaquinto AN, Sung H, Jemal A. Cancer statistics, 2025. *CA Cancer J Clin*. 2025;75:10-45.
11. Aslan NA. Retrospective evaluation of prognostic features and treatment response in chronic lymphocytic leukemia. Hacettepe University; 2013.
12. Küçük S. Prognosis evaluation in chronic lymphocytic leukemia. Aydın Adnan Menderes University; 2024.
13. Smith A, Howell D, Patmore R, Jack A, Roman E. Incidence of haematological malignancy by sub-type: a report from the Haematological Malignancy Research Network. *Br J Cancer*. 2011;105:1684-1692.
14. Diehl LF, Ketchum LH. Autoimmune disease and chronic lymphocytic leukemia: autoimmune hemolytic anemia, pure red cell aplasia, and autoimmune thrombocytopenia. *Semin Oncol*. 1998;25:80-97.
15. Mauro FR, Foa R, Cerretti R, et al. Autoimmune hemolytic anemia in chronic lymphocytic leukemia: clinical, therapeutic, and prognostic features. *Blood*. 2000;95:2786-2792.
16. Barcellini W, Capalbo S, Agostinelli RM, et al; GIMEMA Chronic Lymphocytic Leukemia Group. Relationship between autoimmune phenomena and disease stage and therapy in B-cell chronic lymphocytic leukemia. *Haematologica*. 2006;91:1689-1692.
17. Hallek M, Fischer K, Fingerle-Rowson G, et al; German Chronic Lymphocytic Leukaemia Study Group. Addition of rituximab to fludarabine and cyclophosphamide in patients with chronic lymphocytic leukaemia: a randomised, open-label, phase 3 trial. *Lancet*. 2010;376:1164-1174.
18. Stilgenbauer S, Zenz T, Winkler D, et al; German Chronic Lymphocytic Leukemia Study Group. Subcutaneous alemtuzumab in fludarabine-refractory chronic lymphocytic leukemia: clinical results and prognostic marker analyses from the CLL2H study of the German Chronic Lymphocytic Leukemia Study Group. *J Clin Oncol*. 2009;27:3994-4001.
19. Sellner L, Denzinger S, Dietrich S, et al. What do we do with chronic lymphocytic leukemia with 17p deletion? *Curr Hematol Malig Rep*. 2013;8:81-90.
20. Shanafelt TD, Rabe KG, Kay NE, et al. Age at diagnosis and the utility of prognostic testing in patients with chronic lymphocytic leukemia. *Cancer*. 2010;116:4777-4787.