

International Journal of Health Sciences
Available online at www.sciencescholar.us
Vol. 10 No. 3, December 2026, pages: 264-275
E-ISSN: 2550-696X
https://doi.org/10.53730/ijhs.v10n3.16037

The Role of Complex Karyotype in the Diagnosis of Chronic Lymphocytic Leukemia: Integration of Conventional Cytogenetics and FISH Analysis

Irene Caliendo [a], Francesco Puopolo [b], Carmela Fabbricatore [c], Valeria Belsito Petrizzi [d], Giuseppe Malferà [e] Maria Langella [f] Catello Califano [g]

Manuscript submitted: 27 September 2026, Manuscript revised: 18 October 2026, Accepted for publication: 03 November 2026

Corresponding Author [a]



Keywords

chronic lymphocytic leukemia (CLL);
complex karyotype (CK);
conventional cytogenetic analysis (CCA);
FISH;
high- CK cytogenetic complexity;

Abstract

Complex karyotype (CK) is an emerging high-risk feature in chronic lymphocytic leukemia (CLL). We evaluated its diagnostic and prognostic relevance by integrating conventional cytogenetic analysis (CCA) and fluorescence in situ hybridization (FISH) in 30 patients. CCA identified CK in 23% of cases, including high-complexity profiles (≥5 abnormalities). FISH detected recurrent genomic lesions with established prognostic value: del(13q), trisomy 12, del(11q), del(17p). Integrated analysis improved detection of genomic complexity and refined risk stratification beyond single-lesion assessment. A representative case highlighted the ability of combined approaches to identify high-risk CK with TP53 disruption. These findings support routine integration of CCA and FISH for comprehensive cytogenetic profiling in CLL.

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[a] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[b] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[c] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[d] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[e] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[f] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy
[g] Hematology Unit, Hospital "Andrea Tortora", Pagani, ASL Salerno, Italy

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1 Introduction

Chronic lymphocytic leukemia (CLL) is a clonal lymphoproliferative disorder characterized by the progressive accumulation of mature CD5-positive B lymphocytes in the peripheral blood, bone marrow, and lymphoid tissues. It represents the most common leukemia in Western countries, with a higher incidence in males and a markedly lower prevalence in Eastern populations (Radaelli et al., 2004). The median age at diagnosis is approximately 70 years; more than 40% of cases occur in patients older than 75 years, whereas fewer than 10% are diagnosed before the age of 50 (Brenner et al., 2008). CLL is characterized by marked biological and clinical heterogeneity, ranging from indolent disease requiring no immediate therapy to highly aggressive forms associated with rapid progression and poor survival outcomes. Diagnosis is established by the presence of ≥5 × 10^9/L monoclonal B lymphocytes in the peripheral blood persisting for at least three months, together with a characteristic immunophenotypic profile (CD5+, CD19+, CD23+) confirmed by flow cytometry. Morphologically, leukemic cells are typically small mature lymphocytes. Patterns of bone marrow infiltration may vary and have prognostic relevance (Kipps et al., 2017). The pathogenesis of CLL is associated with recurrent genetic and molecular alterations involving pathways regulating apoptosis, cell-cycle progression, and DNA repair. The most frequent cytogenetic abnormalities include del(13q), del(11q), del(17p), and trisomy 12, each associated with distinct clinical and prognostic implications (Foà et al., 2013; Pekarsky et al., 2015; Rose-Zerilli et al., 2014; Woyach et al., 2014). TP53 disruption, resulting from del(17p) and/or TP53 mutation, is linked with resistance to chemoimmunotherapy and adverse clinical outcomes (Kipps et al., 2017). Beyond recurrent single lesions, increasing attention has focused on complex karyotype (CK), generally defined by the presence of three or more chromosomal abnormalities within the same clone. CK has emerged as a major marker of genomic instability and is associated with disease progression, treatment resistance, and inferior survival outcomes (Visentin et al., 2021). Importantly, CK is biologically heterogeneous and may be further stratified according to the number and type of chromosomal abnormalities. Patients harboring three abnormalities (low-CK) generally show a more favorable clinical course compared with those carrying ≥5 abnormalities (high-CK), which is associated with poor response to therapy (Serafin et al., 2025). Moreover, the presence of more structural abnormalities, including derivative chromosomes or unbalanced translocations (referred to in some studies as CK-2) (Baliakas et al., 2019; Strickland et al., 2017)), identifies a subgroup with particularly aggressive disease and poor prognosis (Rigolin et al., 2018; Visentin et al., 2019). Overall, the biological heterogeneity of CLL underlines the importance of integrating cytogenetic and molecular profiling into routine clinical practice to improve risk stratification and guide therapeutic decision-making.

2 Materials and Methods

Biological samples for CCA included bone marrow (BM), peripheral blood (PB), and, less frequently, solid tissue specimens. Samples were collected under sterile conditions and anticoagulated with sodium or lithium heparin. Bone marrow is most commonly used for cytogenetic investigations in hematologic malignancies. Conventional banding analysis (CBA) in CLL is limited by its labor-intensive workflow and by the requirement for stimulated cell cultures, due to the intrinsically low proliferative activity of neoplastic B cells in vitro

(Haferlach et al., 2007; Baliakas et al., 2022). Current CLL guidelines recommend parallel stimulation with tetradecanoyl phorbol acetate (TPA), IL-2, and DSP30 for optimal detection of CK (Baliakas et al., 2022). In a comparative study conducted on 255 patients with CLL, Kamaso et al. demonstrated that IL-2 - DSP30 mix identified a significantly higher proportion of abnormal karyotypes compared with TPA stimulation alone (50% vs. 39%, p = 0.031) (Kamaso et al., 2024). Moreover, among cases classified as abnormal by both approaches, IL-2 + DSP30 detected a higher number of chromosomal abnormalities and/or abnormal metaphases in 71% of cases. Although CK detection rates were overall comparable between TPA and IL-2 + DSP30 (10% vs. 11%), approximately one-third of CK cases were discordant, mainly due to normal karyotypes or lack of metaphases in TPA-stimulated cultures (Kamaso et al., 2024). Bone marrow aspirates were obtained from the posterior iliac crest under local anesthesia because of the lower associated risk of complications. Peripheral blood samples were also used as an alternative source of leukemic cells according to clinical indication and sample availability. Cell cultures were established using 20×10^6 cells suspended in 10 mL RPMI 1640 medium supplemented with 30% fetal bovine serum, 200 mM L-glutamine, and penicillin–streptomycin (50 µg/mL). For each sample, two parallel cultures were prepared and incubated at 37°C in a humidified atmosphere containing 5% CO_2 for 24 and 72 hours, respectively. The 72-hour cultures were stimulated with DSP30 (2 µM), a CpG oligonucleotide, in combination with interleukin-2 (IL-2; 200 U/mL) to enhance B-cell proliferation. This protocol is particularly relevant in CLL for the low mitotic activity of mature neoplastic B lymphocytes. The combined use of DSP30 and IL-2 promotes proliferation, reduces apoptosis, and improves the detection of chromosomal abnormalities, including CK (Haferlach et al., 2007). Following incubation, colchicine (80 µL) was added to the cultures for 50 minutes at 37°C to arrest cells in metaphase. Cells were subsequently centrifuged at 2500 rpm for 7 minutes and treated with hypotonic solution (0.075 M KCl) at 37°C for 20 minutes. Fixation was performed using Carnoy's fixative (methanol: acetic acid, 3:1), followed by repeated fixation steps. Samples were stored at −20°C for at least 30 minutes before slide preparation. Cell suspensions were dropped onto glass slides, aged at 100°C for 15 minutes, and left overnight. G-banding was performed using the GAW technique (G-banding with acetic saline and Wright stain). Slides were incubated in 2×SSC (saline sodium citrate) solution at 37°C for 20 minutes and subsequently stained with Wright solution for 3 minutes. Metaphases were analyzed by light microscopy. At least 20 metaphases were evaluated at diagnosis and 30 during treatment monitoring. Image acquisition and karyotype analysis were performed using a computerized imaging system, and chromosomal abnormalities were classified according to the International System for Human Cytogenetic Nomenclature (ISCN 2024). Because CCA requires an adequate number of evaluable metaphases and CLL cells exhibit low spontaneous mitotic activity, FISH was employed as a complementary technique. FISH uses fluorescently labeled DNA probes targeting specific chromosomal regions, enabling the identification of recurrent genetic abnormalities through fluorescence signal detection (Ripollés et al., 2006). iFISH allows the evaluation of non-dividing cells and the detection of cryptic abnormalities not identifiable by conventional cytogenetics. Different probe categories were used, including centromeric probes (CEP), directed against α-satellite centromeric DNA sequences for the detection of numerical abnormalities, and locus-specific identifier (LSI) probes targeting defined chromosomal regions. FISH represents a rapid and reliable approach for screening recurrent genetic alterations (Wan et al., 2014; Wan et al., 2017). In the present study, four probes commonly used in CLL were employed to identify the most clinically relevant chromosomal aberrations (Figure 1):

- Vysis LSI ATM probe for the 11q22.3 region;
- Vysis CEP 12 Spectrum Orange DNA Probe Kit;
- Vysis LSI D13S319/LSI 13q34 FISH Probe Kit;
- Vysis LSI TP53 17p13.1 Spectrum orange Probe and Vysis CEP 17 17p11.1 - q11.1 Spectrum Green;

These abnormalities were used to stratify patients into favorable-, intermediate-, and unfavorable-risk prognostic groups and to provide clinically relevant information for therapeutic decision-making. For each analysis, 200 interphase nuclei were evaluated per sample and probe.

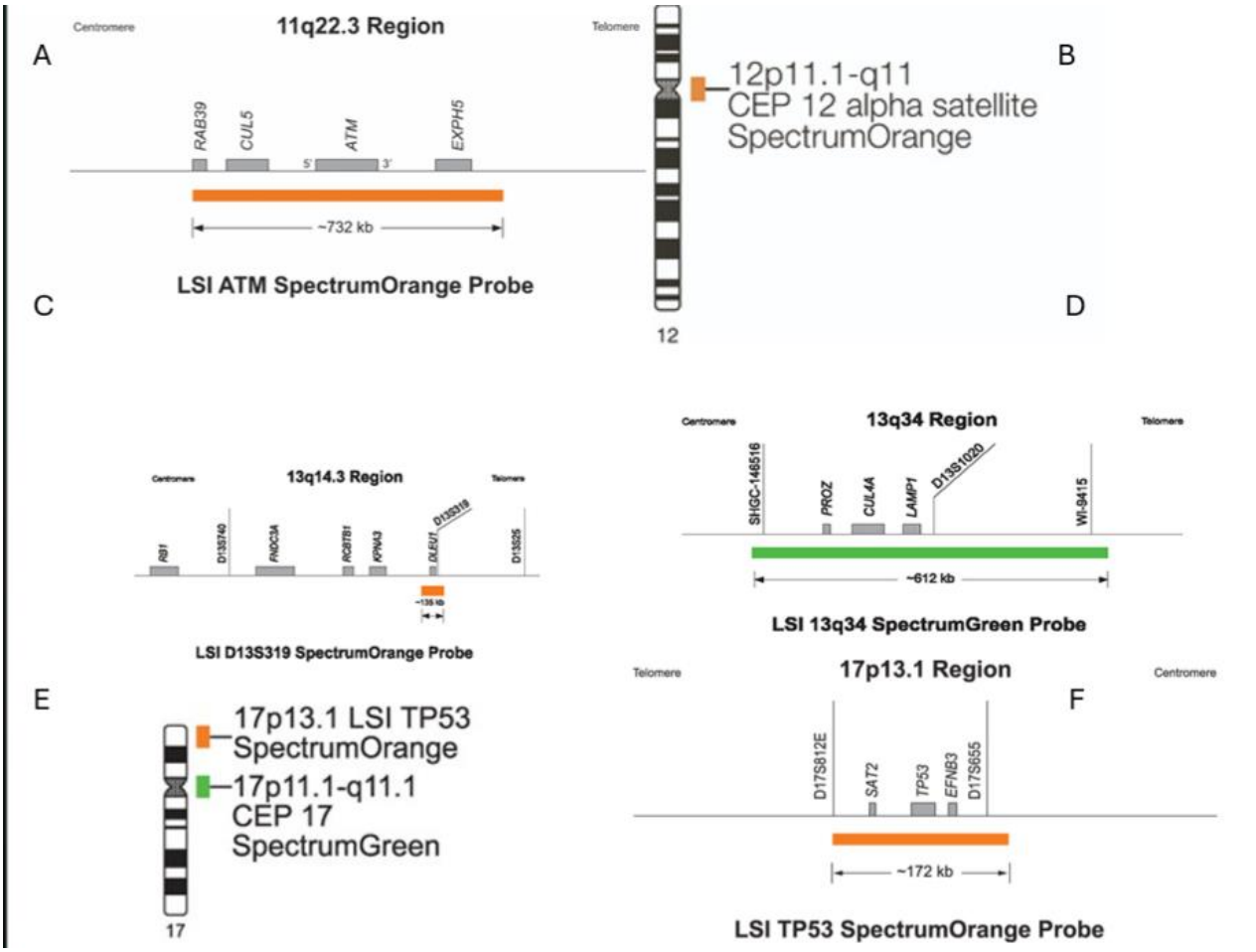


Figure 1. iFISH probe panel: [A] Vysis LSI ATM probe for the 11q22.3 region, which contains several genes, including ATM; [B] Vysis CEP 12 Spectrum Orange DNA Probe Kit, consisting of a Spectrum Orange-labeled probe specific for the α-satellite centromeric region of chromosome 12 (12p11.1–q11); [C] Vysis LSI D13S319/LSI 13q34 FISH Probe Kit: region 13q14.3 containing the D13S319 marker involved in the deletion; [D] Vysis LSI D13S319/LSI 13q34 FISH Probe Kit: region 13q14.3 containing the D13S319 marker involved in the deletion; [E] Vysis LSI TP53 17p13.1 Spectrum orange Probe and Vysis CEP 17 17p11.1 - q11.1 Spectrum Green; [F] Vysis LSI TP53 Spectrum Orange Probe (www.molecular.abbott).

FISH analysis was performed on metaphase and interphase preparations obtained from cytogenetic cultures and stored at −20°C. Slides were pretreated in 2×SSC buffer (pH 7.0) at 37°C for 30 minutes and dehydrated through a graded ethanol series (70%, 90%, and 100%). The hybridization mixture consisted of 7 µL hybridization buffer, 2 µL distilled water, and 1 µL probe per slide. Slides were processed using a Thermobrite humidified system for DNA denaturation and overnight hybridization at 37°C. For LSI probes, denaturation was performed at 74°C for 3 minutes, while CEP12 probes were denatured at 73°C for 2 minutes. Post-hybridization washes were carried out in 0.4×SSC/0.3% NP-40 at 72°C for 2 minutes, followed by 2×SSC/0.1% NP-40 at room temperature for 30 seconds. Slides were counterstained with 4′,6-diamidino-2-phenylindole (DAPI), and fluorescence signals were analyzed using a fluorescence microscope equipped with FITC, TRITC, and DAPI filters (Olympus). At diagnosis, at least 200 interphase nuclei were evaluated per sample, whereas during treatment, at least 400 nuclei were analyzed per slide. All analyses were independently reviewed by at least two operators to ensure reproducibility and reliability of results (Wan et al., 2017).

Study Cohort and Setting

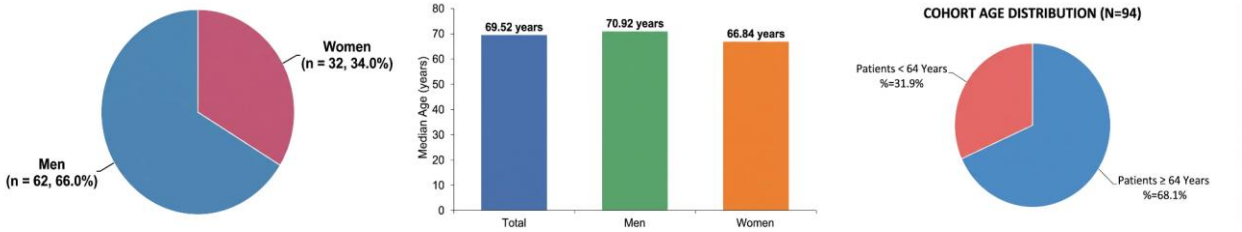


Figure 2. Principal CLL patients' characteristics

The study included bone marrow aspirate and peripheral blood samples obtained from 94 patients diagnosed with chronic lymphocytic leukemia (CLL). All samples were processed at the Oncohematological Cytogenetics Laboratory of the Hematology Unit, A. Tortora Hospital. The laboratory participates in the external quality assessment (CEQ) program coordinated by the Istituto Superiore di Sanità for oncohematological cytogenetics, ensuring standardized quality control in chromosome analysis and reporting. All specimens were processed according to standardized protocols for cytogenetic preparation and FISH analysis. Demographic and clinical characteristics of the study cohort are summarized in Figure 2. Patients were stratified according to age and sex. The cohort reflected the typical epidemiologic features of CLL in Western countries, including male predominance, advanced median age at diagnosis, and a high proportion of patients older than 64 years (Khoral et al., 2017).

3 Results and Discussions

Figure 3 illustrates the cytogenetic profile of the 94 patients evaluated between 2022 and 2026, showing the proportions of cases with a normal karyotype, a complex karyotype, and a karyotype with < 3 chromosomal abnormalities.

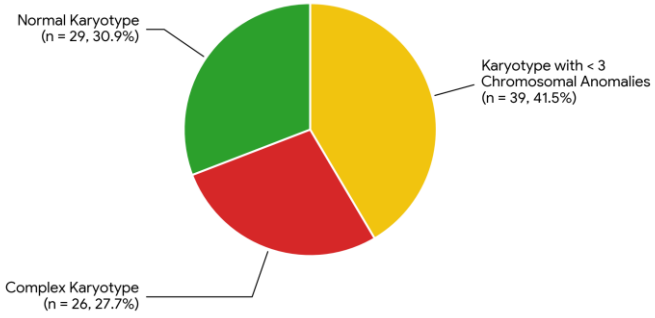


Figure 3. Proportions of cytogenetic findings in 94 patients studied from 2022 to 2026. Distribution of normal karyotype, complex karyotype, and karyotype harboring < 3 chromosomal abnormalities

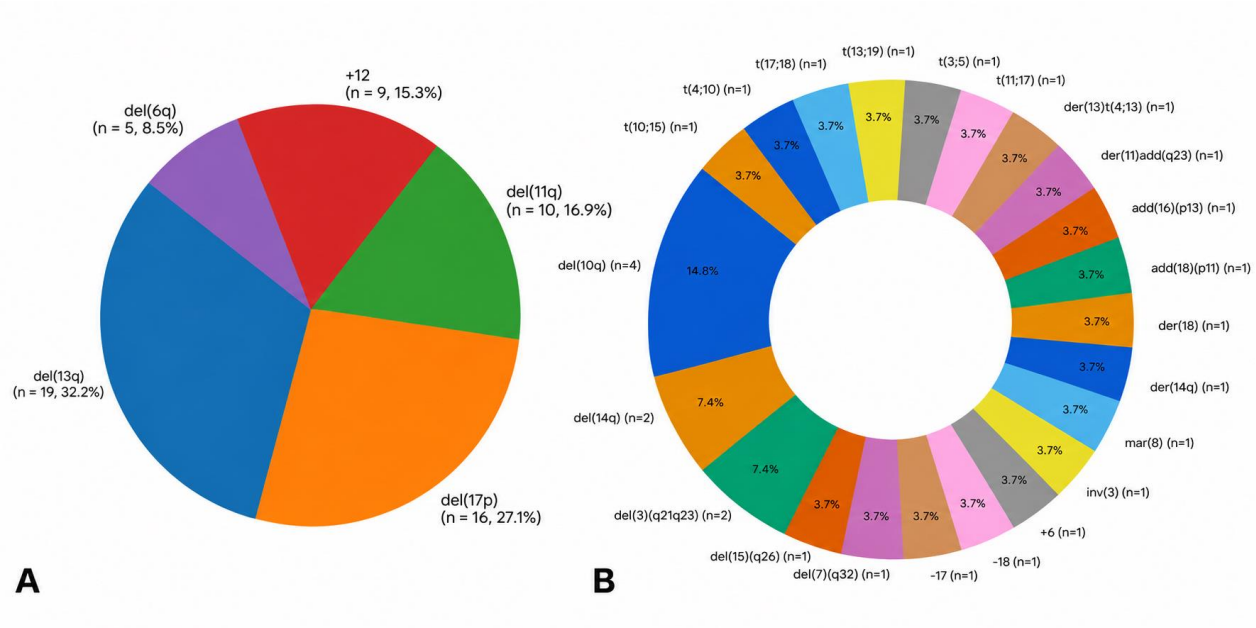


Figure 4. [A] Percentages of chromosomal abnormalities found in CLL patients through CCA and FISH; [B] Percentages of chromosomal abnormalities found in CLL patients through CCA

Cytogenetic profiling revealed a distinct hierarchy of chromosomal alterations within the cohort. As shown in figure 4A, five major recurrent lesions accounted for most detected aberrations, with del(13q) (32.2%) and del(17p) (27.1%) representing the most prevalent events, followed by del(11q), trisomy 12, and del(6q). Conversely, a broad spectrum of lower-frequency and rare cytogenetic lesions, comprising low-incidence structural rearrangements, translocations, and numerical anomalies, was observed across the remaining positive cases in figure 4B. CCA and FISH are highly complementary modalities for the genomic characterization of chronic lymphocytic leukemia CLL. While targeted FISH panels detect specific recurrent aberrations, CCA identifies additional structural and numerical alterations, enabling a more comprehensive evaluation of genomic complexity. These cytogenetic insights carry substantial clinical relevance for prognostic stratification, disease monitoring, and therapeutic decision-making, particularly in patients harboring a complex karyotype (CK), a hallmark of aggressive clinical behavior. To illustrate this diagnostic synergy, we present a representative case of high-risk CK in CLL, diagnosed in September 2015 at the Hematology Unit of Andrea Tortora Hospital. CCA revealed the following karyotype (Figure 5): 46, XY, del(11)(q22), +12, der(17)t(17;18)(p11;p11),-18[10], defining a complex karyotype (≥ 3 aberrations; in this instance, 5 independent lesions). Interphase FISH analysis (Figure 6) confirmed the presence of *TP53* (17p13.1) deletion, *ATM* (11q22.3) deletion, and trisomy 12 in 90% of analyzed nuclei. The seamless integration of CCA and FISH provided an accurate mapping of the patient's cytogenetic landscape, establishing placement within the high-risk CK subgroup.

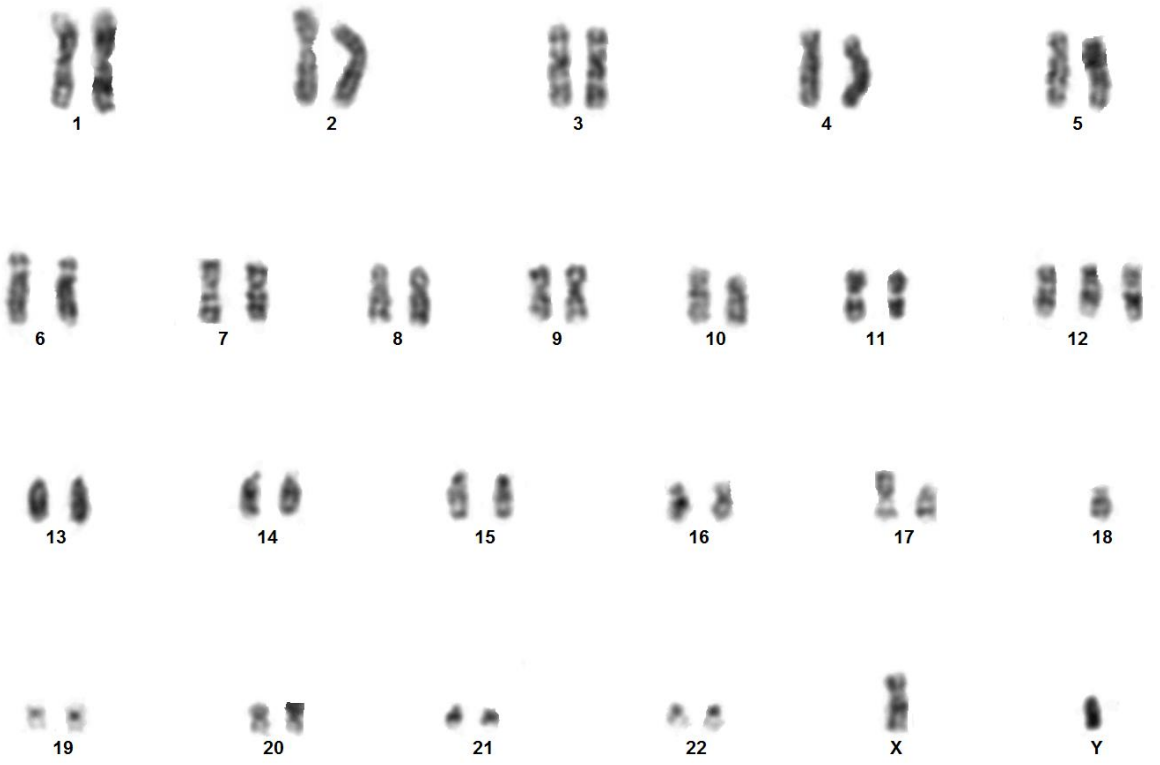


Figure 5. Complex karyotype of patient: 46, XY, del (11) (q22), +12, der (17), t (17;18) (p11; p11), -18. The karyotype was defined using Cytovision Software used for the CCA in the cytogenetics

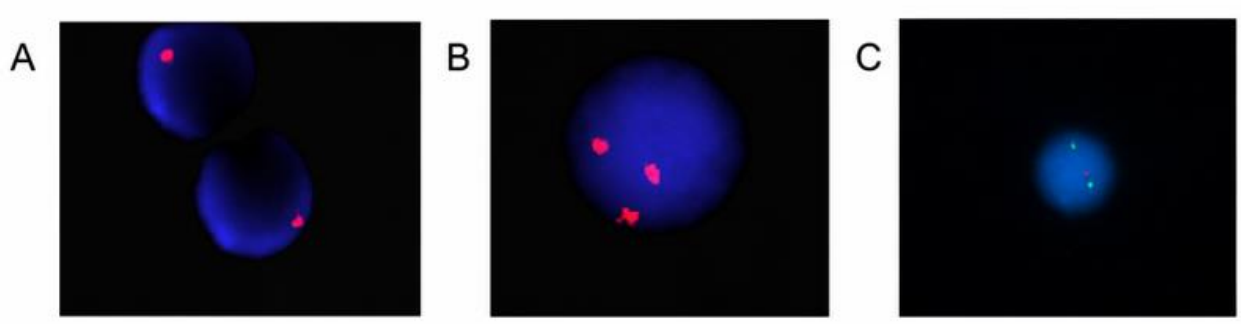


Figure 6. FISH probe signals panel of clinical case. [A] Abnormal hybridization of LSI *ATM* Spectrum Orange Probe associated to deletion of *ATM* locus; [B] CEP 12 Spectrum Orange Probe signals related to trisomy 12; [C] Vysis LSI TP53 Spectrum Orange/ CEP 17 Spectrum Green Probes related to deletion of 17p13.1.

4 Conclusion

The analysis of 30 patients with chronic lymphocytic leukemia (CLL) highlights the importance of integrating conventional chromosome analysis (CCA) and fluorescence in situ hybridization (FISH) for comprehensive genomic characterization and prognostic assessment. By identifying chromosomal abnormalities and

evaluating karyotypic complexity, CCA provides valuable information for disease characterization, risk stratification, therapeutic decision-making, and disease monitoring, complementing the findings obtained through targeted molecular analyses of recurrent gene mutations. The assessment of chromosomal architecture may retain prognostic value even when FISH results are non-informative, as the latter cannot fully capture overall karyotypic complexity. Previous studies have demonstrated that highly complex karyotypes (CK ≥ 5) are associated with unfavorable clinical outcomes, whereas lower levels of karyotypic complexity appear to confer an adverse prognosis primarily in the presence of TP53 abnormalities (Baliakas et al., 2019). Highly complex karyotypes have recently emerged as adverse prognostic factors in patients treated with venetoclax-based regimens, while those harboring fewer than five chromosomal abnormalities showed more favorable therapeutic responses (Furstenau et al., 2023). CK assessment represents a clinically relevant tool for refining risk stratification and informing therapeutic management in patients with CLL, supporting the complementary role of cytogenetic and molecular approaches in clinical practice.

Ethics Statement

This study was conducted in accordance with the principles of the Declaration of Helsinki. The data analyzed were derived from samples collected during routine clinical practice at the public laboratory of conventional and molecular cytogenetics of the hematology unit of P.O. "A. Tortora", Pagani, Asl Salerno, Italy. Informed consent was obtained from all subjects prior to sample collection. All data were anonymized before analysis, and no additional interventions or experimental procedures were performed for the purposes of this study.

Author Contributions

Caliendo Irene and Puopolo Francesco contributed equally to this work. Other authors listed have made a substantial, direct, and intellectual contribution to the work and approved it for publication.

Conflict Of Interest Statement

The authors declare that they have no known competing financial or non-financial interests that could have appeared to influence the work reported in this paper.

Acknowledgments

We are grateful to two anonymous reviewers for their valuable comments on the earlier version of this paper.

References

Baliakas, P., Espinet, B., Mellink, C., Jarosova, M., Athanasiadou, A., Ghia, P., ... & ERIC, the European Research Initiative on CLL. (2022). Cytogenetics in chronic lymphocytic leukemia: ERIC perspectives and recommendations. *Hemasphere*, *6*(4), e707.

Baliakas, P., Jeromin, S., Iskas, M., Puiggros, A., Plevova, K., Nguyen-Khac, F., ... & Stamatopoulos, K. (2019). Cytogenetic complexity in chronic lymphocytic leukemia: definitions, associations, and clinical impact. *Blood, The Journal of the American Society of Hematology*, *133*(11), 1205-1216.

Brenner, H., Gondos, A., & Pulte, D. (2008). Trends in long-term survival of patients with chronic lymphocytic leukemia from the 1980s to the early 21st century. *Blood, The Journal of the American Society of Hematology*, *111*(10), 4916-4921.

Foà, R., Del Giudice, I., Guarini, A., Rossi, D., & Gaidano, G. (2013). Clinical implications of the molecular genetics of chronic lymphocytic leukemia. *Haematologica*, *98*(5), 675.

Fürstenau, M., Thus, Y. J., Robrecht, S., Mellink, C. H., van der Kevie-Kersemaekers, A. M., Dubois, J., ... & Kater, A. P. (2023). High karyotypic complexity is an independent prognostic factor in patients with CLL treated with venetoclax combinations. *Blood*, *142*(5), 446-459.

Haferlach, C., Dicker, F., Schnittger, S., Kern, W., & Haferlach, T. (2007). Comprehensive genetic characterization of CLL: a study on 506 cases analysed with chromosome banding analysis, interphase FISH, IgVH status and immunophenotyping. *Leukemia*, *21*(12), 2442-2451.

Kamaso Navarro, J., Puiggros Metje, A. M., Salido Galeote, M., Melero, C., Rodríguez-Rivera, M., Gimeno Vázquez, E., ... & Espinet Solà, B. (2024). Complex karyotype detection in chronic lymphocytic leukemia: a comparison of parallel cytogenetic cultures using TPA and IL2+ DSP30 from a single center. Cancers. 2024; 16 (12): 2258.

Khoral, P., Atenafu, E. G., Craddock, K. J., Schimmer, A., & Chang, H. (2017). Prognostic effect of complex karyotype, monosomal karyotype, and chromosome 17 abnormalities in B-cell acute lymphoblastic leukemia. *Clinical Lymphoma Myeloma and Leukemia*, *17*(4), 215-219. https://doi.org/10.1016/j.clml.2017.02.009

Kipps, T. J., Stevenson, F. K., Wu, C. J., Croce, C. M., Packham, G., Wierda, W. G., ... & Rai, K. (2017). Chronic lymphocytic leukaemia. *Nature reviews Disease primers*, *3*(1), 16096.

Pekarsky, Y., & Croce, C. M. (2015). Role of miR-15/16 in CLL. *Cell Death & Differentiation*, *22*(1), 6-11.

Redaelli, A., Laskin, B. L., Stephens, J. M., Botteman, M. F., & Pashos, C. L. (2004). The clinical and epidemiological burden of chronic lymphocytic leukaemia. *European journal of cancer care*, *13*(3), 279-287.

Rigolin, G. M., Saccenti, E., Guardalben, E., Cavallari, M., Formigaro, L., Zagatti, B., ... & Cuneo, A. (2018). In chronic lymphocytic leukaemia with complex karyotype, major structural abnormalities identify a subset of patients with inferior outcome and distinct biological characteristics. *British journal of haematology*, *181*(2), 229-233.

Ripollés, L., Ortega, M., Ortuño, F., González, A., Losada, J., Ojanguren, J., ... & Caballín, M. R. (2006). Genetic abnormalities and clinical outcome in chronic lymphocytic leukemia. *Cancer genetics and cytogenetics*, *171*(1), 57-64. https://doi.org/10.1016/j.cancergencyto.2006.07.006

Rose-Zerilli, M. J., Forster, J., Parker, H., Parker, A., Rodríguez, A. E., Chaplin, T., ... & Strefford, J. C. (2014). ATM mutation rather than BIRC3 deletion and/or mutation predicts reduced survival in 11q-deleted chronic lymphocytic leukemia: data from the UK LRF CLL4 trial. *haematologica*, *99*(4), 736.

Serafin, A., Ruocco, V., Cellini, A., Angotzi, F., Bonaldi, L., Trentin, L., & Visentin, A. (2025). Management strategies for patients with chronic lymphocytic leukaemia harbouring complex karyotype. *British Journal of Haematology*, *206*(3), 832-841.

Strickland, S. A., Sun, Z., Ketterling, R. P., Cherry, A. M., Cripe, L. D., Dewald, G., ... & ECOG-ACRIN Cancer Research Group. (2017). Independent prognostic significance of monosomy 17 and impact of karyotype complexity in monosomal karyotype/complex karyotype acute myeloid leukemia: results from four ECOG-ACRIN prospective therapeutic trials. *Leukemia research*, *59*, 55-64. https://doi.org/10.1016/j.leukres.2017.05.010

Visentin, A., Bonaldi, L., Rigolin, G. M., Mauro, F. R., Martines, A., Frezzato, F., ... & Trentin, L. (2021). The complex karyotype landscape in chronic lymphocytic leukemia allows the refinement of the risk of Richter syndrome transformation. haematologica, 107(4), 868.

Visentin, A., Bonaldi, L., Rigolin, G. M., Mauro, F. R., Martines, A., Frezzato, F., ... & Trentin, L. (2019). The combination of complex karyotype subtypes and IGHV mutational status identifies new prognostic and predictive groups in chronic lymphocytic leukaemia. *British journal of cancer*, *121*(2), 150.

Wan, T. S. (2014). Cancer cytogenetics: methodology revisited. *Annals of laboratory medicine*, *34*(6), 413.

Wan, T. S. (2017). Cancer cytogenetics: an introduction. *Cancer Cytogenetics*, 1-10.

Woyach, J. A., Furman, R. R., Liu, T. M., Ozer, H. G., Zapatka, M., Ruppert, A. S., ... & Byrd, J. C. (2014). Resistance mechanisms for the Bruton's tyrosine kinase inhibitor ibrutinib. *New England Journal of Medicine*, *370*(24), 2286-2294.

Biography of Authors



Irene Caliendo
Biologist. Medical Genetics Specialist. Biology Department Health Manager. Head of the Conventional and Molecular Cytogenetics Laboratory for Hematology-Oncology, "A. Tortora" Hospital, ASL Salerno. Professor of Biology and Medicine at the Universities of Salerno and Naples.
Email: irene.caliendo10@gmail.com



Francesco Puopolo
Biologist. Medical Genetics Resident.
Professor and Head of the Department of Medicine and Health Professions at the e-campus Salerno center, Salerno Formazione Business School.
Former Research Fellow in General Pathology at Salerno University.
Email: francescop910@hotmail.com

Carmela Fabbricatore
Biologist. Clinical Biochemistry Specialist. Biologist Specialist at the Conventional and Molecular Cytogenetics Laboratory for Hematology-Oncology, P.O. "A. Tortora" Hospital, ASL Salerno.
Email: fabbri_83@libero.it



Valeria Belsito Petrizzi
Medical Director, Hematology Specialist, P.O. "A. Tortora" Hospital. ASL Salerno.
Email: m.belsitopetrizzi@gmail.com



Giuseppe Malferà
Biologist. Medicine and Surgery Student. Professor at Naples University and Former AIL researcher.
Email: Giuseppe.malfera1997@gmail.com



Maria Langella
Medical Director, Hematology Specialist. Head of Hematological Diagnostics, P.O. "A. Tortora", ASL Salerno.
Email: marialangella@gmail.com

Catello Califano
Medical Director, Hematology Specialist. Head of Hematology Unit, P.O. "A. Tortora", ASL Salerno.
Email: c.califano@aslsalerno.it