22ème المؤتمر الوطني لأمراض الدم
Congrès National d'Hématologie
Dakhla 2026 الداخلة
Du 14 au 16 mai

IMPACT OF ADDITIONAL CYTOGENETIC ABNORMALITIES ON THE EVOLUTION OF CHRONIC MYELOID LEUKEMIA: A RETROSPECTIVE STUDY FROM NORTHERN MOROCCO

T. Arahou[1], O. Hari[1], S. Belkadi[1], I. El Boutahiri[1], S. Regragui[1] K. Sammoud[2], A. Najdi[2]
[1] Department of Clinical Hematology, University Hospital Center Mohamed VI, Tangier, Morocco.
[2] Laboratory of Epidemiology and Public Health, Faculty of Medicine and Pharmacy of Tangier, Abdelmalek Essaadi University, Morocco.

EP-112

INTRODUCTION

Chronic myeloid leukemia (CML) is a clonal myeloproliferative disorder characterized by the presence of the Philadelphia (Ph) chromosome, resulting from the reciprocal translocation t(9;22)(q34;q11). This rearrangement generates the BCR::ABL1 fusion gene, which encodes a constitutively active tyrosine kinase responsible for the leukemogenic process. This makes CML the first malignancy specifically targeted by tyrosine kinase inhibitors (TKIs), with IMATINIB as the standard frontline therapy (1).
Additional cytogenetic abnormalities (ACAs) reflect genomic instability and are considered high-risk features, yet their prognostic value at diagnosis remains uncertain (2). This study aimed to characterize the baseline clinical and biological features of patients with chronic phase (CP) CML in northern Morocco and to evaluate the impact of ACAs at diagnosis on treatment response and disease evolution under first-line IMATINIB therapy.

METHODS

This was a retrospective descriptive-analytic study conducted at the Department of Clinical Hematology, University Hospital of Tangier. We only included patients with newly diagnosed CP-CML treated with first-line IMATINIB between January 2019 and December 2023. Baseline data were collected at diagnosis, including conventional cytogenetics with ≥20 metaphases. Molecular monitoring of BCR::ABL1 transcript levels was performed at 6, 12, and 18 months. Major molecular response (MMR) at 12 months and progression to AP or BP were used to assess treatment outcomes.

RESULTS

PATIENT CHARACTERISTICS

44 Patients	F/M 28/16	50 years (15-94) Median age

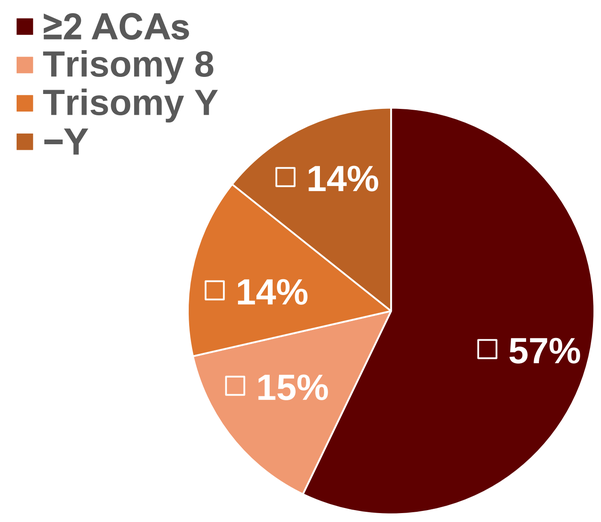
BASELINE FEATURES

Feature	n	%
Splenomegaly	38	86.4%
ACA at Diagnosis	7	15.9%

BIOLOGICAL DATA AT DIAGNOSIS

Median WBC: 250×10⁹/L (33–500)
Median Hb: 10 g/dL (7–13)
Median Platelets: 400×10⁹/L (160–1500)
Median peripheral blasts: 31% (10–67)
Median basophils: 2% (1–10)
Bone marrow blasts: 2.5% (1–5)

DISTRIBUTION OF ACAs



Wang et al. 2016 — Poor prognosis: ≥2 ACAs (n=4) | Good prognosis: +8, +Y, −Y (n=3)

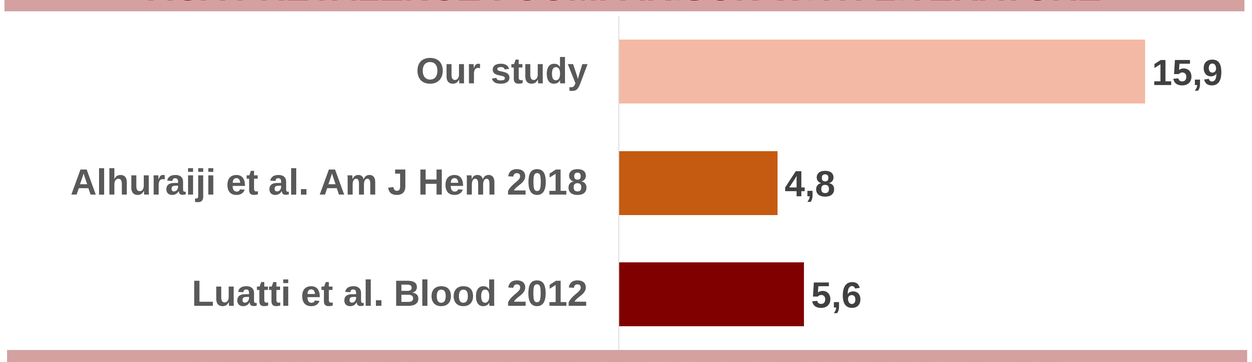
BASELINE COMPARISON ACA(−) vs ACA(+)

Characteristic	ACA(−) n=37	ACA(+) n=7	*p*
Age (median, yrs)	50 (15–94)	53 (32–80)	0.683
WBC ×10⁹/L	250 (33–500)	340 (30–540)	0.312
Hemoglobin g/dL	9.8 (7–13)	8.2 (5.4–10.1)	**0.011 ★**
Platelets ×10⁹/L	400 (160–1500)	370 (200–1000)	0.376

TREATMENT RESPONSE TO 1st LINE IMATINIB

	ACA(−) n=37	ACA(+) n=7	*p*
MMR at 12 months	37.8%	28.5%	1.000
Transformation (blast phase)	0%	28.5%	**0.022 ★**

ACA PREVALENCE : COMPARISON WITH LITERATURE



HEMOGLOBIN : COMPARISON WITH LITERATURE

	ACA(−) n=37	ACA(+) n=7	*p*
Luatti et al.	12.0 (6.4–17.5)	11.8 (8.2–16.0)	0.770
Alhuraiji et al.	12.0 (7.2–15.9)	12.0 (8.8–15.8)	1.000
Our Study	9.8 (7–13)	8.2 (5.4–10.1)	**0.011 ★**

DISCUSSION

The prevalence of ACAs (15.9%) was higher than reported in international series (~5%) (1,2), possibly reflecting the genetic diversity of our population. Notably, 57% of ACA+ cases were classified as poor prognosis by Wang et al., suggesting a potentially more aggressive cytogenetic profile. Consistent with both reference studies (1,2), no significant difference in MMR at 12 months was observed between groups, suggesting that IMATINIB achieves comparable molecular responses regardless of baseline cytogenetic status. However, blast phase transformation occurred exclusively in ACA(+) patients (28.5%, p=0.022), confirming ACAs as a marker of leukemic progression risk. The significantly lower hemoglobin in ACA(+) patients (p=0.011) contrasts with both reference studies (p=0.77 and p=1.00), reflecting possibly more advanced marrow involvement.
Limitations: small sample size, single-centre, retrospective design.

CONCLUSION

ACAs were frequent in our cohort and associated with more severe anemia at diagnosis and an increased risk of leukemic transformation, underscoring their clinical relevance in CP-CML. These findings highlight the genetic heterogeneity of CML in our population and support the need for a large, multicenter Moroccan study to better define the prognostic impact of ACAs.

BIBLIOGRAPHY

(1) Luatti S & al. Additional chromosomal abnormalities in Philadelphia-positive clone: adverse prognostic influence on frontline imatinib therapy: a GIMEMA Working Party on CML analysis. Blood. 2012;120(4):761–767.
(2) Alhuraiji A & al. Prognostic significance of additional chromosomal abnormalities at diagnosis in chronic myeloid leukemia treated with frontline tyrosine kinase inhibitors. Am J Hematol. 2018;93(1):84–90