Precipio Spotlight:

BCR::ABL1 Panel Testing with ABL1 Resistance Reflex Testing Provides Crucial Patient Monitoring Data to Identify Early Therapy Resistance To Guide Treatment Adjustments for TKI-Resistant CML Patient

Abstract

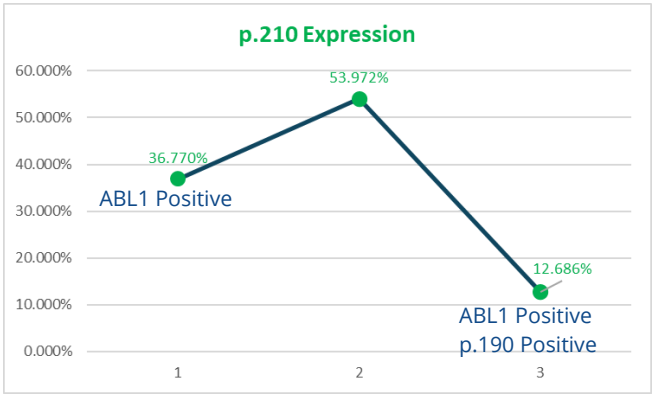
This Precipio Spotlight investigates the utility of implementing comprehensive breakpoint testing for BCR::ABL1 oncogene expression and ABL1 gene mutation analysis in the context of managing treatment for chronic myeloid leukemia (CML) patients undergoing tyrosine kinase inhibitor (TKI) therapy. This underscores the significance of early detection of TKI drug resistance indicators to facilitate timely adjustments in patient therapy, thereby improving treatment outcomes and patient care management.

Case Background

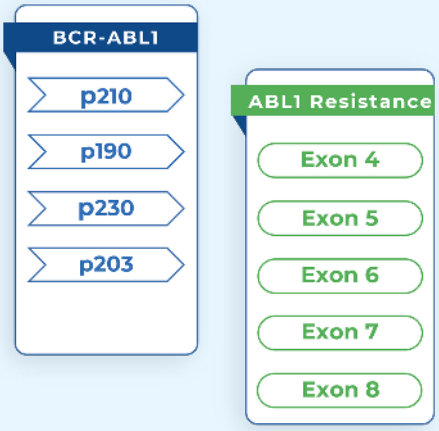
- 78 YO | Male
- History of CML
- Iron deficiency anemia secondary to blood loss
- Anemia unspecified

Case Work Up and Findings

- Bloodhound BCR::ABL1 Panel (p.190, p.203, p.210, p.230) run
- ABL1 Resistance panel run
- Change in CML-related molecular target expressions over monitoring intervals



Technology Advantage



Bloodhound BCR::ABL1 and ABL1 Resistance Panels

The Precipio Difference:
Comprehensive oncogene-related monitoring for CML patients can help detect early indications of TKI resistance. By monitoring ABL1 status along with broader fusion status, Precipio was able to detect an early indicator for the subsequent elevation in p.210 expression as well as the co-expression of other CML-related fusion isoforms.

More Case Studies

For more information please contact:
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MOLECULAR

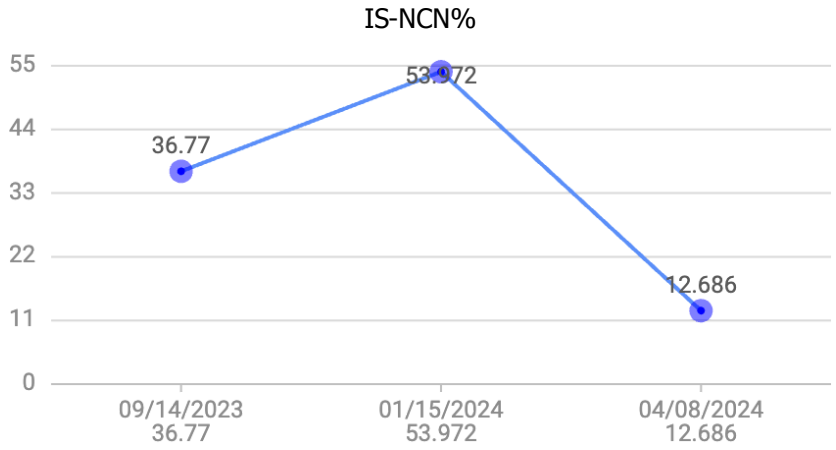
Patient: John Doe
DOB/Gender: XX/XX/19XX (78 yrs) - Male
Patient ID/MRN: 12345
Date Collected: XX/XX/20XX XX:XX

Case#: PXX-XXXXX
Status: Final
Report Category:
Detected

Provider: Jane Smith, MD
XYZ Hematology/Oncology
Tel: + 800-123-4567
fax: +800-765-4321

DIAGNOSIS:

Peripheral blood:
- BCR::ABL1 p210 fusion was detected at 12.686% IS-NCN.
- BCR::ABL1 p190 fusion was detected.
- No BCR::ABL1 p203 fusion was detected.
- No BCR::ABL1 p230 fusion was detected.
- ABL1 mutation analysis: ABL1 exon 8 variant detected (p.E459K).



INTERPRETATION

(HemeScreen BCR::ABL1): Mutations within the BCR/ABL1 kinase domain of patients with chronic myeloid leukemia or acute lymphoblastic leukemia with Philadelphia chromosome are the most commonly identified mechanism associated with resistance to kinase inhibitors. It has been reported that most patients with detectable BCR/ABL1 kinase domain mutations are imatinib resistant or resistant to other kinase inhibitors.

High Resolution Melt analysis was performed to identify BCR/ABL1 fusion isoforms (p190, p210, p230, p203) for diagnostic, therapeutic, monitoring and drug-response of Philadelphia chromosome positive leukemic cells. BCR/ABL1 translocations in the major breakpoint cluster region resulting in fusion protein are seen in nearly all cases of chronic myelogenous leukemia (CML), acute lymphoblastic leukemia (ALL), acute myelogenous leukemia (AML) and myeloproliferative neoplasms (MPN/MDS).

p190 BCR/ABL1 fusion encodes micro transcripts e19a2 common in Philadelphia-positive B-ALL and has been reported in 1% of CML cases. p203 BCR/ABL1 fusion encodes transcripts e13a3 (b2a3), a precursor to p210 fusion. p210 BCR/ABL1 fusion encodes major transcripts e14a2 (b2a2) or e13a2 (b3a2) proteins common in CML. p230 BCR/ABL1 fusion encodes transcripts e19a2 is known as µ-BCR/ABL1 transcript and common in neutrophilic-chronic myeloid leukemia.