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Review

BCR::ABL1 Mutational Profiling in US Patients With Chronic-phase Chronic Myeloid Leukemia on Tyrosine Kinase Inhibitors

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Abstract

Background/Aim: Widespread use of BCR::ABL1 tyrosine kinase inhibitors (TKIs) has transformed the clinical landscape of chronic myeloid leukemia (CML). However, *BCR::ABL1* mutation-driven treatment resistance challenges optimal care and outcomes. The purpose of this study was to ascertain real-world practices concerning such mutational profiling in US hematology and oncology practices, which were mainly community-based.

Materials and Methods: This is the first chart review of *BCR::ABL1* kinase domain mutational profiling in the US. In this study of the Cardinal Health Oncology Provider Extended Network (OPEN), 13 hematologists and/or oncologists selected charts of adults with chronic-phase CML for approximately equal numbers of patients who underwent such testing (Cohort 1; n=26) or did not (Cohort 2; n=25).

Results: Across most time points, failure and warning signs by molecular testing were not significantly more frequent in patients who did or did not undergo *BCR::ABL1* mutational profiling. Conversely, similar frequencies of optimal milestones were observed in patients with or without testing. Patients who underwent *BCR::ABL1* mutational profiling were more likely to have splenomegaly (p=0.0069) versus those who did not.

Conclusion: There were few significant differences in failure and warning signs by molecular testing in patients who did or did not undergo mutational profiling. Patients who underwent *BCR::ABL1* mutational profiling were significantly more likely to have splenomegaly, which is not included in consensus guidelines as a reason to conduct such testing.

continued

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Taken together, these findings raise potential concerns about consensus guideline adherence as a means to optimize CML management for TKI resistance.

Keywords: *BCR::ABL1*, chronic myeloid leukemia, mutation, treatment resistance, tyrosine kinase inhibitors, review.

Introduction

The development and widespread use of effective and well-tolerated BCR::ABL1 tyrosine kinase inhibitors (TKIs) have transformed the treatment landscape of chronic myeloid leukemia (CML), essentially normalizing life expectancy for most patients with CML (1). However, *BCR::ABL1* mutation-driven resistance to TKIs after switching therapy continues to be a clinical challenge. Even after switching from imatinib to a second-generation TKI, 37% to 52% of patients do not respond, while 23% to 26% lose their initial major cytogenetic response within 2 years (2-6).

Other adverse outcomes associated with TKI resistance and regimen change include reduced health-related quality of life (7), as well as increased health resource utilization and costs per increasing lines of therapy (8, 9).

According to evidence-based consensus guidelines, *BCR::ABL1* kinase domain mutational profiling should be contemplated if treatment milestones [expressed as BCR::ABL1 on the International Scale (IS)] are not met (10-13). For instance, the National Comprehensive Cancer Network recommends consideration of such testing if BCR::ABL1 IS exceeds 10% at 3 to 12 months and >1% to 10% at 12 months (12). The 2020 European LeukemiaNet (ELN) guidelines stated that, "In the absence of [*BCR::ABL1* kinase domain] mutations, there can be no clear recommendation for any particular [second-generation] TKI: all second-line TKIs are effective, but there are no studies comparing the TKIs with each other" (11). Patient comorbidities also inform such treatment decisions.

Although *BCR::ABL1* mutational profiling is important to prevent further treatment resistance, an early survey of US and European hematologists and oncologists found that >60% of US respondents were not acquainted with *BCR::ABL1* kinase domain testing or had not ordered such profiling (14). In the real-world, multinational Treatment and Management of CML in Real Practice: eGm EvaluaTion (TARGET) (UK) study of 257 patients with chronic-phase CML (CP-CML) receiving front-line TKIs (*n*=203 imatinib) from 2013 to 2017, *BCR::ABL1* kinase domain mutational analysis was conducted in fewer than half (49%) of patients whose TKIs were switched because of treatment resistance, and 23% of patients with treatment failure according to ELN standards did not change TKIs (15). In the overall TARGET study (16), only about half (51%) of the health care provider (HCP) respondents performed molecular monitoring as frequently as recommended by the ELN.

Contemporary practices concerning such testing in US real-world clinical settings are not well documented. The objective of this observational study, conducted mainly in community clinical settings, was to ascertain real-world US provider practices concerning *BCR::ABL1* mutational testing in patients with CP-CML and raise awareness concerning its relevance.

Materials and Methods

Datasets. This study consisted of a retrospective US, multisite, physician-abstracted chart review of CP-CML patients as a cross-sectional physician survey. Participating HCPs were identified from the Cardinal Health Oncology Provider Extended Network (OPEN), a geographically diverse and group purchasing organization (GPO) and an electronic medical record (EMR) software agnostic community. This network includes more than 7,000 HCPs from across the US, predominantly inclusive of community practices (>75%) and ranging in size from solo practitioners to physicians practicing in hospital systems.

Eligible HCPs treated at least one eligible patient with CP-CML, conducted molecular monitoring, and had accessibility

to *BCR::ABL1* mutational studies. When completing data abstraction, physicians were instructed to randomly select patients meeting the following patient eligibility criteria: 1) confirmed diagnosis of CP-CML; 2) ≥18 years of age at CP-CML diagnosis; 3) initiated first-line (1L) TKI therapy on or after January 1, 2019, and had ≥12 months of follow-up before data collection; and 4) had ≥3 months of follow-up data after initiation of second-line (2L) TKI. Excluded were charts of patients who 1) received treatment for CML as part of a clinical trial, 2) had been diagnosed with any other malignancy, or 3) were in accelerated- or blast-phase CML at diagnosis. A new line of therapy was defined as starting an alternative TKI and not dose escalating, interrupting, or otherwise adjusting the same TKI.

Cohort 1 (*n*=26) included patients who underwent mutational analysis, while Cohort 2 (*n*=25) comprised individuals who did not. Before abstracting patient data into electronic case report forms (eCRFs), physicians completed a brief, one-time survey to collect information related to their characteristics and current practices for assessing outcomes of CML treatments. Patient-level data collected in the eCRF pertained to demographics, clinical characteristics, CML testing patterns (*e.g.*, frequency, assay), and treatment patterns (*e.g.*, regimens, dates of initiation/discontinuation, rationale for discontinuation).

Quality control (QC) was conducted by Cardinal Health Specialty Solutions (CHSS) and included extensive testing of the survey and the eCRF to ensure functionality across web-based user environments, looping logic to ensure proper alignment of data-related fields (required responses to certain fields before entering data into subsequent fields), and other programmatic checks to ensure reduced input of erroneous data (*e.g.*, maximum number of patients, allowable date ranges).

After data collection, CHSS clinical research staff reviewed each submitted eCRF for implausible dates (*e.g.*, date of death before last date of treatment), nonstandard treatments (*e.g.*, treatment regimens not known to be used for CML), or other clinical data that were inconsistent with known clinical standards and outcomes. In addition to reviewing the data submitted by the clinical research team, the study statistician conducted an analysis of submitted data to identify any data points inconsistent with the study population average (outliers). Data flagged during QC checks that could not be resolved were excluded from analysis.

A minimum of 10 physicians (*N*=13) representing unique sites completed the survey and collected data for a target sample of approximately 50 patients. Each provider could complete eCRFs for up to 5 patients, in order to minimize potential bias and oversampling. From the CRFs, providers who had more than 5 patients meeting eligibility criteria were advised to select, and abstract de-identified data from, patients at random. They entered data for subsequent patients consecutively in chronological order if they had fewer than 5 patients.

During the data collection period, physicians abstracted medical-chart data documented at the time of CML diagnosis and during follow-up visits for patients under their care and then entered the data into eCRFs. The data collection period extended over a 2-week interval in June 2023.

Data points collected in the eCRF included: 1) HCP demographics and clinical characteristics (*e.g.*, treatment setting community or academic); 2) baseline patient demographic and clinical factors at diagnosis and TKI treatment initiation; 3) reasons for and timing of performing mutational analysis; as well as 4) treatment patterns. Participating physicians performed their own medical chart reviews.

Statistical analysis. This study was largely descriptive, with no *a priori* specified hypotheses. As such, the final sample size informed whether statistical comparisons were appropriate. All data processing and analysis were performed using SAS v9.4 (SAS Institute, Cary, NC, USA). All variables were summarized using frequency counts and proportions for dichotomous and other categorical variables, while measures of centrality (mean, median) and dispersion [minimum, maximum, standard deviation (SD), interquartile range (IQR), as appropriate] were used for continuous variables. Analyses were conducted using *t*-tests, Wilcoxon rank sum, ANOVA, or Chi-square/

Fisher's Exact analyses as appropriate. A two-sided α=0.05 was considered statistically significant.

Ethical approval. All abstracted chart data were de-identified in compliance with the Health Insurance Portability and Accountability Act. Ethics committee approval was not required for this observational study.

Results

Most HCPs were in large private community practices; approximately 15% practiced at academic medical centers or affiliated teaching hospitals. About 75% to 80% of HCPs practiced in the south (46.2%) or west (30.8%) and in either urban (53.8%) or suburban (30.8%) settings. All providers practiced medical oncology and/or hematology. Most patients (60.8%) were men, white (68.6%), and commercially insured (62.7%); 27.5% were Medicare beneficiaries (Table SI). The median (range) patient age at diagnosis was 55.0 (20.0-78.0) years (Table SII). A total of 94.1% of patients had an ECOG performance status of 0 or 1. No patient progressed to blast-phase CML, and all patients were alive at the time of the chart review (94.1% on active therapy).

The mean (SD) number of lines of therapy was 2.1 (0.3), and the mean (SD) time from diagnosis to 1L treatment initiation was 2.5 (3.1) months (Table SII). Median (range) treatment duration was 14.0 (1.0-120.0) months in 1L, 12.0 (3.0-37.0) months in 2L, and 15.0 (4.0-35.0) months in 3L (Table SII). Patients who had mutational profiling were significantly more likely to have mild or moderate splenomegaly (Fisher's exact test p=0.0069) compared to their counterparts without such testing.

First-line therapy was mostly with imatinib (n=32, 62.7%), followed by dasatinib (n=10, 19.6%) and nilotinib (n=9, 17.6%). Second-line treatment was most frequently with dasatinib (n=19, 37.3%), followed by bosutinib (n=15, 29.4%), nilotinib (n=13, 25.5%), and ponatinib (n=3, 5.9%). Only one patient received treatment with asciminib, as 3L therapy (data not shown because numbers of patients in 3L were small).

Reasons for discontinuing first and second salvage therapies included clinically defined disease progression (treatment resistance) in 41.2% and 33.3%, as well as toxicity/intolerance in 37.3% and 50.0%, respectively. The frequency of mutational profiling at baseline was 17.6% (Table I). Most testing at baseline was performed using next-generation sequencing (NGS) or quantitative polymerase chain reaction (PCR) (Table I). During 1L treatment, the frequency of mutational testing was 21.6%, mainly by quantitative PCR (63.6%), followed by NGS (27.3%) (Table II). A higher frequency of mutational testing was observed in second salvage treatment: 41.2%. Once again, the leading modality was quantitative PCR (66.7%), followed by NGS (28.6%) (Table III).

Table IV and Table V summarize cytogenetic/molecular testing by line of therapy. In 1L, frequencies of cytogenetic/molecular testing were higher at treatment initiation (82.4%), 3 months (56.9%), and 6 months (60.8%), and lower at 12 months. No such testing was conducted in 11.8% of patients during 1L treatment (Table IV). In 2L, frequencies of cytogenetic/molecular testing were also higher at treatment initiation (60.8%), 3 months (56.9%), and 6 months (66.7%), and lower at 12 months (49.0%). No testing was conducted in 23.5% of patients during 2L treatment.

Physicians did not consistently perform mutational profiling according to consensus treatment guidelines. In 1L, warning (25.0% *vs.* 0) and failure (4.2% *vs.* 0) signs (according to BCR::ABL1 IS levels) were significantly more frequent in patients with (*vs.* without) mutational testing at 6 months (p=0.04) but not 3 months (warning 4.2% *vs.* 4.8%; failure 8.3% *vs.* 4.8%; p=0.66) or 12 months (warning 20.8% *vs.* 0; failure 8.3% *vs* 9.5%; p=0.15) (Table IV). In the 2L setting, warning and failure signs were not significantly more frequent in patients with (*vs.* without) mutational testing at 3 (warning 5% *vs.* 0; failure 5% *vs.* 0; p=1.00), 6 (warning 15.0% *vs.* 5.3%; failure 0 *vs.* 0; p=0.84), and 12 (warning 0 *vs.* 0; failure 0 *vs.* 5.3%; p=0.39) months (Table V). However, numbers of patients with warning or failure signs were small. On the other hand, the frequencies of optimal treatment milestones were also high in patients

Table I. *Mutational testing completed during treatment.*

	All patients (N=51)	Mutational testing performed at/ before second TKI initiation: Yes (n=26)	Mutational testing performed at/ before second TKI initiation: No (n=25)
At initial diagnosis, n (%)			
Yes	9 (17.6)	9 (34.6)	0
No	42 (82.4)	17 (65.4)	25 (100)
Type of mutational testing completed at initial diagnosis, n (%)*			
Quantitative PCR	5 (55.6)	5 (55.6)	0
Digital PCR	3 (33.3)	3 (33.3)	0
Sanger sequencing	1 (11.1)	1 (11.1)	0
NGS	7 (77.8)	7 (77.8)	0
Other	0	0	0

NGS, Next-generation sequencing; PCR, polymerase chain reaction; TKI, tyrosine kinase inhibitor. *The denominator for "types of mutational testing completed at initial diagnosis" is 9 ("yes" to testing at initial diagnosis).

Table II. *Mutational testing completed during treatment in patients who received 1L treatment.*

	All patients (N=51)	Mutational testing performed at/ before second TKI initiation: Yes (n=26)	Mutational testing performed at/ before second TKI initiation: No (n=25)
Patients who received 1L treatment (denominator)			
During 1L treatment, n (%)			
Yes	11 (21.6)	11 (42.3)	0
No	40 (78.4)	15 (57.7)	25 (100.0)
Type of mutational testing completed during 1L treatment, n (%)			
Quantitative PCR	7 (63.6)	7 (63.6)	0
Digital PCR	1 (9.1)	1 (9.1)	0
Sanger sequencing	0	0	0
NGS	3 (27.3)	3 (27.3)	0
Other	0	0	0

1L, First-line treatment; NGS, next-generation sequencing; PCR, polymerase chain reaction; TKI, tyrosine kinase inhibitor.

who underwent mutational testing. In both 1L and 2L treatments, there was a statistically significant trend toward more frequent ordering of fluorescence *in situ* hybridization (FISH) for cytogenetic/molecular testing among patients receiving mutational profiling (Chi-square $p \leq 0.001$) (Table IV and Table V).

Discussion

Evidence from the first chart review study of mainly community provider practices concerning *BCR::ABL1* mutational profiling in the management of CP-CML suggests that US hematologists and oncologists do not consistently perform *BCR::ABL1* mutational profiling according to consensus treatment guidelines. At most timepoints, failure or warning signs were not significantly more frequent in patients who did or did not undergo mutational testing. Conversely, optimal treatment signs were also not significantly different in these two groups, meaning that many patients received testing that was not warranted by the consensus guidelines. Splenomegaly was also significantly more frequent in patients with (*vs.* without)

Table III. *Mutational testing completed during treatment in patients who received 2L treatment.*

	All patients (N=51)	Mutational testing performed at/ before second TKI initiation	
		Yes (n=26)	No (n=25)
Patients who received 2L treatment (denominator)			
During 2L treatment, n (%)			
Yes	21 (41.2)	21 (80.8)	0
No	30 (58.8)	5 (19.2)	25 (100.0)
Type of mutational testing completed during 2L treatment, n (%)			
Quantitative PCR	14 (66.7)	14 (66.7)	0
Digital PCR	1 (4.8)	1 (4.8)	0
Sanger sequencing	0	0	0
NGS	6 (28.6)	6 (28.6)	0
Other	0	0	0

2L, Second-line treatment; NGS, next-generation sequencing; PCR, polymerase chain reaction; TKI, tyrosine kinase inhibitor.

BCR::ABL1 mutational profiling. Although spleen size may be an important early sign of CML resistance, it is not currently a consensus-guided reason to conduct such testing.

These findings may be important in a setting of TKI treatment failure because mutational testing informs subsequent decisions to utilize different TKIs. 1) The "gatekeeper" T315I kinase domain mutation is resistant to imatinib and all second-generation TKIs; 2) V299L, G250E, and F317L mutations are resistant to bosutinib; 3) F317L/V/I/C and V299L mutations are resistant to dasatinib; 4) Y253H, E255K/V, and F359V/C/I mutations are resistant to nilotinib; and 5) A337T, P465S, and F359V/I/C mutations are resistant to the allosteric regulator specifically targeting the myristoyl pocket (STAMP) inhibitor asciminib (12). Olverembatinib (as monotherapy), an investigational novel third-generation TKI with broad-spectrum *BCR::ABL1* mutational coverage, has demonstrated activity against compound mutations (genetic variants characterized by two or more mutations within the same *BCR::ABL1* allele), including T315I-inclusive compound mutations, which can complicate treatment with other TKIs (17, 18). A phase 1b trial demonstrated favorable tolerability and efficacy of olverembatinib in patients with resistance or intolerance to third-generation TKI ponatinib and/or the STAMP inhibitor asciminib (17).

Several observational studies have been conducted to assess the effectiveness of CML care, mainly in patients with CP-CML. Most of these studies have focused on the burdens of 1) under-monitoring with respect to consensus-guided cytogenetic and molecular milestone testing (19) and 2) later-line TKI management in general (20) rather than *BCR::ABL1* mutational profiling in particular. For instance, a French retrospective multicenter study (chart review) conducted before the approval of asciminib demonstrated that patients harboring the T315I mutation received up to six lines of TKIs (20).

Real-world evidence (21) [including the multicenter SIMPLICITY prospective cohort study (NCT01244750) (22)] shows that only 40% or fewer of patients undergo PCR molecular tests three to four times within the first year (especially early within treatment) after their CML diagnosis as recommended by both the ELN (10, 11) and National Comprehensive Cancer Network (12). Of interest, and paralleling the present findings, one study showed that US community-based oncologists were more likely to conduct molecular monitoring in patients with enlarged spleens (23). It is possible that tangible manifestations of disease could trigger providers to intensify monitoring because of perceived worse disease or higher risk.

Observational studies have pointed toward a range of enhanced outcomes in patients with CP-CML who receive

Table IV. *Cytogenetic and molecular testing during 1L treatment.*

		Mutational testing performed at/before second TKI initiation			
	All patients (N=51)	Yes (n=26)	No (n=25)	Test	*p*-Value
Cytogenetic/molecular testing completed during 1L treatment, n (%)					
Patients who received 1L (denominator)	51 (100.0)	26 (100.0)	25 (100.0)	NC	
During 1L treatment, n (%)					
Yes	45 (88.2)	24 (92.3)	21 (84.0)	Fisher's Exact	0.4189
No	6 (11.8)	2 (7.7)	4 (16.0)		
Type of cytogenetic/molecular testing completed during 1L treatment among patients who received 1L treatment, n (%)					
Bone marrow biopsy	29 (56.9)	17 (65.4)	12 (48.0)	Chi-Square	0.2102
FISH	26 (51.0)	19 (73.1)	7 (28.0)	Chi-Square	0.0013
PCR	39 (76.5)	20 (76.9)	19 (76.0)	Chi-Square	0.9381
Testing not performed	6 (11.8)	2 (7.7)	4 (16.0)	Fisher's Exact	0.4189
Timepoint(s) when cytogenetic/molecular testing was completed during 1L treatment among patients who received 1L treatment, n (%)					
At treatment initiation	42 (82.4)	24 (92.3)	18 (72.0)	Fisher's Exact	0.0751
At 3 months	29 (56.9)	12 (46.2)	17 (68.0)	Chi-Square	0.1153
At 6 months	31 (60.8)	15 (57.7)	16 (64.0)	Chi-Square	0.6446
At 12 months	20 (39.2)	11 (42.3)	9 (36.0)	Chi-Square	0.6446
At 14 months	1 (2.0)	0	1 (4.0)	Fisher's Exact	0.4902
Testing not performed	6 (11.8)	2 (7.7)	4 (16.0)	Fisher's Exact	0.4189
Reason for completing cytogenetic/molecular testing during 1L treatment among patients who received 1L treatment, n (%)					
Baseline assessment	38 (74.5)	22 (84.6)	16 (64.0)	Chi-Square	0.0913
Assessing residual disease	8 (15.7)	7 (26.9)	1 (4.0)	Fisher's Exact	0.0496
Management of CP-CML	22 (43.1)	12 (46.2)	10 (40.0)	Chi-Square	0.6573
Monitor for response/progression	38 (74.5)	18 (69.2)	20 (80.0)	Chi-Square	0.3777
Determining risk/prognosis	0	0	0		
Other	0	0	0		
Testing not performed	6 (11.8)	2 (7.7)	4 (16.0)	Fisher's Exact	0.4189
Results from cytogenetic/molecular testing during 1L reported by provider among patients who had testing performed, n (%)					
Patients who had cytogenetic/molecular testing performed (denominator)	45 (100.0)	24 (100.0)	21 (100.0)	NC	
Results reported, n (%)					
Yes	41 (91.1)	22 (91.7)	19 (90.5)	Fisher's Exact	1.0000
No	4 (8.9)	2 (8.3)	2 (9.5)		
Type of cytogenetic/molecular response assessed during 1L treatment among patients who had testing performed, n (%)					
Only cytogenetic response reported	3 (6.7)	3 (12.5)	0	Fisher's Exact	0.2364
Only molecular response reported	21 (46.7)	10 (41.7)	11 (52.4)	Chi-Square	0.4723
Response assessment type reported during 1L	36 (80.0)	19 (79.2)	17 (80.9)	Fisher's Exact	1.0000
% *BCR::ABL1* IS	36 (100.0)	19 (100.0)	17 (100.0)	NC	
Molecular response	28 (77.8)	14 (73.7)	14 (82.4)	Fisher's Exact	0.6950
Results not reported	4 (8.9)	2 (8.3)	2 (9.5)	Fisher's Exact	1.0000
Achievement of treatment milestones during 1L based on % *BCR::ABL1* (IS), among patients with cytogenetic/molecular testing performed					
Patients who had cytogenetic/molecular testing performed (denominator)	45 (100.0)	24 (100.0)	21 (100.0)	NC	

Table IV. *Continued*

Table IV. *Continued*

		Mutational testing performed at/before second TKI initiation			
	All patients (N=51)	Yes (n=26)	No (n=25)	Test	*p*-Value
3 months post initiation, n (%)					
Optimal	25 (55.6)	15 (62.5)	10 (47.6)	Fisher's Exact	0.6596
Warning	2 (4.4)	1 (4.2)	1 (4.8)		
Failure	3 (6.7)	2 (8.3)	1 (4.8)		
Response not reported	15 (33.3)	6 (25.0)	9 (42.9)		
6 months post initiation, n (%)					
Optimal	24 (53.3)	11 (45.8)	13 (61.9)	Fisher's Exact	0.0417
Warning	6 (13.3)	6 (25.0)	0		
Failure	1 (2.2)	1 (4.2)	0		
Response not reported	14 (31.1)	6 (25.0)	8 (38.1)		
12 months post initiation, n (%)					
Optimal	17 (37.8)	9 (37.5)	8 (38.1)	Fisher's Exact	0.1527
Warning	5 (11.1)	5 (20.8)	0		
Failure	4 (8.9)	2 (8.3)	2 (9.5)		
Response not reported	19 (42.2)	8 (33.3)	11 (52.4)		

1L, First-line treatment; 2L, second-line treatment; 3L, third-line treatment; CML, chronic myeloid leukemia; CP-CML, chronic-phase chronic myeloid leukemia; FISH, fluorescence *in situ* hybridization; LOT, line of therapy; NC, not calculated; NGS, next-generation sequencing; PCR, polymerase chain reaction; TKI, tyrosine kinase inhibitor.

consensus-recommended molecular monitoring, including less frequent disease progression, longer progression-free survival, higher treatment adherence, as well as lower health-care costs secondary to fewer hospitalizations and emergency-room visits (24-27). It is possible that more frequent molecular monitoring enables physicians to adjust therapy more effectively by identifying impending reductions in treatment responses in a more proactive manner. However, a US physician survey identified difficulties in implementing consensus guidelines, including limited familiarity and resources in general and concerns about the cost of monitoring in particular (28).

The pivotal question for future research is whether such associations exist between conducting consensus-guided *BCR::ABL1* mutational profiling and favorable treatment outcomes. In addition to large prospective, longitudinal studies, one observational-research approach to evaluate this possibility might be a case-control study to evaluate the frequency of such testing in CP-CML patients with (*vs.* without) disease progression or in patients with shorter (*vs.* longer) event-free, progression-free, or overall survival. If such favorable associations can be established, and given that consensus guidelines clearly recommend *BCR::ABL1* profiling in a setting of TKI failure, a secondary research objective might be to identify barriers hindering adoption of such testing among HCPs. Denial of insurance coverage does not appear to be a barrier. Various modalities related to electronic health records (*e.g.*, reminders and alerts, templates and order sets, data tracking) could conceivably be used in the future to promote and monitor adherence to consensus guidelines related to *BCR::ABL1* mutational profiling in the event of TKI treatment resistance.

Limitations of the current study include its retrospective, descriptive nature and the overall small number of physicians (*N*=13) and patients (*N*=51) included, particularly patients with warning and failure signs. These limited numbers of participating HCPs reflected resource constraints and not the overall familiarity of Cardinal Health OPEN providers with monitoring guidelines (and hence an unwillingness to participate). HCPs were self-selected, voluntarily opting to participate in the study. Although we acknowledge the potential for participation bias, we do not have data on non-responding providers and therefore cannot speculate

Table V. *Cytogenetic and molecular testing during 2L treatment.*

		Mutational testing performed at/before second TKI initiation			
	All patients (N=51)	Yes (n=26)	No (n=25)	Test	*p*-Value
Cytogenetic/molecular testing completed during 2L treatment, n (%)					
Patients who received 2L treatment (denominator)	51 (100.0)	26 (100.0)	25 (100.0)	NC	
During 2L treatment, n (%)					
Yes	39 (76.5)	20 (76.9)	19 (76.0)	Chi-Square	0.9381
No	12 (23.5)	6 (23.1)	6 (24.0)		
Type of cytogenetic/molecular testing completed during 2L treatment, n (%)*					
Bone marrow biopsy	7 (13.7)	6 (23.1)	1 (4.0)	Fisher's Exact	0.0993
FISH	18 (35.3)	15 (57.7)	3 (12.0)	Chi-Square	0.0006
PCR	38 (74.5)	19 (73.1)	19 (76.0)	Chi-Square	0.8108
Testing not performed	12 (23.5)	6 (23.1)	6 (24.0)	Chi-Square	0.9381
Timepoint(s) when cytogenetic/molecular testing was completed during 2L treatment among patients who received 2L treatment, n (%)*					
At treatment initiation	31 (60.8)	19 (73.1)	12 (48.0)	Chi-Square	0.0667
At 3 months	29 (56.9)	12 (46.2)	17 (68.0)	Chi-Square	0.1153
At 6 months	34 (66.7)	15 (57.7)	19 (76.0)	Chi-Square	0.1656
At 12 months	25 (49.0)	13 (50.0)	12 (48.0)	Chi-Square	0.8864
Other					
Every 3 months	2 (3.9)	0	2 (8.0)	Fisher's Exact	0.2353
Testing not performed	12 (23.5)	6 (23.1)	6 (24.0)	Chi-Square	0.9381
Results from cytogenetic/molecular testing during 2L reported by provider among patients who had testing performed, n (%)					
Patients who had cytogenetic/molecular testing performed (denominator)	39 (100.0)	20 (100.0)	19 (100.0)	NC	
Results reported, n (%)					
Yes	36 (92.3)	19 (95.0)	17 (89.5)	Fisher's Exact	0.6050
No	3 (7.7)	1 (5.0)	2 (10.5)		
Type of cytogenetic/molecular response assessed during 2L treatment among patients who had testing performed, n (%)					
Only cytogenetic response reported	1 (2.6)	1 (5.0)	0	Fisher's Exact	1.0000
Only molecular response reported	21 (53.8)	9 (45.0)	12 (63.2)	Chi-Square	0.2556
Response assessment type reported during 2L	35 (89.7)	18 (90.0)	17 (89.5)	Fisher's Exact	1.0000
% *BCR::ABL1* IS	35 (100.0)	18 (100.0)	17 (100.0)	NC	
Molecular response	32 (91.4)	16 (88.9)	16 (94.1)	Fisher's Exact	1.0000
Results not reported	3 (7.7)	1 (5.0)	2 (10.5)	Fisher's Exact	0.6050
Achievement treatment milestones during 2L based on % *BCR::ABL1* (IS), among patients with cytogenetic/molecular testing performed					
Patients who had cytogenetic/molecular testing performed (denominator)	39 (100.0)	20 (100.0)	19 (100.0)	NC	
3 months post initiation, n (%)					
Optimal	28 (71.8)	14 (70.0)	14 (73.7)	Fisher's Exact	1.0000
Warning	1 (2.6)	1 (5.0)	0		
Failure	1 (2.6)	1 (5.0)	0		
Response not reported	9 (23.1)	4 (20.0)	5 (26.3)		

Table V. *Continued*

Table V. *Continued*

		Mutational testing performed at/before second TKI initiation			
	All patients (N=51)	Yes (n=26)	No (n=25)	Test	*p*-Value
6 months post initiation, n (%)					
Optimal	31 (79.5)	15 (75.0)	16 (84.2)	Fisher's Exact	0.8430
Warning	4 (10.3)	3 (15.0)	1 (5.3)		
Failure	0	0	0		
Response not reported	4 (10.3)	2 (10.0)	2 (10.5)		
12 months post initiation, n (%)					
Optimal	26 (66.7)	15 (75.0)	11 (57.9)	Fisher's Exact	0.3896
Warning	0	0	0		
Failure	1 (2.6)	0	1 (5.3)		
Response not reported	12 (30.8)	5 (25.0)	7 (36.8)		

*Not mutually exclusive. 1L, First-line treatment; 2L, second-line treatment; 3L, third-line treatment; CML, chronic myeloid leukemia; CP-CML, chronic-phase chronic myeloid leukemia; FISH, fluorescence *in situ* hybridization; LOT, line of therapy; NC, not calculated; NGS, next-generation sequencing; PCR, polymerase chain reaction; TKI, tyrosine kinase inhibitor.

on their reasons for non-participation, including whether workload may have been a factor. To support the time and effort required, participating HCPs were compensated at fair market value for their contributions. While self-selection introduces the possibility of bias, participating HCPs represented a diverse range of practice settings and geographic regions, which may help to mitigate concerns and support the representativeness of the sample. Given its naturalistic, observational nature, this study captured real-world practices of its participant HCPs; for example, it is not known whether all laboratories that performed *BCR::ABL1* mutational profiling were IS-standardized, and there was no provision for testing at a single central facility to standardize the data. On the other hand, it is known that such profiling (*e.g.*, by qPCR, Sanger sequencing, NGS) was targeted to the *BCR::ABL1* kinase domain only.

No patient progressed to blast-phase disease, and relatively few individuals were treated with ponatinib or asciminib. Data on true clinical treatment resistance and other clinical outcomes, including cytogenetic and hematologic responses as well as progression-free and overall survival (as a function of testing *vs.* not testing), were not available in this analysis, although there were chart data on warning and failure signs on molecular testing, and HCPs could report resistance as a reason for treatment discontinuation and mutational testing. The purpose of this study was to evaluate HCP behaviors related to mutational testing; as an observational study, it is less informative about patient-level characteristics, including gender and baseline genotype, such as proportions of patients with kinase domain mutations in general and T315I genetic aberrations in particular. The frequency of mutational profiling at baseline was 17.6%, although this is not currently recommended by consensus guidelines (10-13). The finding that splenomegaly was significantly more frequent in patients who underwent mutational testing is based on unadjusted association that may merely reflect higher perceived disease burden or other correlated factors. This is an observational association and not necessarily causal in nature; splenomegaly may constitute a marker of provider concern and not an independent driver of *BCR::ABL1* mutational profiling. This chart review included only patients with CP-CML [although a recent report suggested increasing detection of BCR::ABL1 p190 transcripts in adults with acute myeloid leukemia (29)] and treatment with TKIs and not other approved CML treatments (*e.g.*, hydroxyurea, busulfan, cytarabine, hypomethylating agents, and omacetaxine mepesuccinate) or investigational agents (ELVN-001, PF-114, TERN-701) or treatment adjuncts (30). Finally, although definitions of molecular response milestones have not changed, certain

terminology has been altered in the recently published 2025 ELN recommendations (10). The respective terms "optimal", "warning", and "failure" in the 2020 guidelines have been replaced by "favorable", "warning", and "unfavorable" in the 2025 iteration.

Conclusion

Consensus-guided *BCR::ABL1* mutational profiling informs clinical decisions in the event of TKI failure, enabling practitioners to tailor treatments to patient genotypes and avoid subsequent resistance. This observational, real-world study suggests that US community healthcare provider testing behaviors may warrant closer attention. These findings should be further evaluated in prospective studies involving larger, more diverse prescriber and patient populations including greater numbers of individuals with CP-CML and suboptimal TKI treatment responses.

Supplementary Material

The supplementary data are available at: https://doi.org/10.5281/zenodo.17713005.

Data Availability

Data may be shared after approval of a proposal by an Ascentage Pharma Group International independent review committee. Sharing is subject to a confidentiality agreement and intellectual property review. Data will be available 9 to 36 months after publication. To request data relating to this study, please contact the corresponding author.

Conflicts of Interest

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Authors' Contributions

All Authors played a role in conceiving the study, analyzing the data, drafting, reviewing, revising, and/or approving the manuscript; and the decision to submit it for publication. All Authors had access to all data and full autonomy over all decisions concerning content. Drs. Danielle Gentile and Bryce Van Doren, as well as Ms Sarah Gordon, JaLyna Laney, and Karthika Jothivel, with Cardinal Health Specialty Solutions, made contributions to data acquisition and analysis, as well as study administration and oversight.

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Artificial Intelligence (AI) Disclosure

No artificial intelligence (AI) tools, including large language models or machine learning software, were used in the preparation, analysis, and presentation of this manuscript.

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