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Case Report

A real- world experience with asciminib in elderly CML-CP patients intolerant to previous

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ABSTRACT

Tyrosine kinase inhibitors (TKIs) have transformed the management of chronic myeloid leukemia in chronic phase (CML-CP). However, long-term therapy is often limited by intolerance, adverse events, and quality of life (QOL) concerns. Asciminib, is the 1st and only approved BCR. ABL1 inhibitor that works by STAMP (Specifically Targeting the ABL Myristoyl Pocket), has demonstrated efficacy and tolerability in patients previously treated with ≥2 TKIs. We report two elderly CML-CP patients with intolerance to multiple TKIs who achieved sustained responses with asciminib. A 60-year-old woman with CML-CP, diabetes, hypertension, and CKD. After intolerance to Dasatinib and Nilotinib (due to recurrent pleural effusion), she was switched to Asciminib in March 2025. Since then, her leukemia remains under complete molecular control, and her overall condition is stable with manageable comorbidities. A 77-year-old male with CML (chronic phase, November 2022) presented with weight loss, anorexia, and fatigue. Initial TKIs (Nilotinib, Dasatinib, Imatinib) led to cytopenias/intolerance, but since June 2024 on Asciminib 80 mg OD, he has achieved deep molecular response (BCR-ABL 0.139% as of July 2025) and remains clinically stable with manageable cytopenias. These real-world cases highlight the clinical utility of asciminib in elderly, comorbid CML-CP patients who are intolerant to the previous TKIs. Asciminib provided durable molecular responses and superior tolerability, consistent with trial data, and represents a valuable therapeutic option in this challenging patient population.

Keywords: Chronic myeloid leukemia, Chronic phase, Asciminib, Tyrosine kinase inhibitor intolerance, Elderly patients, Comorbidities, Real-world evidence, Case report

INTRODUCTION

Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm defined by the presence of the Philadelphia chromosome, which generates the BCR-ABL1 fusion oncoprotein with constitutive tyrosine kinase activity. This abnormal signaling drives uncontrolled myeloid proliferation and remains the principal therapeutic target in CML-CP.[1,2]

The introduction of tyrosine kinase inhibitors (TKIs) targeting the ATP-binding site of BCR-ABL1 has transformed CML into a largely manageable chronic disease, with many patients now achieving near-normal life expectancy.2 However, a proportion of patients either experience treatment failure and require multiple lines of therapy due to resistance or intolerance to therapy.[3] Asciminib is a first-in-class BCR-ABL1 inhibitor that Specifically Targets the ABL1 Myristoyl Pocket (STAMP works by STAMP (Specifically Targeting the ABL Myristoyl Pocket). It is widely approved for the treatment of adult patients with CML in chronic phase (CP) who are either resistant or intolerant to ≥ 2 prior TKIs or those who harbor the gatekeeper T315I mutation.[4,5] The pivotal phase III ASCEMBL trial demonstrated that asciminib was superior to bosutinib in patients with CML-CP who had

received ≥2 prior TKIs, achieving significantly higher rates of major molecular response (MMR) at week 24 with fewer discontinuations due to adverse events.[6]

The long-term results from ASCEMBL support asciminib as a suitable treatment option for patients with resistance to or intolerance of previously treated TKIs. With the EOS (End of Study) Update- with nearly 4 years of long term data from ASCEMBL, Asciminib demonstrated superior efficacy, safety, tolerability and improved HRQOL (Health Related Quality of Life) over bosutinib. At week 156, the major molecular response (MMR) rates remained higher 33.8% vs 10.5% for Asciminib Vs Bosutinib. The drug continued to cause fewer grade ≥3 adverse events (59.6% vs 68.4%) and fewer AEs leading to treatment discontinuation (8.3% vs 27.6%) as compared to bosutinib.[7]

Despite robust trial evidence, there is limited real-world literature describing the practical role of asciminib especially in elderly CML-CP patients with multiple comorbidities and intolerance to standard TKIs. We present two such cases—an elderly male and an elderly female—who achieved sustained molecular responses and improved tolerability with asciminib, highlighting its clinical utility in challenging real-world settings.

CASE REPORT

Case 1

A 60-year-old female presented in April 2023 with generalized weakness, easy fatigability, and loss of appetite. Past medical history included diabetes mellitus, hypertension, chronic kidney disease (CKD), bilateral cataract, and diabetic retinopathy. On examination, she had pallor without hepatosplenomegaly. Initial labs revealed hemoglobin 8.4 g/dl, WBC 60.38 × 10^3/µl, platelets 129 × 10^9/l, and BCR-ABL1 transcript level of 16.86% (IS).

Treatment course

April 2023 – August 2024: Initiated on dasatinib 100 mg OD, achieving molecular response but developed recurrent pleural effusions requiring repeated drainage. Dose modifications were attempted, but pleural effusions persisted, leading to discontinuation.

September 2024 – December 2024: Switched to nilotinib; however, the patient continued to have unresolved pleural effusions and intolerance.

March 2025: Due to intractable intolerance, the patient was initiated on asciminib 80 mg OD. At baseline, BCR-ABL1 was undetectable.

April–June 2025: Continued asciminib with stable CBC, no recurrence of pleural effusion, stable CKD, and BCR-ABL1 negativity.

By July 2025, the patient was clinically stable, asymptomatic, and sustained molecular remission with good drug tolerability.

Table 1 provides detailed treatment journey and examination findings.

Table 1: Clinical characteristics and treatment course of the patient.

Date	Findings/ lab results	Treatment
14/04/2023	CBC: Hb 8.4 g/dl, WBC 60.38 ×10³/mm³, Platelets 1.29 ×10⁵/mm³ qPCR BCR-ABL: 16.8645% (baseline)	Start Dasatinib 100 mg OD
28/04/2023	CBC: Hb 8.4 g/dl, WBC 60.38 ×10³/mm³, Platelets 1.29 ×10⁵/mm³	Continue Dasatinib 100 mg OD. Start Entecavir 0.5 mg OD
29/05/2023	CBC: Hb 6.8 g/dl, WBC 6.78 ×10³/mm³, Platelets 1.30 ×10⁵/mm³	Continue Dasatinib 100 mg OD and Entecavir 0.5 mg OD. Monitor renal function and uric acid
31/05/2023	Hepatitis profile: Negative (HepB surface Ag, HPLC normal) CBC: Hb 7.2 g/dl, WBC 3.3 × 10³ /mm³, Platelets 1.23 ×10⁵/mm³	No evidence of nutritional anemia. Continue the same treatment
08/06/2023	Patient feels better, with less fatigue No splenomegaly. K/L ratio 72.4/18 (as per previous note)	Continue Dasatinib 100 mg OD, Entecavir 0.5 mg OD. Follow-up as per protocol
06/09/2023	Myeloma profile: Showed MGUS-like features, CrCL-16 ml/min. mixed pallor, Platelets: 211,000/µL. Hb improved from 7.4----8.4 g/dl qPCR BCR-ABL: 0.086%- Good MR	Anemia treated with EPO 10,000 units Patient started taking Dasatinib 100 mg alternate days on her own

Continued.

Date	Findings/ lab results	Treatment
28/11/2023	Platelets dropped- 42,000 BCR-ABL: Not detectable	Dose decreased to 50 mg
08/12/2023	CBC: Hb 8.6 / TLC 3150 / Platelets 105,000, ANC 1.48	On Dasatinib 50 mg
02/02/2024	Stable, asymptomatic CBC: Hb 8.6 / TLC 3110 / Platelets 91,000, ANC 1.74 BCR-ABL: Not detectable	Still on Dasatinib 50 mg
11/03/2024	Follow up: BCR-ABL: Not detectable	Dose increased Dasatinib to 70mg
09/04/2024	CBC: Hb 5.9 / Platelets 40,000, ANC 1.61	Dasatinib 70 mg, SC 2.43, TB 0.24
14/07/2024	CBC: Hb 9 / TLC 5700 / Platelets 183,000 Stable counts, asymptomatic	Continued Dasatinib 70 mg
07/08/2024	Admitted with breathlessness, AKI on CKD, pleural effusion Massive bilateral pleural effusion → ICD + tapping Recent CXR → mild residual effusion BCR-ABL (September 2024): → Negative Exam: pallor+, pedal edema+, basal crepts Complaints: weakness, DOE	Dasatinib stopped, switched to Nilotinib
16/09/2024	Still in MMR (BCR-ABL negative as of September 2024) Clinically: weakness, DOE Monitoring for effusion resolution	Follow-up on Nilotinib
28/12/2024	Still having pleural effusion (left-sided)	Continue Nilotinib until Asciminib is available
13/03/2025	BCR-ABL: Not detectable HbA1C: 6.2%, FBC: WBC: 9.3, Hb 9.4, Platelets 193,000	Due to Dasatinib-induced intractable pleural effusion and persistence despite nilotinib, Started Asciminib 80mg OD
21/04/2025	CBC: Hb 9.1, WBC 4340, Platelets 154,000 BCR- ABL: Not detectable	Continue Asciminib
17/06/2025	BCR -ABL: Not detectable	Continue Asciminib

Despite achieving significant molecular responses on both dasatinib and nilotinib, intolerance remained the major limiting factor in this patient's treatment journey. On dasatinib, the patient developed pleural effusions, a well-documented adverse event associated with long-term use, which significantly impacted quality of life and necessitated drug discontinuation. Similarly, nilotinib, although effective in achieving undetectable BCR-ABL1 levels, was not tolerated due to treatment-related adverse effects. These episodes highlight that intolerance can pose as great a challenge as resistance in clinical practice, particularly in patients who might otherwise achieve deep and durable molecular responses. Thus, management strategies must not only aim for efficacy but also prioritize tolerability and patient comfort to ensure sustained adherence and long-term outcomes. Figure 1 below shows the trends in BCR- ABL Levels during TKI therapy.

Current status (as of July 2025)

On Asciminib 80 mg OD.

BCR-ABL 0.139% (deep molecular response, MMR+)

No major dose interruptions have been reported since starting Asciminib.

Clinically stable, with manageable cytopenia.

Conclusion: This case highlights the clinical challenge of managing an elderly CML-CP patient with intolerance to multiple TKIs (Nilotinib, Dasatinib, Imatinib). Despite suboptimal tolerance and repeated treatment interruptions with earlier TKIs, the patient achieved sustained molecular response and improved tolerability on Asciminib.

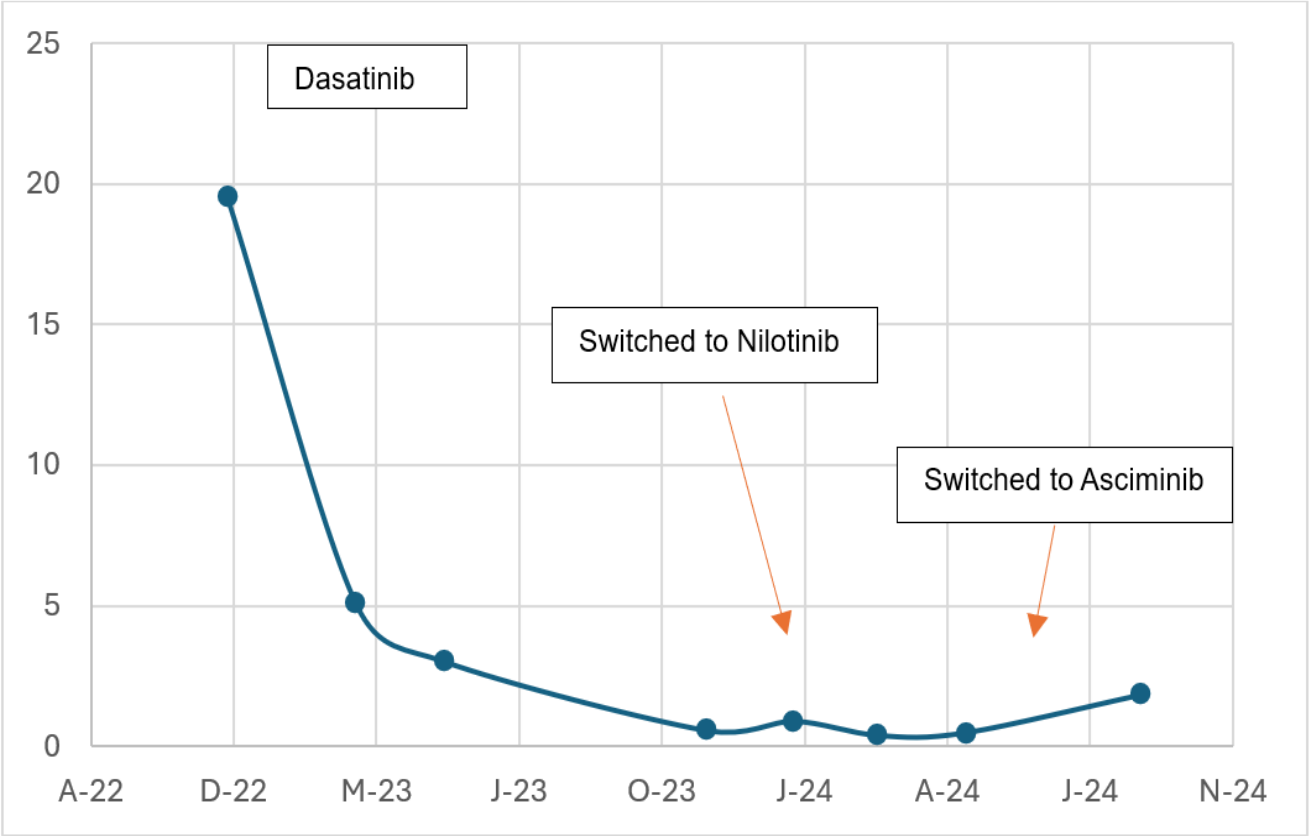

Figure 1: BCR -ABL levels during the treatment journey.

X axis: M-Year format, Y Axis: BCR ABL levels (%).

Case 2

A 77-year-old male presented in November 2022 with one month of weight loss, anorexia, weakness, and fatigue. Examination revealed pallor and splenomegaly (spleen palpable 4–5 cm). There was no history of diabetes, hypertension, ischemic heart disease, or bleeding diathesis.

Baseline investigations: qPCR BCR-ABL (December 2022): 19.548% (International Scale)

Hemogram: anemia, leukocytosis, thrombocytopenia (details as per timeline)

Echocardiogram: normal.

Treatment timeline: Nilotinib 300 mg BID (December 2022 – April 2023): Stopped due to severe thrombocytopenia (platelets 13,000/µL). BCR-ABL reduced to 3.03% but therapy discontinued.

Dasatinib 50–100 mg (May 2023 – October 2023): Multiple interruptions for cytopenias and intolerance (fatigue, nocturia, backache). BCR-ABL: 4.03% (August 2023) → 0.56% (November 2023).

Imatinib 400 mg (October 2023 – June 2024): Initially tolerated, but recurrent cytopenias and fluctuating molecular response (BCR-ABL: 0.90% January 2024 → 0.39% March 2024 → 0.49% June 2024).

Asciminib 40–80 mg daily (June 2024 – ongoing): Well tolerated, Achieved progressive molecular reduction (BCR-ABL: 0.496% August 2024 → 0.248% December 2024 → 0.721% March 2025 → 0.139% July 2025).

The patient continues asciminib with good tolerability and improved performance status. Table 2 below shows the lab findings and treatment till date.

Although the patient demonstrated molecular responses with nilotinib, dasatinib, and imatinib, treatment intolerance and recurrent cytopenias were the predominant limiting factors. Nilotinib was discontinued due to severe thrombocytopenia, dasatinib required multiple interruptions due to fatigue, nocturia, and cytopenias, and imatinib was complicated by fluctuating cytopenias and unstable molecular response. These cumulative adverse effects significantly impacted the patient's quality of life and continuity of therapy, ultimately necessitating the switch to asciminib. Figure 2 shows the trends in BCR-ABL Levels during TKI therapy.

Table 2: Clinical characteristics and treatment course of the patient.

Date	Findings/ lab results	Treatment
27/12/2022	CML-CP confirmed, Spleen palpable (4–5 cm), HbA1c 5.6% (normal) 2D ECHO done (normal heart) - Few VPCs (heart rhythm changes) but asymptomatic Baseline qPCR BCR-ABL: 19.548%	Tasigna (Nilotinib) 300 mg twice daily since 02/12/22 17/12- Patient not taking Tasigna for last 4-5 days
03/02/2023	CBC- Hb 11.1, WBC 3.45, Platelets 13,000	Tasigna induced thrombocytopenia Plan: Stop Tasigna and give supportive care
13/03/2023	CBC: Hb 12.3, WBC 4.56, Platelets 299,000, ANC 2.64, LFT normal; TB 0.52	Restart Tasigna 300 mg once daily at 11:00 am (empty stomach, no food/drink 2h before/after)
27/03/2023	CBC: Hb 11.5, WBC 6.35, Platelets 149,000, Neutrophils 74%, Lymphs 20%, Eos 1%, Baso 1%, ECG QTc 429 ms; qPCR (22-March-23) BCR-ABL: 5.074%	Reached CHR after step up dose–450mg/day Continue Tasigna 300 mg OD
29/04/2023	Hb 11.9, TLC 4.03, Platelets 55,000, ANC 2.18, N54 L44 M1 E1	Stop Tasigna. Plan to switch to Dasatinib
13/05/2023	Hb 12.3, TLC 2.98, Platelets 55,000, N33 L60 M6 E1 BCR- ABL: 3.0318%	Admit for 1 day. Plan: BMA + BMBx + Flow + FISH + QPCR for BCR-ABL + NGS for myeloid malignancy
01/06/2023	No abnormal cells. QPCR: 5.074 (22/03/23) → 3.0318 (23/05/23). CBC (01/06/23): Hb11.1, TLC 5.29, Platelets 183,000, ANC 3.23	Dasatinib started 50 mg (24/05/2023). Increased to 100 mg (02/06/2023)
05/08/2023	Complaints: loss of appetite, generalized weakness (1 month), nocturia disturbing sleep	Started on Dasatinib 50 mg since 24/05/2023 and again cytopenic and on last visit on 01/07/2023 dose reduced to 50mg from 100mg
07/10/2023	QPCR BCR-ABL: (02/08/2023): 4.0305% Occasional backache - No pallor / splenomegaly - BP 110/70Hb 11.7, TLC 4.60, Platelets 1,95,000 N 55%, L 38%, M 4%, E 3%	Multiple dose interruptions - Intolerance to Tasigna & Dasatinib - Started Veenat 400 mg (from 14/10/2023)
11/11/2023	Hb 10.7, TLC 2.17, Platelets 62,000 N 34%, L 61%, M 2%, E 2% QPCR for BCR-ABL: 0.5694%	Veenat 400 mg
20/01/2024	Hb 8.2, TLC 1.57, Platelets 45,000, N28 L67 M4 E1 ANC 440, SCr 0.92, BCR -ABL: 0.9007%	Continue Veenat
08/03/2024	CBC: 7.2 / 200 / 82,000 N52 L42 M6 E1, ANC 820 Fatigue, difficulty climbing stairs, pallor +, spleen not palpable, BP 120/70 BCR- ABL: 0.3988%, IRMA-Negative	on Veenat 400 mg; No dose interruptions after starting Veset

Continued.

Date	Findings/ lab results	Treatment
06/06/2024	6.9 / 2700 / 155; N40 L48 E6 M4 → Platelets recovered IRMA – Neg (22/05/2024); Last QPCR BCR-ABL: 0.4960% (18/05/2024)	Multiple dose interruptions due to cytopenia. Trials with Nilotinib/Dasatinib/Imatinib
03/08/2024	Get dizziness only after salty food- Continue treatment BP: 110/70 No pallor Observation OK . Last QPCR BCR-ABL: 0.4960%	On Asciminib 40 mg since 22/06/2024
31/08/2024	Complaints of dizziness, not definitive	Started Asciminib 40mg (22/06/2024)- ↑ dose to 80mg daily (04/08/2024)
28/09/2024	BCR- ABL: 1.84% (Decreasing trend)	Continuing Asciminib 80 mg OD
18/12/2024	BCR- ABL: 0.248%	Continue asciminib
07/03/2025	CBC: 10/2.66/75,000 BCR-ABL: 0.721%	Continue asciminib
21/07/2025	BCR -ABL: 0.139%	Continue asciminib

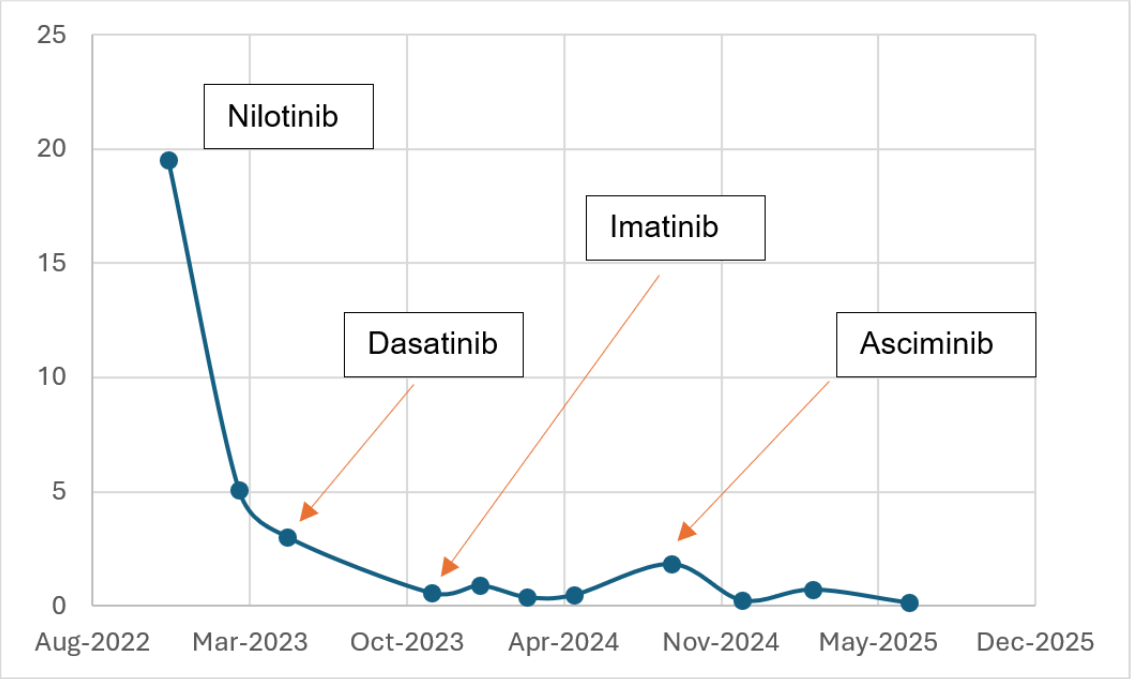

Figure 2: Shows the trends in BCR ABL Levels during TKI therapy

This case emphasizes the importance of individualized therapy in elderly CML patients with TKI intolerance. Asciminib represents a valuable treatment option, providing effective molecular response with good tolerability even after multiple TKI failures.

DISCUSSION

Tyrosine kinase inhibitors (TKIs) remain the backbone of therapy for chronic myeloid leukemia in chronic phase (CML-CP). First- and second-generation TKIs such as imatinib, dasatinib, and nilotinib have markedly improved survival outcomes in Chronic phase similar to that of age-matched control.[8] However, despite their efficacy, longterm tolerability remains a challenge, especially in patients with advanced age, frailty, or significant comorbidities.

Adverse effects such as dasatinib-related pleural effusion, nilotinib-associated vascular events, and metabolic

derangements can result in repeated dose interruptions, discontinuations, and impaired quality of life.[9]

The first case presented highlights a 60-year-old female with diabetes, hypertension, CKD, and diabetic retinopathy who developed recurrent dasatinib-induced pleural effusions and subsequently intolerance to nilotinib. Pleural effusion is among the most frequent adverse events of dasatinib, occurring in up to 28% of patients, and is more common in elderly individuals and those with pre-existing comorbidities.[10] Nilotinib, though devoid of pleural effusion risk, carries a well-documented association with vascular and metabolic toxicities, rendering it unsuitable for long-term use in patients with cardiovascular risk factors or metabolic syndrome.[11]

The second case involves an elderly Male with intolerance to prior TKIs, further underscoring the clinical dilemma in this subgroup. Elderly CML patients often pose unique management challenges—balancing disease control with preservation of functional status and minimizing drug-related morbidity. In such cases, newer therapeutic options with improved safety profiles are urgently needed.

Asciminib, a first-in-class STAMP inhibitor, represents a paradigm shift in CML therapy. Unlike ATP-competitive TKIs, it targets the myristoyl pocket of ABL1, allowing for selective inhibition with reduced off-target effects.[4] In the phase III ASCEMBL trial, asciminib demonstrated superior efficacy compared with bosutinib in patients with prior TKI exposure, while maintaining a favorable safety profile with lower rates of vascular and fluid retention events.[5-7] Importantly, its tolerability in comorbid populations has been increasingly recognized in real-world reports.[12]

In both of our cases, asciminib provided a durable molecular response, excellent tolerability, and stabilization of comorbid conditions. Neither patient experienced recurrence of pleural effusion, vascular events, or metabolic derangements during follow-up. This suggests that asciminib may be particularly beneficial in patients where toxicity profiles of conventional TKIs limit therapeutic choices.

Although in the case mentioned, the patient achieved an excellent molecular response with a reduction in BCR-ABL1 transcripts from 16.86% at baseline to 0.086% on dasatinib, and subsequently reached undetectable levels on nilotinib, treatment intolerance emerged as the major limiting factor. Both dasatinib-related pleural effusions and nilotinib-associated adverse events necessitated treatment discontinuation, despite the depth of molecular response. This scenario underscores a key challenge in CML management—balancing efficacy with tolerability, particularly in patients with comorbidities.

Unmet needs in CML therapy extend beyond achieving deep molecular responses; intolerance and quality of life (QoL) remain critical determinants of long-term treatment success. For physicians, efficacy is often the primary goal, but for patients, side effects and daily QoL disruptions may weigh more heavily in their overall treatment journey. This highlights the importance of shared decision-making between physician and patient to ensure treatment strategies are individualized.[13] The switch to asciminib in this case provided a balance of efficacy and improved tolerability, demonstrating its potential to address both clinical and patient-centered unmet needs.

These cases reinforce the emerging role of asciminib as an effective and safer alternative in the management of TKI-intolerant CML-CP patients, particularly those with high comorbidity burden. Further real-world evidence and longer-term follow-up are warranted to confirm its role in optimizing treatment outcomes in this challenging subgroup.

Limitations

While our experience is limited to two patients, these cases add to the growing real-world evidence supporting asciminib's role in clinical practice. Real-world studies are crucial, as trial populations often under-represent elderly patients and those with significant comorbidities. The ability of asciminib to provide durable molecular responses with improved tolerability in such challenging patients suggests that its use could be expanded earlier in the treatment sequence, especially for those with intolerance rather than resistance.

CONCLUSION

These two cases underscore the clinical utility of asciminib in elderly, comorbid patients with CML-CP who are intolerant to multiple TKIs. By providing sustained molecular response and enhanced tolerability, asciminib aligns with clinical trial evidence while addressing an important real-world gap. Future studies should further explore its role in frontline settings and in optimizing treatment goals for patients at high risk of TKI intolerance.

Thus, Asciminib emerges as an effective and well-tolerated option in real-world practice for patients with CML-CP who are resistant or intolerant to multiple prior TKIs, including elderly and comorbid patients.

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