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Navigating Treatment Challenges in Metastatic Gastric Gastrointestinal Stromal Tumor (GIST): A Case Report of Sequential Four-Line Targeted Therapy and Palliative Transition in a Resource-Limited Setting

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Zahraa A. Mohammed [1], Anmar Z. Alharganee [1, 2], Tabarek M. Obaid [1], Alhussein Ashraf Kadhim [1]

1. Department of Medical Oncology, Baghdad Medical City, Baghdad, IRQ 2. Iraqi Cancer Council, Ministry of Health, Baghdad, IRQ

Corresponding author: Anmar Z. Alharganee, aalharganee@gmail.com

Abstract

Gastrointestinal stromal tumors (GISTs) represent the primary mesenchymal neoplasms of the gastrointestinal tract and are driven predominantly by oncogenic gain-of-function mutations in KIT or platelet-derived growth factor receptor alpha. Although small-molecule tyrosine kinase inhibitors (TKIs) have transformed the management of advanced disease, administering multiple lines of systemic therapy, managing secondary drug resistance, controlling toxicities, and navigating drug supply chain disruptions in low- and middle-income countries present complex clinical challenges. Here, we present the case of a 55-year-old man who presented with a large primary gastric fundic GIST and synchronous bilobar liver metastases. Diagnostic core needle biopsy confirmed a spindle cell GIST demonstrating positivity for CD117 and CD34, a Ki-67 proliferation index of approximately 2%, and negativity for pan-cytokeratin. Over a three-year clinical course, the patient received four sequential lines of TKI therapy: frontline imatinib mesylate, 400 mg orally daily; second-line sunitinib malate, 37.5 mg orally daily on a modified two-week on and two-week off schedule. Sunitinib treatment was associated with a reduction in the reported tumor diameter from 13.3 cm to 7.6 cm (approximately 42.9%), based on the available imaging measurements; third-line regorafenib, 160 mg orally daily, administered three weeks on and one week off, which was complicated by severe drug-induced hypothyroidism, with a thyroid-stimulating hormone level of 25.34 mIU/L, for which levothyroxine replacement was initiated; and fourth-line ripretinib, 150 mg orally daily, following progression during regorafenib treatment. Subsequent radiological disease progression, worsening anemia, lower extremity edema secondary to hypoalbuminemia, drug supply interruptions, and a lack of clinical benefit after the third cycle of ripretinib led to a timely transition to best supportive palliative care. Multiline management of GISTs in resource-limited settings requires serial cross-sectional CT monitoring, individualized TKI treatment modifications in response to toxicity, and a timely transition to palliative care when further treatment provides no meaningful clinical benefit.

Categories: Oncology, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: choi criteria, gastrointestinal stromal tumor (gist), imatinib resistance, palliative care, sunitinib, tyrosine kinase inhibitors (tkis)

Introduction

Gastrointestinal stromal tumors (GISTs) originate from the interstitial cells of Cajal or their committed precursors within the muscularis propria of the gastrointestinal tract, accounting for 1-2% of gastrointestinal malignancies [1,2]. Approximately 75-80% of GISTs harbor activating mutations in the KIT proto-oncogene, 10-15% harbor mutations in platelet-derived growth factor receptor alpha (PDGFRA), and 10-15% lack mutations in either gene and are classified as wild-type disease [3,4]. The introduction of small-molecule tyrosine kinase inhibitors (TKIs) fundamentally altered the natural history of metastatic GISTs, enabling prolonged disease control in some patients [5].

Frontline imatinib mesylate provides durable clinical disease control in most patients, achieving a median overall survival exceeding 50 months in landmark trials such as the Southwest Oncology Group (SWOG) S0033 study [5,6]. However, resistance to TKIs commonly develops during treatment. One established mechanism involves the emergence of secondary mutations in the KIT gene, which may alter drug sensitivity and contribute to disease progression. These mechanisms are described in the literature but were not molecularly confirmed in the present patient [7,8]. Standard global clinical practice guidelines, including those from the National Comprehensive Cancer Network and the European Society for Medical Oncology, recommend sequential use of approved TKI therapies: frontline imatinib, second-line sunitinib, third-line regorafenib, and fourth-line ripretinib [8-11].

In low- and middle-income countries and resource-limited health care settings, administering sequential

TKI therapy involves distinct operational and clinical obstacles [3,12]. These challenges include the lack of access to, or prohibitive cost of, routine next-generation sequencing (NGS) or circulating tumor DNA mutational profiling to guide the selection of salvage targeted agents, such as avapritinib for PDGFRA exon 18 D842V mutations [12-14]. Furthermore, clinical management is hindered by cumulative drug-induced toxicities, including thyroid dysfunction, hand-foot skin reactions, hypertension, fatigue, and cytopenias, as well as financial barriers and frequent drug supply chain disruptions [7,9,10]. These systemic challenges often necessitate pragmatic modifications to dosing schedules. In selected patients, alternative sunitinib dosing schedules may be considered to improve tolerability, although the choice of schedule should be individualized. Examples include sunitinib administered two weeks on and one week off or two weeks on and two weeks off [7,8,15].

Additionally, clinicians in resource-constrained environments rely heavily on serial cross-sectional CT to monitor treatment response and identify radiological disease progression. However, limited access to advanced imaging analyses and molecular testing may complicate the assessment of secondary drug resistance and decisions regarding the transition to supportive palliative care [9,11,16,17].

To illustrate these real-world clinical and operational challenges, we present the case of a 55-year-old man with metastatic gastric GIST who received four sequential lines of targeted TKI therapy in a resource-limited setting. This case highlights the challenges associated with treatment-related toxicity, disease progression, interruptions in drug availability, and limited access to molecular profiling. Although established molecular mechanisms of resistance are discussed in the literature, KIT and PDGFRA mutation testing was not performed in this patient; therefore, the specific molecular mechanism underlying disease progression could not be determined.

Case Presentation

A 55-year-old man with metastatic gastric GIST presented with a several-month history of postprandial fullness, vague epigastric pain, recurrent vomiting, melena, and involuntary weight loss. At baseline, the patient had an Eastern Cooperative Oncology Group (ECOG) performance status of 1. He had no personal medical history or family history of malignancy [1,2]. Physical examination demonstrated a palpable, nontender epigastric mass [1]. Esophagogastroduodenoscopy identified a large (7-8 cm) submucosal gastric mass with central ulceration and contact bleeding extending to the esophagogastric junction [1,2]. Histopathological examination of the core needle biopsy revealed a submucosal proliferation of bland spindle cells arranged in a syncytial pattern, with elongated nuclei and pale cytoplasm [3,4]. Subsequent immunohistochemical analysis confirmed a spindle cell GIST, demonstrating diffuse positivity for CD117 (c-KIT) and CD34, a Ki-67 proliferation index of approximately 2%, and negative staining for pan-cytokeratin [3,4]. The mitotic count was not reported in the available pathology description. Molecular testing for KIT and PDGFRA mutations was not performed because molecular profiling was unavailable in the treating setting.

Baseline staging contrast-enhanced CT (CECT) revealed a large exophytic gastric mass measuring 8.0 × 6.5 × 6.1 cm, with a mural thickness of up to 5.7 cm, abutting the left hemidiaphragm and spleen. The scan also demonstrated confluent bilobar hypodense liver metastases, with a segment II lesion measuring 7.6 × 6.8 cm, and upper abdominal lymphadenopathy. The initial CT report described the gastric mass as involving the fundus and cardia, whereas subsequent reports referred to the lesion as a gastric cardia mass. Additionally, multiple small bilateral pulmonary parenchymal and pleural-based nodules were reported, with the largest measuring approximately 4 mm in the right upper lobe. The baseline report described these nodules without definitively establishing their metastatic nature. No prior imaging was available for comparison (Figure *1*) [3,5].

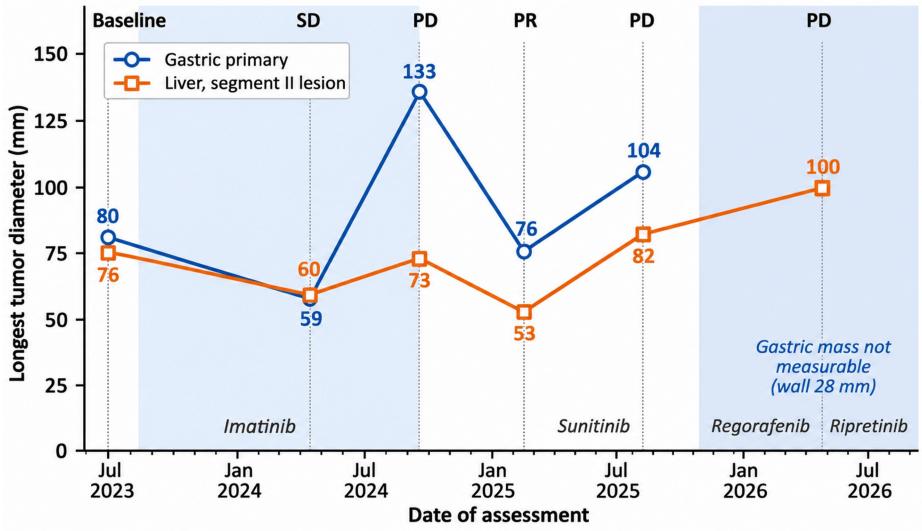


FIGURE 1: Tumor dynamics.

Longitudinal measurements of the longest tumor diameter (mm) for the primary gastric mass (blue line) and hepatic segment II metastasis (orange line) from baseline diagnosis (June 2023) through sequential TKI therapies, including imatinib, sunitinib, regorafenib, and ripretinib. Key radiological response milestones, including SD, PR, and PD, as evaluated by CECT imaging, are indicated alongside therapeutic switches.

CECT, contrast-enhanced CT; PD, progressive disease; PR, partial response; SD, stable disease; TKI, tyrosine kinase inhibitor

Over a three-year clinical course, the patient was managed sequentially with four lines of targeted TKI therapy (Figure 2, Tables 1, 2).

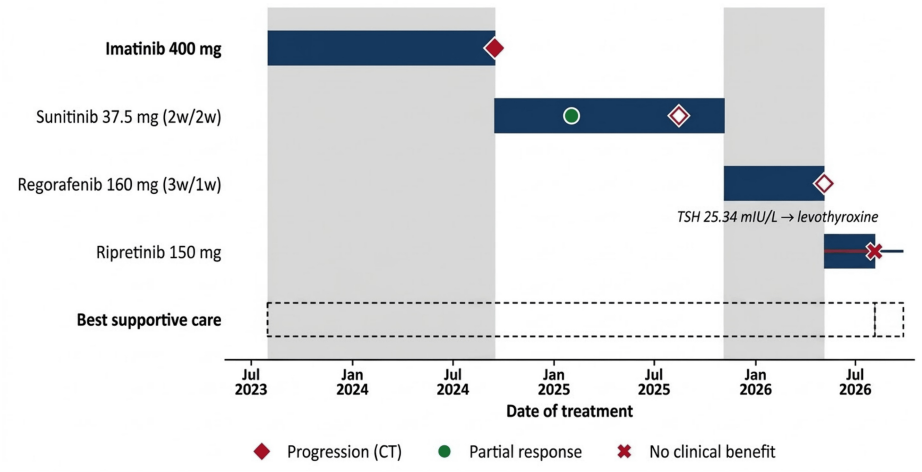


FIGURE 2: Sequential treatment protocol timeline.

Schematic timeline illustrating the sequential systemic treatment regimen across four lines of targeted TKI therapy: frontline imatinib (400 mg daily), second-line sunitinib (37.5 mg daily on a modified two-week on and two-week off schedule because of toxicity), third-line regorafenib (160 mg daily on a three-week on and one-week off schedule), and fourth-line ripretinib (150 mg daily), followed by a transition to best supportive palliative care. Key clinical and biochemical monitoring parameters, including TKI-induced hypothyroidism treated with levothyroxine, are annotated along the treatment continuum.

TKI, tyrosine kinase inhibitor

Line of therapy	Drug and schedule	Primary target domain (general molecular mechanisms)	Major toxicities and management	Response and clinical outcome	Citations
First line	Imatinib 400 mg PO daily	Primary KIT exon 11/exon 9	Grade 1 eyelid edema and cramps; normal TSH	PFS: 13 months; initial disease control followed by progression	[5,6]
Second line	Sunitinib 37.5 mg PO daily (two weeks on/two weeks off)	Secondary ATP-binding pocket (KIT exons 13/14)	Grade 2 hand-foot skin reaction and hypertension	PFS: 11 months; partial response (42.9% reduction in size)	[7,8]
Third line	Regorafenib 160 mg PO daily (three weeks on/one week off)	Secondary activation loop (KIT exons 17/18)	Hypothyroidism (TSH = 25.34); levothyroxine initiated	PFS: nine months; progression	[9,10]
Fourth line	Ripretinib 150 mg PO daily	Broad switch-pocket inhibitor (KIT/PDGFRA)	Anemia, hypoalbuminemic edema, and drug supply interruptions	PFS: approximately three months; no benefit followed by transition to palliative care	[8,11]

TABLE 1: Multiline systemic TKI treatment sequence summary.

Citations in this table reference the pivotal clinical trials, clinical practice guidelines, and dosing evidence that establish the therapeutic rationale and toxicity management strategies applied across each sequential line of treatment in this patient. The exon profiles listed represent general drug-target and resistance mechanisms reported in the literature, not mutations detected in this patient. KIT/PDGFRA molecular testing was not performed.

ATP, adenosine triphosphate; PDGFRA, platelet-derived growth factor receptor alpha; PFS, progression-free survival; TKI, tyrosine kinase inhibitor; TSH, thyroid-stimulating hormone

Date range	Systemic TKI line	Gastric primary mass	Hepatic metastatic burden	Extrahepatic status	Overall response status	Citations
June 2023	Pre-TKI biopsy	Submucosal spindle cell GIST	-	-	Confirmed GIST (CD117+, CD34+)	[3,4]
June 2023	Baseline staging	8.0 × 6.5 × 6.1 cm (mural thickness, 5.7 cm)	Largest segment II lesion: 7.6 × 6.8 cm	Upper abdominal lymph nodes	Baseline staging	[3,5]
August 2023-August 2024	First line: imatinib	Wall thickness: 2.4 cm; mass: 4.9 × 5.9 cm	Largest segment II lesion: 5.6 × 6.0 cm	Lung nodules resolved	SD	[5,6]
September 2024	First-line failure	Enlarged: 13.3 × 9.3 × 9.5 cm	Enlarged: segment II, 7.3 × 5.8 cm	Para-aortic lymph nodes; ascites	PD	[7,8]
September 2024-August 2025	Second line: sunitinib	Regressed: 7.6 × 7.2 cm (1.4 cm)	Regressed: segment II, 5.3 × 4.1 cm	Lymph node resolution; HFSR	PR, followed by PD	[7,8]
August 2025-May 2026	Third line: regorafenib	Wall thickness: 2.8 cm (>15% increase in enhancement)	Enlarged: segment II, 10.0 × 7.5 cm	TSH elevation (25.34)	PD	[9,10]
June 2026	Fourth line: ripretinib	Advanced gastric involvement	Multifocal liver lesions	Anemia; leg edema	No benefit; transition to palliative care	[8,11]

TABLE 2: Longitudinal master summary of imaging, tumor burden, and radiological evaluation.

Citations in this table correspond to benchmark clinical trials, imaging classification standards, and evidence-based guidelines used to evaluate radiological tumor burden and define disease control or progression at each treatment milestone.

GIST, gastrointestinal stromal tumor; HFSR, hand-foot skin reaction; PD, progressive disease; PR, partial response; SD, stable disease; TKI, tyrosine kinase inhibitor; TSH, thyroid-stimulating hormone

Frontline therapy with imatinib mesylate 400 mg orally once daily was initiated in August 2023, achieving good clinical disease control, with body weight maintained between 88 and 93 kg [5,6]. CECT in April 2024

confirmed stable disease, with complete resolution of the bilateral pulmonary parenchymal nodules and stabilization of gastric mural thickness at 2.4 cm. Hepatic metastases also remained stable, with the segment II lesion measuring 5.6 × 6.0 cm [5,6]. Serial laboratory evaluations showed that thyroid-stimulating hormone (TSH) levels remained within normal limits (1.38-1.92 mIU/L).

In September 2024, follow-up CECT revealed overt disease progression, with the primary gastric mass expanding to 133 × 93 × 95 mm, the segment II hepatic lesion expanding to 73 × 58 × 42 mm, the emergence of new para-aortic lymphadenopathy measuring 10 × 7 mm, and mild ascites (Figure *1*) [7,8]. Second-line sunitinib malate 37.5 mg orally daily was initiated on a modified schedule of two weeks on and two weeks off to mitigate grade 2 hand-foot skin reactions and fatigue [7,8]. Reassessment with CECT demonstrated a marked partial response, with regression of the primary gastric mass to 76 × 72 × 68 mm and reduction of the main liver mass to 53 × 41 × 34 mm, along with resolution of retroperitoneal lymphadenopathy (Table *2*) [7,8].

The patient achieved a prolonged period of disease control during this treatment line (Figure *3*). By August 2025, CECT confirmed secondary resistance to sunitinib, showing reexpansion of the primary gastric mass to 98 × 78 × 104 mm, enlargement of hepatic lesions, and new omental metastatic deposits [9,10]. The patient was transitioned to third-line regorafenib 160 mg orally once daily, administered three weeks on and one week off [9,10]. During regorafenib treatment, laboratory testing revealed an elevated TSH level of 25.34 mIU/L. Levothyroxine was initiated at 50 µg daily and subsequently increased to 75 µg daily. Regorafenib treatment was continued during thyroid hormone replacement. Subsequent testing showed a decrease in TSH to 2.15 mIU/L following levothyroxine treatment. Free thyroxine results and detailed documentation of thyroid-related symptoms were unavailable in the reviewed records; therefore, the clinical severity of the thyroid dysfunction could not be fully characterized [10].

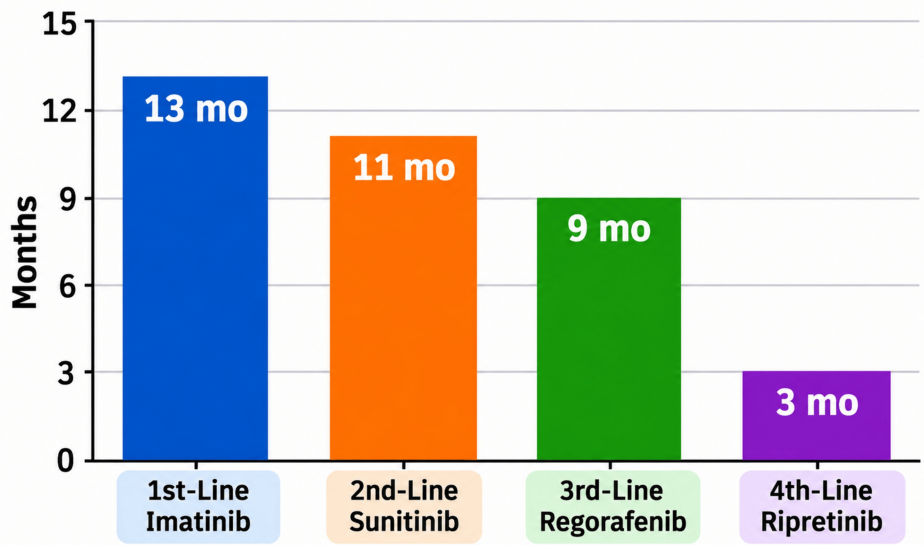


FIGURE 3: PFS.

Bar chart illustrating the PFS intervals achieved across sequential TKI regimens in a patient with metastatic gastric GIST: first-line imatinib (13 months), second-line sunitinib (11 months), third-line regorafenib (nine months), and fourth-line ripretinib (three months).

GIST, gastrointestinal stromal tumor; PFS, progression-free survival; TKI, tyrosine kinase inhibitor

CECT performed in May 2026 was reported as showing progressive disease according to Choi criteria. Gastric wall thickness remained approximately stable at 28 mm, despite a reported increase of more than 15% in post-contrast enhancement. Additional findings included enlargement of the largest hepatic lesion to 100 × 75 mm, new splenic lesions described as resulting from direct invasion by the adjacent gastric wall, and enlargement of the epigastric omental deposit to 32 × 29 mm (Figure *1*, Table *2*) [9,10]. Quantitative tumor attenuation measurements in Hounsfield units were unavailable in the provided report; therefore, the reported Choi classification could not be independently verified from the available data (Figure *3*).

In June 2026, fourth-line ripretinib 150 mg orally once daily was initiated [8,11]. Precision medicine considerations included planning for NGS profiling to assess PDGFRA exon 18 mutations and evaluate potential eligibility for avapritinib [11,12]. However, before discontinuation of ripretinib, the patient's clinical condition deteriorated, with progressive anemia (hemoglobin nadir, 8.2 g/dL), edema, and hypoalbuminemia (serum albumin, 21.4 g/L). The available laboratory results from the last assessment

before ripretinib discontinuation included urea, 37.0 mg/dL; creatinine, 0.8 mg/dL; uric acid, 6.6; total bilirubin, 0.46 mg/dL; alanine aminotransferase, 17.8 U/L; aspartate aminotransferase, 44.8 U/L; and alkaline phosphatase, 140 U/L [8,11]. During this period, the patient's clinical condition deteriorated, with a decline in functional status to ECOG 3. The patient preferred to continue anticancer treatment; however, the worsening clinical condition limited the feasibility of further anticancer therapy.

Formal reevaluation following three cycles of ripretinib, with CT imaging performed in September 2026, demonstrated interval enlargement of the heterogeneously enhancing exophytic gastric cardia mass. Multiple hypodense hepatic lesions involving both lobes, mild abdominopelvic ascites, and multiple para-aortic lymph nodes were also reported. These findings documented radiological disease progression during the later course of treatment and demonstrated a lack of clinical benefit (Figure *3*) [8,11]. Following radiologically documented disease progression, the patient transitioned to best supportive palliative care. The reported symptom burden included abdominal pain, reduced appetite, and decreased oral intake, without dyspnea. Supportive interventions included analgesia, albumin administration, intravenous hydration, and blood transfusion. Gastrointestinal intervention was assessed as having no role in the patient's management. Recognizing therapeutic exhaustion, the clinical team implemented a timely transition to best supportive palliative care (Figure *2*) [8,11,17].

Discussion

Choi response criteria vs Response Evaluation Criteria in Solid Tumors (RECIST) 1.1 in GIST surveillance

In solid tumor oncology, therapeutic response is traditionally evaluated using the RECIST 1.1, which primarily relies on changes in tumor size [16]. In GISTs, however, TKI-induced cellular responses produce rapid intratumoral myxoid degeneration, edema, hemorrhage, and devascularization, leading to a marked decrease in CT density, measured in Hounsfield units, without immediate volumetric tumor shrinkage [9,16]. Structural changes in tumor attenuation often serve as early indicators of therapeutic efficacy or emerging secondary resistance [4,18]. The integration of Choi criteria is therefore clinically important. These criteria define partial response as a greater than 10% decrease in the sum of the longest tumor diameters or a greater than 15% decrease in CT density, and progressive disease as a greater than 20% increase in tumor diameter, the development of new metastatic lesions, or a greater than 15% increase in post-contrast CT attenuation within existing lesions [16].

In the present case, the May 2026 CT report classified the findings as progressive disease according to Choi criteria. Although gastric wall thickness remained approximately stable at 28 mm, the report described an increase of more than 15% in post-contrast enhancement, enlargement of the largest hepatic lesion, new splenic lesions attributed to direct invasion by the adjacent gastric wall, and enlargement of the omental deposit. Quantitative tumor attenuation measurements in Hounsfield units were unavailable in the provided report; therefore, the Choi-based classification could not be independently verified. The overall imaging findings were reported as disease progression; however, the specific molecular mechanism of acquired resistance could not be established [9,16].

Comparison of case outcomes with landmark clinical trials

The clinical course of the present case illustrates both the real-world benefits and systemic barriers associated with multiline sequential TKI therapy for metastatic GISTs in a resource-constrained setting [3,12]. Comparing the patient's clinical outcomes with those reported in pivotal trials highlights key observations regarding therapeutic efficacy, progression patterns, and toxicity management [5-11,17].

In the landmark SWOG S0033 study, frontline imatinib mesylate achieved a median progression-free survival (PFS) of approximately 18-20 months in patients with unselected metastatic GISTs [5,17]. Our patient experienced a reported PFS interval of approximately 13 months on frontline imatinib before radiological disease progression [6,19]. Although this duration can be considered in the context of outcomes reported in clinical trials, KIT and PDGFRA mutation testing was not performed in this patient. Radiological progression indicated a loss of disease control during imatinib treatment; however, the specific molecular mechanism of resistance could not be established in this case. This patient-specific observation is derived from the clinical records and should not be interpreted as a direct comparison with trial outcomes [8,18,20].

In the pivotal phase III trial evaluating second-line sunitinib malate following imatinib failure, the median PFS was 5.6 months, with objective partial response rates typically below 10% because sunitinib predominantly induces disease stabilization [6]. Our patient achieved a marked radiological response, with a reduction in the reported tumor diameter from 13.3 cm to 7.6 cm, corresponding to an approximate 42.9% decrease, and a PFS of 11 months [7,21]. Although the INTRIGUE trial reported mutation-associated differences in treatment outcomes, molecular profiling was not performed in our patient. Therefore, his clinical course cannot be linked to a specific KIT mutation profile, and the mechanism underlying subsequent disease progression during sunitinib treatment cannot be established in this case [8,22,23]. These observations describe the patient's clinical course and do not establish that the treatment schedule was superior to the standard regimen.

In the phase III GRID trial, third-line regorafenib demonstrated a median PFS of 4.8 months, compared with 0.9 months with placebo [7]. Additional studies have investigated regorafenib activity in previously treated GISTs, including its activity against tumors harboring different KIT mutations [9,19]. Our patient achieved a PFS of nine months, exceeding the median reported in the clinical trial [9,10]. Notably, the emergence of drug-induced hypothyroidism, with a TSH elevation to 25.34 mIU/L, was managed with levothyroxine replacement. Thyroid dysfunction is a recognized adverse effect associated with this class of TKIs. These laboratory findings and the treatment response are derived from the patient's medical records and should be distinguished from published trial findings.

Choi criteria were used to assess radiological changes during follow-up, including increased post-contrast enhancement and subsequent disease progression with direct splenic invasion. These findings illustrate the potential value of Choi criteria in GIST surveillance, although imaging alone cannot establish the underlying molecular mechanism of resistance [9,16].

In the landmark phase III INVICTUS trial, the fourth-line switch-control inhibitor ripretinib yielded a median PFS of 6.3 months and a significant overall survival benefit compared with placebo in heavily pretreated patients [8]. Our patient received ripretinib for approximately three months before experiencing clinical deterioration and subsequently transitioning to supportive palliative care [8,11,17]. The shorter disease-control duration observed in this case (approximately three months, compared with the 6.3-month median reported in INVICTUS) occurred in the context of progressive disease, clinical deterioration, severe anemia, hypoalbuminemia-related edema, and interruptions in drug availability. However, the contribution of each factor to the shorter duration of disease control cannot be determined from this single case [8,12,17]. Although compound KIT mutations involving the ATP-binding pocket and activation loop have been described as mechanisms of resistance to TKIs, their presence could not be confirmed in this patient. The decision to transition to palliative care was based on the patient's clinical deterioration and the lack of meaningful benefit from further treatment [18,24,25].

Mechanisms of secondary resistance and molecular heterogeneity

Secondary resistance to imatinib in GISTs is commonly associated with secondary mutations in KIT that affect kinase domain structure and sensitivity to TKIs [8,18,20]. Secondary KIT mutations may be distributed heterogeneously across different metastatic sites and may influence sensitivity to subsequent TKIs. Mutations in the ATP-binding pocket (exons 13 and 14) and activation loop (exons 17 and 18) have been associated with different patterns of drug sensitivity [18,24]. In some cases, mutations involving these regions may coexist in cis, potentially contributing to resistance to multiple TKIs [24,25]. However, KIT and PDGFRA molecular testing was not performed in our patient; therefore, these mechanisms are discussed as established findings in the literature and cannot be confirmed as the cause of resistance in this individual case [8,9,22]. Recent molecular insights demonstrate that secondary mutations in the activation loop and ATP-binding pocket can occasionally occur on the same allele in cis, creating highly resistant clones that are refractory to standard TKIs [18,20,25].

Toxicity management, dosing flexibility, and palliative transition

Multikinase inhibitors such as regorafenib and sunitinib commonly cause thyroid dysfunction through destructive thyroiditis and impaired thyroid hormone synthesis [10,24]. Regular laboratory monitoring identified hypothyroidism during regorafenib treatment, for which levothyroxine replacement was initiated. Furthermore, adapting the sunitinib schedule to two weeks on and two weeks off was used to manage grade 2 hand-foot skin reactions while maintaining treatment exposure and clinical efficacy, in keeping with modern clinical strategies. However, a single case cannot establish the efficacy of the modified schedule or demonstrate its equivalence to the standard regimen. The available data were insufficient to determine whether cumulative drug exposure was maintained [7,15,26].

In resource-limited health care settings lacking upfront genomic sequencing, TKI rotation generally follows standard empirical sequential lines of therapy (imatinib, sunitinib, regorafenib, and ripretinib) [5,7-9].

Reserving targeted NGS for multidrug-resistant disease, such as assessing PDGFRA exon 18 mutations to determine eligibility for avapritinib, may represent a pragmatic, cost-effective precision medicine strategy. The optimal timing of molecular testing should be guided by clinical considerations, local resources, and applicable guidelines [12-14]. After three cycles of ripretinib, progressive anemia, hypoalbuminemia-related edema, a lack of meaningful clinical benefit, and interruptions in drug availability contributed to the decision to transition to best supportive palliative care, with an emphasis on symptom control and quality of life [11,17].

Conclusions

This case illustrates the challenges of managing metastatic gastric GIST in a resource-limited setting, including treatment-related toxicity, radiologically documented disease progression, and limited access to molecular profiling and targeted therapies. The patient's clinical course involved sequential targeted therapies, serial CT assessments, management of documented toxicities, and a transition to palliative care

following further disease progression and clinical deterioration. As a single case, this report cannot establish the efficacy of the treatment sequence, demonstrate the superiority of a modified dosing schedule, or determine whether alternative management decisions would have changed the outcome. It highlights the practical importance of access to molecular diagnostics, appropriate targeted therapies, and supportive palliative care for patients with rare malignancies in resource-limited settings.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Zahraa A. Mohammed, Anmar Z. Alharganee

Acquisition, analysis, or interpretation of data: Zahraa A. Mohammed, Anmar Z. Alharganee, Tabarek M. Obaid, Alhussein Ashraf Kadhim

Drafting of the manuscript: Zahraa A. Mohammed, Anmar Z. Alharganee

Critical review of the manuscript for important intellectual content: Zahraa A. Mohammed, Anmar Z. Alharganee, Tabarek M. Obaid, Alhussein Ashraf Kadhim

Supervision: Anmar Z. Alharganee

Disclosures

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