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Imetelstat: A First-in-Class Telomerase Inhibitor for the Treatment of Patients With Lower-Risk Myelodysplastic Syndromes and Anemia

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Abstract

Imetelstat is a first-in-class, direct, and competitive inhibitor of telomerase enzymatic activity that selectively induces apoptosis of malignant clones and allows for recovery of erythropoiesis. Imetelstat was approved by the United States Food and Drug Administration in June 2024 and the European Medicines Agency in March 2025 for the treatment of certain patients with lower-risk (low to intermediate-1) myelodysplastic syndromes (LR-MDS) with transfusion-dependent anemia who have failed or lost response to or are ineligible for erythropoiesis-stimulating agents. Imetelstat is infused at 7.1 mg/kg (active dose, equivalent to 7.5 mg/kg sodium salt) intravenously over 2 hours once every 4 weeks. In the pivotal IMerge trial in LR-MDS, significantly more patients treated with imetelstat vs. placebo, respectively, achieved ≥ 8-week RBC-transfusion independence (TI; 40% [95% confidence interval [CI] = 30.9–49.3] vs. 15% [95% CI = 7.1–26.6]) and ≥ 24-week RBC-TI (28% [95% CI = 20.1–37.0] vs. 3% [95% CI = 0.4–11.5]). The safety profile of imetelstat was characterized primarily by cytopenias, including neutropenia (incidence of 74% any grade and 68% grade 3–4 events) and thrombocytopenia (75% and 62%, respectively). Grade 3 to 4 hematologic events occurred early in the treatment and had a median duration of 1.9 weeks for neutropenia and 1.4 weeks for thrombocytopenia; cases resolved to grade ≤ 2 within 2 weeks in 81% and 86% of cases, respectively, with limited severe complications. This review highlights key topics related to the use of imetelstat in patients with LR-MDS, including its mechanism of action, clinical efficacy and safety data, dosing and administration, management of adverse events, and notable clinical practice implications.

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Myelodysplastic syndromes (MDS) are a group of hematologic malignancies characterized by ineffective hematopoiesis, cytopenias, and inherent risk for developing acute myeloid leukemia (AML). Myelodysplastic syndromes are derived from and propagated by malignant clones that arise due to the acquisition of genetic and epigenetic alterations in normal hematopoietic stem cells (Li et al., 2022). Myelodysplastic syndromes affect an estimated 3.9 in 100,000 people in the United States (US; 2017–2021) but is more prevalent in older adult populations (≥ 65 years: 26.1/100,000; ≥ 75 years: 40.0/100,000); however, true age-adjusted incidences are difficult to estimate (National Cancer Institute, 2023; Zeidan et al., 2019). Approximately two thirds of initial MDS diagnoses are lower risk (LR) according to the Revised International Prognostic Scoring System (IPSS-R; Carraway & Saygin, 2020; Steensma, 2018). Lower-risk myelodysplastic syndromes often present with nonspecific symptoms that may include fatigue, diminished appetite, bruising, and recurrent bacterial infections due to anemia, thrombocytopenia, and/or neutropenia (Sekeres & Taylor, 2022).

The primary treatment goal in LR-MDS is to reduce disease-related symptoms, notably those associated with anemia. Red blood cell (RBC) transfusions can provide temporary improvements; however, RBC-transfusion dependence (TD) can develop and is associated with poor outcomes, decreased quality of life, and increased risk of iron overload (Carraway & Saygin, 2020; Fenaux et al., 2021; Sekeres & Taylor, 2022). Erythropoiesis-stimulating agents (ESA; e.g., recombinant erythropoietin [EPO; Epogen], darbepoetin alfa [Aranesp]) are first-line options for eligible patients (i.e., serum EPO ≤ 500 units/L) but have limited duration of response (Carraway & Saygin, 2020; Fenaux et al., 2021; Sekeres & Taylor, 2022). Lenalidomide (Revlimid) is indicated for RBC-TD anemia due to LR-MDS with 5q deletion, which accounts for ~15% of all MDS cases (Celgene Corporation, a Bristol-Myers Squibb Company, 2023; Schanz et al., 2012). Luspatercept (Reblozyl), an erythroid maturation agent, is indicated for anemia in patients with RBC-TD LR-MDS who are ESA-naive (regardless of ring sideroblast [RS] status) or who are ESA-refractory with RS (Celgene Corporation, a Bristol-Myers Squibb Company, 2024), but data in patients with non-RS or non-*SF3B1*–mutated disease are less robust, requiring further investigation (Della Porta et al., 2024). Hypomethylating agents (e.g., azacitidine [Vidaza], decitabine, and cedazuridine [Inqovi]) are commonly used in the second-line setting but are associated with treatment-emergent cytopenias and adverse events (AE), requiring ongoing supportive care (Carraway & Saygin, 2020; Fenaux et al., 2021; Haumschild et al., 2024; Sekeres & Taylor, 2022).

In June 2024, imetelstat (Rytelo) was approved for the treatment of adults with LR-MDS with RBC-TD anemia requiring ≥ 4 RBC units/8 weeks who have not responded to or have lost response to or are ineligible for ESAs in the US (Geron Corporation, 2024). The European Union approval was granted in March 2025 for the treatment of patients with TD anemia due to very low, low, or intermediate risk non-del(5q)-MDS who had unsatisfactory response to or were ineligible for ESAs (Geron Corporation, 2025).

MECHANISM OF ACTION

Imetelstat is a first-in-class, 13-mer oligonucleotide inhibitor of human telomerase that acts as a direct competitive inhibitor of telomerase enzymatic activity. Telomeres are nucleoprotein structures on the end of chromosomes. The replicative lifespan of a cell is regulated by telomere length, which progressively shortens with each cell division until a critical threshold is reached that triggers replicative senescence and apoptosis (Jafri et al., 2016; Kerbauy & Deeg, 2007; Tavares et al., 2017). In malignant cells, human telomerase reverse transcriptase expression is often upregulated, leading to reactivation of telomerase, enabling cancer cells to maintain critically short telomere length, acquire cellular immortality, proliferate uncontrollably, and avoid apoptosis (Harley, 2008; Lichtsteiner et al., 1999; Nakamura et al., 1997). While there is transient signal of telomerase activity in early progenitor stem cells and activated cells, telomerase activity is not detected in normal adult tissues (Bodnar et al., 1996; Wright et al., 1996). Thus, the high level of telomerase expression common to most cancers and lack of telomerase activity in most normal tissue counterparts

support telomerase inhibition as a rational target for the treatment of a wide range of cancers, including LR-MDS (Fan et al., 2021; Harley, 2008).

CLINICAL TRIALS

IMerge (MDS3001; NCT02598661) is a phase II/III trial, with an ongoing extension, assessing the efficacy and safety of imetelstat in patients with LR-MDS by IPSS who were RBC-TD (requiring ≥ 4 RBC units/8 weeks) and relapsed/refractory to or ineligible for ESAs at enrollment (Platzbecker et al., 2024; Steensma et al., 2021). In the double-blind phase III study, patients were randomized 2:1 to receive imetelstat (n = 118) or placebo (n = 60), stratified by prior RBC transfusion burden (4–6 units or > 6 units/8 weeks) and IPSS risk group (low or intermediate-1). Disease was assessed every 12 weeks for the first 72 weeks, then every 24 weeks until treatment discontinuation, and, if possible, within 30 days of the last dose for discontinued patients. Bone marrow aspirate was collected at screening and every 24 weeks thereafter (or at the time of a suspected response). Patients were heavily transfused (median 6 units/8 weeks [interquartile range, 6–8]), and 90% and 6% of patients had prior ESA or luspatercept treatment, respectively (Platzbecker et al., 2024). The primary endpoint of ≥ 8-week RBC-transfusion independence (TI) was achieved by 40% and 15% of imetelstat- and placebo-treated patients, respectively; among these responders, the median duration of RBC-TI was 51.6 weeks vs. 13.3 weeks (Platzbecker et al., 2024). More patients in the imetelstat vs. placebo groups achieved the key secondary endpoint of ≥ 24-week RBC-TI (28% vs. 3%, respectively) and ≥ 1-year RBC-TI (18% vs. 2%; post-hoc). Hematologic improvement-erythroid per International Working Group 2018 (key secondary endpoint) was achieved in 42% vs. 13% of patients, respectively (Platzbecker et al., 2024). In 8-week RBC-TI responders, the median (range) change in hemoglobin from baseline was 3.55 g/dL (−0.07 to 13.76) for imetelstat and 0.80 g/dL (−0.16 to 1.67) for placebo. As measured by the Functional Assessment of Chronic Illness Therapy (FACIT)-Fatigue scale, 50% (n = 59/118) of imetelstat-treated patients reported sustained, meaningful improvement in fatigue (placebo, n = 23/57 [40%]), where imetelstat had a shorter median time to the first reported sustained improvement (28 weeks [95% confidence interval (CI) = 16.6–64.1]) vs. placebo (65 weeks [95% CI = 24.0–not estimable]). Subgroup analyses according to baseline disease characteristics (e.g., RS and mutational statuses, transfusion burden, telomerase activity) demonstrated comparable achievement of ≥ 8-week RBC-TI among imetelstat-treated patients (Platzbecker et al., 2024). Cytogenetic responses (complete or partial) were observed in 35% (n = 9/26) of imetelstat-treated patients vs. 15% (n = 2/13) of placebo recipients. Imetelstat-treated patients also demonstrated reductions in variant allele frequency (i.e., clearance) of the most frequently mutated genes associated with MDS (*SF3B1, TET2, DNMT3A*, and *ASXL1*) and reduction in RS cell counts, suggesting disease-modifying potential for imetelstat.

ADVERSE EVENTS

The IMerge trial showed a manageable safety profile for imetelstat. Neutropenia, thrombocytopenia, and anemia were the most frequently reported any-grade treatment-emergent AEs (TEAE) in the IMerge phase III trial (Table 1; Platzbecker et al., 2024). Most nonhematologic TEAEs occurred at low grades. Patient incidence of serious TEAE was 32% for imetelstat vs. 22% for placebo.

The most common grade 3 to 4 TEAEs in imetelstat-treated patients in phase III were neutropenia (68%) and thrombocytopenia (62%), compared with 3% and 8% for placebo, respectively. The median duration of grade 3 to 4 neutropenia by laboratory assessment in imetelstat-treated patients was short (1.9 weeks); resolution to grade ≤ 2 occurred within 4 weeks for most patients (81% [226/279]). Similarly, the median duration of grade 3 to 4 thrombocytopenia was 1.4 weeks, and 86% of events resolved to grade ≤ 2 within 4 weeks. For most patients, these TEAEs occurred early during treatment, within the first three cycles (Platzbecker et al., 2024). There were similar rates of severe clinical consequences of neutropenia and thrombocytopenia (e.g., grade 3 to 4 bleeding events or infections and grade 3 febrile neutropenia) between the imetelstat and placebo groups.

Of the 58 (49%) imetelstat-treated patients requiring dose reductions, most were due to neutropenia (67%) and thrombocytopenia (47%). In the

Table 1. TEAEs Reported in ≥ 10% of Patients After Imetelstat in the IMerge Trial

TEAE, *n* (%)	Imetelstat (*N* = 118)		Placebo (*N* = 59)	
	Any grade	Grade 3–4	Any grade	Grade 3–4
Patients with ≥ 1 TEAE	117 (99)	107 (91)	59 (100)	28 (47)
Thrombocytopenia	89 (75)	73 (62)	6 (10)	5 (8)
Neutropenia	87 (74)	80 (68)	4 (7)	2 (3)
Anemia	24 (20)	23 (19)	6 (10)	4 (7)
Asthenia	22 (19)	0	8 (14)	0
COVID-19	18 (15)[a]	3 (3)[b]	8 (14)[a]	3 (5)[b]
Headache	15 (13)	1 (1)	3 (5)	0
ALT increased	14 (12)	3 (3)	4 (7)	2 (3)
Diarrhea	14 (12)	1 (1)	7 (12)	1 (2)
Peripheral edema	13 (11)	0	8 (14)	0
Leukopenia	12 (10)	9 (8)	1 (2)	0
Hyperbilirubinemia	11 (9)	1 (1)	6 (10)	1 (2)
Constipation	9 (8)	0	7 (12)	0
Pyrexia	9 (8)	2 (2)	7 (12)	0

Note. ALT = alanine aminotransferase; TEAE = treatment-emergent adverse event.
[a]Includes COVID-19, asymptomatic COVID-19, and COVID-19 pneumonia.
[b]Only COVID-19 pneumonia events were classified as grade 3–4 events for COVID-19.

imetelstat group, the most common TEAEs leading to treatment discontinuation were neutropenia (5%) and thrombocytopenia (3%). Disease progression occurred in 6% of imetelstat-treated patients and 8% of placebo recipients; progression to AML occurred in 2 patients and 1 patient, respectively. Overall, 27 patients died during the trial, with most occurring in the posttreatment follow-up period (19 in the imetelstat arm and 8 in the placebo arm; Platzbecker et al., 2024).

Results of a QTc substudy of IMerge show low proarrhythmic risk with imetelstat treatment (Lennox et al., 2025). Other reports suggest that clinically meaningful drug interactions are unlikely with imetelstat (Lennox et al., 2024).

DOSAGE AND ADMINISTRATION

Imetelstat is available in two strengths for reconstitution per the intended dose (31.4 mg/mL) based on patient weight as a single-dose vial of lyophilized powder (Geron Corporation, 2024). The recommended dosage of imetelstat is 7.1 mg/kg active dose (equivalent to 7.5 mg/kg imetelstat sodium) administered intravenously (IV) over 2 hours every 4 weeks (Geron Corporation, 2024; Platzbecker et al., 2024). Patients should be monitored for ≥ 1 hour after imetelstat infusion for infusion-related reactions (IRR). Pretreatment with diphenhydramine (or equivalent; 25–50 mg, IV or oral) and hydrocortisone (or equivalent; 100–200 mg, IV or oral) ≥ 30 minutes before infusion is recommended to mitigate IRRs. Due to the risk of cytopenias with imetelstat, blood counts should be routinely monitored while on treatment. Management of grade 2 to 4 TEAEs may require dose adjustments, delays, or interruptions as outlined in Table 2. Imetelstat should be discontinued if no decrease in RBC transfusion burden after six doses (24 weeks of treatment) or unacceptable toxicity occurs. There is no indication that imetelstat is emetogenic and, therefore, does not require antiemetic prophylaxis.

CLINICAL PRACTICE IMPLICATIONS

Since the recent approval for the treatment of certain adults with LR-MDS with RBC-TD anemia either unable to receive ESAs or have disease relapsed or refractory to ESAs, imetelstat use is increasing in the clinic (Geron Corporation, 2024). Additional clinical research and real-world evidence will continue to inform treatment decision-making in this evolving landscape.

Table 2. Imetelstat Dosing Recommendations for the Treatment of Patients With LR-MDS

FDA-recommended dosage			
7.1 mg/kg intravenously over 2 hours every 4 weeks (equivalent to 7.5 mg/kg imetelstat sodium)			
Recommended dose reduction schedule for grade 3–4 TEAEs			
First reduction			5.6 mg/kg every 4 weeks (equivalent to 6.0 mg/kg imetelstat sodium)
Second reduction			4.4 mg/kg every 4 weeks (equivalent to 4.7 mg/kg imetelstat sodium)
Dosage modifications for grade 3–4 hematologic TEAEs			
TEAE	**Severity grade[a]**	**Event**	**Treatment modification**
Thrombocytopenia	3	First	Delay imetelstat until recovery of platelets to 50 × 10^9/L; restart at same dose level
		Second and third	Delay imetelstat until recovery of platelets to 50 × 10^9/L; restart at 1 dose level lower
		Fourth	Discontinue imetelstat
	4	First and second	Delay imetelstat until recovery of platelets to 50 × 10^9/L; restart at 1 dose level lower
		Third	Discontinue imetelstat
Neutropenia	3	First	Delay imetelstat until recovery of ANC to 1 × 10^9/L; restart at same dose level
		Second and third	Delay imetelstat until recovery of ANC to 1 × 10^9/L; restart at 1 dose level lower
		Fourth	Discontinue imetelstat
	4	First and second	Delay imetelstat until recovery of ANC to 1 × 10^9/L; restart at 1 dose level lower
		Third	Discontinue imetelstat
Dosage modifications for grade 2–4 TEAEs			
Infusion-related reactions	2 or 3	First and second	Interrupt the imetelstat infusion until resolution of the adverse reaction or until the intensity of the reaction decreases to grade 1; restart infusion at 50% of the infusion rate administered before the adverse reaction
		Third	For grade 2, stop infusion; may restart at next cycle. For grade 3, permanently discontinue imetelstat
	4	First	Stop infusion, administer supportive care as appropriate and permanently discontinue imetelstat
Other adverse reactions, including elevated liver function tests	3 or 4	First and second	Delay imetelstat until recovery of adverse reactions to grade 1 or baseline; restart at 1 dose level lower
		Third	Permanently discontinue imetelstat

Note. ANC = absolute neutrophil count; FDA = United States Food and Drug Administration; LR-MDS = lower-risk myelodysplastic syndromes; TEAE = treatment-emergent adverse event.
[a]Severity based on National Cancer Institute Common Terminology Criteria for Adverse Events, version 4.03.

Given that cytopenias are a common symptom of LR-MDS, as well as an AE after imetelstat treatment, it is important to understand how to address initiating treatment in patients with existing cytopenias. All patients in the IMerge trial must have had an absolute neutrophil count (ANC) ≥ 1.5 × 10^9/L and platelets ≥ 75 × 10^9/L for study inclusion. Therefore, per prescribing information, complete blood cell counts should be performed before administration, weekly for the first two cycles, and as clinically indicated thereafter. For neutropenia, administration of growth factors and anti-infective therapies for treatment or prophylaxis may be given, as appropriate. For thrombocytopenia, platelet transfusions may be warranted. Additionally, dose modification instructions in the prescribing information indicate dose delay, dose reduction, or discontinuation. Nevertheless, if at the time of dosing ANC fall below 1 × 10^9/L, for example, imetelstat should be withheld. One option could be to potentially administer prophylactic myeloid growth factors in the case of prolonged neutropenia. According to the prescribing information, if an ANC of < 1.0 × 10^9/L followed by recovery to > 1.0 × 10^9/L occurs twice, imetelstat should be resumed one dose level lower than the prior dose (Geron Corporation, 2024). In this situation, weekly monitoring should be sufficient until resolution. Data available for patients with cytopenias that are more severe (e.g., have lower ANC or platelets) than allowed per IMerge inclusion criteria and treated with imetelstat are needed.

Infusion-related reactions occurred in 8% of patients, and grade 3 to 4 IRRs in 1.7% in IMerge. Premedication with diphenhydramine and hydrocortisone as prophylaxis against IRRs, per prescribing information, may mitigate these effects (Geron Corporation, 2024).

CONCLUSION

In the IMerge phase III trial, 40% of imetelstat-treated patients with RBC-TD LR-MDS who were relapsed/refractory or ineligible for ESAs achieved ≥ 8-week RBC-TI, with 28% and 18% maintaining RBC-TI for ≥ 24 weeks and ≥ 1 year, respectively. Cytopenias were the most common TEAEs observed, but most occurred early during treatment, were transient, and were manageable with supportive care. Imetelstat is unique in the treatment landscape in that its activity was robust regardless of RS status and in patients with a high transfusion burden. Future results from ongoing imetelstat trials in myelofibrosis (randomized phase III monotherapy trial [NCT04576156]; phase Ib imetelstat plus ruxolitinib trial, [NCT05371964]), high-risk MDS/AML (single-arm, phase II monotherapy [NCT05583552]), pediatric relapsed/refractory AML, MDS, or juvenile myelomonocytic leukemia trial (NCT06247787), and the additional IMerge phase III long-term follow-up are eagerly anticipated. ●

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