Pomalidomide Is Active in the Treatment of Anemia Associated With Myelofibrosis


ABSTRACT

Purpose
Thalidomide and lenalidomide can alleviate anemia in myelofibrosis. However, their value is undermined by their respective potential to cause peripheral neuropathy and myelosuppression. We therefore evaluated the safety and therapeutic activity of another immunomodulatory drug, pomalidomide.

Methods
In a phase II randomized, multicenter, double-blind, adaptive design study, four treatment arms were evaluated: pomalidomide (2 mg/d) plus placebo, pomalidomide (2 mg/d) plus prednisone, pomalidomide (0.5 mg/d) plus prednisone, and prednisone plus placebo. Pomalidomide was administered for up to 12 28-day treatment cycles. Prednisone (30 mg/d) was given in a tapering dose schedule during the first three cycles. Response was assessed by International Working Group criteria.

Results
Eighty-four patients with myelofibrosis-associated anemia were randomly assigned to the aforementioned treatment arms: 22, 19, 22, and 21, respectively. Response in anemia was documented in 20 patients, including 15 who became transfusion independent. Response rates in the four treatment arms were 23% (95% CI, 5% to 41%), 16% (95% CI, 0% to 33%), 36% (95% CI, 16% to 56%), and 19% (95% CI, 2% to 36%). The corresponding figures for patients receiving ≥3 cycles of treatment (n = 62) were 38%, 23%, 40%, and 25%. Response to pomalidomide with or without prednisone was durable (range, 3.2 to 16.9+ months) and significantly better in the absence of leukocytosis (37% v 8%; P = .01; JAK2V617F or cytogenetic status did not affect response. Grade ≥3 toxicities were infrequent and included (in each treatment arm) neutropenia (9%; 16%; 5%; 5%), thrombocytopenia (14%; 16%; 9%; 5%), and thrombosis (9%; 5%; 0%; 0%).

Conclusion
Pomalidomide therapy at 0.5 or 2 mg/d with or without an abbreviated course of prednisone is well tolerated in patients with myelofibrosis and active in the treatment of anemia.

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INTRODUCTION

Lenalidomide and pomalidomide are second generation IMiDs immunomodulatory drugs that are created by chemical modification of thalidomide with the intent to reduce toxicity and enhance anticancer and immunological activity. In the United States, thalidomide is approved by the US Food and Drug Administration for use in acute erythema nodosum leprosum and, in combination with dexamethasone, in newly diagnosed multiple myeloma. Pomalidomide is currently not US Food and Drug Administration–approved but is known to have therapeutic activity in multiple myeloma,7,8 All three drugs display antiangiogenic, anti-tumor necrosis factor (TNF) -α, and T-cell costimulatory activity. Lenalidomide and pomalidomide are more potent than thalidomide in this regard, and were also recently shown to inhibit T-regulatory cell proliferation and suppressor function. The composite effect of these immune associated with transfusion-dependent anemia and a deletion 5q cytogenetic abnormality and, in combination with dexamethasone, in relapsed multiple myeloma. Pomalidomide is currently not US Food and Drug Administration–approved but is known to have therapeutic activity in multiple myeloma, and T-cell costimulatory activity. Lenalidomide and pomalidomide are more potent than thalidomide in this regard, and were also recently shown to inhibit T-regulatory cell proliferation and suppressor function. The composite effect of these immune
and cytokine modulatory properties is believed to underlie the mechanism of action for these drugs and the rationale for investigating their therapeutic value in a variety of other conditions including myelofibrosis.

The abnormal cytokine milieu in myelofibrosis is believed to contribute to its cardinal disease features including anemia, constitutional symptoms, cachexia, and extramedullary hematopoiesis.10 Therefore, based on their aforementioned anticytokine and antiangiogenic properties, it was reasonable to explore the therapeutic activity of immunomodulatory compounds in myelofibrosis. Single-agent thalidomide, at doses levels of ≥ 100 mg/d, is poorly tolerated in patients with myelofibrosis and long-term use causes peripheral neuropathy.11-16 Furthermore, if one were to apply the International Working Group for Myelofibrosis Research and Treatment (IWG-MRT) criteria,17 the anemia response rates seen with single-agent thalidomide therapy might be as low as 10% and unlikely to exceed 20%. The same can be said regarding the antianemia effect of lenalidomide in myelofibrosis, in the absence of a deletion 5q cytogenetic abnormality.18,19 Lenalidomide use in myelofibrosis is further limited by its potential to cause severe myelosuppression.18 Therefore, we studied pomalidomide, hoping for better toxicity profile and improved erythropoietic activity in myelofibrosis.

**Methods**

**Study Design**

The current study was a phase II, prospective, randomized, multicenter, double-blind, active-control, parallel-group study to determine the safety of and to select a treatment regimen of pomalidomide either as single-agent or in combination with prednisone to study further in patients with myelofibrosis. Study patients were randomly assigned to four treatment arms: pomalidomide (2 mg/d) and placebo, pomalidomide (2 mg/d) and prednisone, pomalidomide (0.5 mg/d) and prednisone, and prednisone (active control). The primary objective of the statistical analysis was to select, for further study, one of the pomalidomide regimens. An adaptive selection design using a Bayesian β prior/binomial likelihood model with regular monitoring was used to potentially eliminate early those regimens that were unlikely to be effective and, ultimately, to select the most effective pomalidomide regimen.20 A maximum of 80 subjects were expected to enroll in the current study, 20 per treatment arm, barring early stopping.

**Protocol Therapy**

Pomalidomide 2 or 0.5 mg/d (or placebo) was administered for up to 12 28-day treatment cycles with the option to continue treatment beyond that point in the presence of treatment response and absence of unacceptable toxicity. These doses of pomalidomide were selected based on previous observations from Schey et al21 who established the maximum tolerated dose (MTD) of pomalidomide in refractory/relapsed multiple myeloma at 2 mg/d. Prednisone (or placebo) was given in combination with pomalidomide (or placebo) in a tapering dose schedule during the first three cycles only: starting dose of 30 mg/d during the first cycle, 15 mg/d the second cycle, and 15 mg every other day the third cycle. Pomalidomide (or placebo) dose reduction by 50% was allowed in case of adverse events that warranted such an action. Dose escalation was not allowed. During or within 28 days of protocol therapy, use of growth factors or any other treatment for myelofibrosis was not allowed. The usual strict guidelines for contraception (and avoidance of breast feeding) while on immunomodulatory compound therapy were followed for both male and female patients.21

**Patients**

Multiple centers (n = 10) from the Unites States and Europe participated in this study after institutional review board approval. Eligibility criteria included a diagnosis of primary, polycythemia vera, or postessential thrombocythemia myelofibrosis, according to the WHO criteria.22 Additional requirements included a hemoglobin level lower than 10 g/dL or RBC transfusion dependence (the latter required documentation of transfusion with ≥ 2 units of packed RBCs, for a hemoglobin level of < 8.5 g/dL, during the 28 days before protocol entry),17 an absolute neutrophil count ≥ 1 × 10^9/L, a platelet count ≥ 50 × 10^9/L, a creatinine level ≤ 2 mg/dL, direct bilirubin level lower than 2 times the upper limit of normal, and blood transaminase level ≤ 3 times the upper limit of normal unless attributed to extramedullary hematopoiesis. Patients with prior exposure to pomalidomide, lenalidomide, or thalidomide were excluded. Also excluded were patients with history of deep vein thrombosis or pulmonary embolism within 1 year of study entry. Response was assessed by the IWG-MRT criteria.17 Toxicity was assessed by the National Cancer Institute Common Toxicity Criteria for Adverse Events, version 3.23

**Results**

A total of 104 patients were screened and 84 fulfilled protocol eligibility criteria and were randomly assigned to one of four treatment arms: pomalidomide (2 mg/d) plus placebo (n = 22), pomalidomide (2 mg/d) plus prednisone (n = 19), pomalidomide (0.5 mg/d) plus prednisone (n = 22), and prednisone plus placebo (n = 21). The four treatment arms were well balanced in terms of baseline patient characteristics including disease duration and prior therapy (Table 1).

**Response Rates and Duration**

After a median treatment period of 4.6 months (range, 0.3 to 18) for all patients and 10 months (range, 6 to 17.7) for patients still on-study, a total of 20 responses were documented and all signified improvement in anemia and none represented reduction in spleen size. Responders included 15 patients who became RBC transfusion independent. Response rates among the four treatment arms were: 23% (pomalidomide 2 mg/d plus placebo; 95% CI, 5% to 41%), 16% (pomalidomide 2 mg/d plus prednisone; 95% CI, 0% to 33%), 36% (pomalidomide 0.5 mg/d plus prednisone; 95% CI, 16% to 56%), and 19% (prednisone plus placebo; 95% CI, 2% to 36%). The corresponding figures for patients receiving at least three cycles of treatment (n = 62) were 38%, 23%, 40%, and 25%. Median response duration was 6.5 months (range, 2.3 to 16.9). Median time to response in the four arms was 2 months (range, 1 to 7), 3 months (range, 1 to 3), 2 months (range, 1 to 7), and 3 months (range, 1 to 4). To date, three of the four prednisone responders have relapsed after response duration of 2.3 to 5.5 months (Table 2). The median response duration in the 16 patients who responded to pomalidomide with or without prednisone was 7.8 months (range, 3.2 to 16.9) and two have relapsed during this period (Table 2).

**Predictors of Response**

Univariate analysis in patients receiving pomalidomide (0.5 or 2 mg/d) with or without prednisone (n = 63) did not show a correlation between response and Lille score24 (P = .31), myelofibrosis subtype (primary vs postessential thrombocythemia vs postpolycythemia vera; P = .50), platelet count (P = .64), RBC transfusion dependency at baseline (P = .58), age (P = .32), or sex (P = .96). Similarly, the presence of either JAK2V617F (number assessable = 84) or abnormal cytogenetics (number assessable = 47) did not affect response; 11 (31%) of 36 JAK2V617F-positive patients responded as opposed to 5 (19%) of 27 mutation-negative patients (P = .28); six
(40%) of 15 patients with abnormal cytogenetics responded as opposed to four of 21 without (P = .17). None of the six responders with abnormal cytogenetics had complex abnormalities; the simple cytogenetic lesions included monosomy 7 ± r(7) in two patients and del(13q), del(20q), der(6)t(1q;6p), and trisomy 9 in one patient each. The latter patient also had a t(X;14)(q26;q32) abnormality. Two patients with del(5q) were randomly assigned to the prednisone only arm and did not respond. Leukocyte count higher than 10 × 10^9/L or palpable spleen size 10 cm correlated with lower response. Two (8%; 95% CI, 0% to 19%) of 25 patients with leukocytosis responded as opposed to 14 (37%; 95% CI, 22% to 52%) of 38 without (P = .01); five (15%; 95% CI, 3% to 27%) of 33 patients with marked splenomegaly responded as opposed to 11 (37%; 95% CI, 20% to 54%) of 30 without (P = .05). However, only leukocytosis remained significant during multivariable analysis (Table 3).

**Treatment Effect on Other Blood Parameters**

This study did not include patients with platelet counts of lower than 50 × 10^9/L or absolute neutrophil counts of lower than 1 × 10^9/L; therefore, the drug's effect on clinically relevant thrombocytopenia or neutropenia could not be accurately assessed. However, a greater than 50% increase in platelet count was recorded in six (43%) of 14 patients who received pomalidomide (0.5 or 2 mg/d) with or without prednisone and had baseline platelet counts in the range of 50 to 100 × 10^9/L (baseline platelet counts of 64 to 86 × 10^9/L increased to peak levels of 148 to 293 × 10^9/L). Unlike our previous experience with lenalidomide, serum lactate dehydrogenase level was seldom affected by pomalidomide with or without prednisone therapy (normalized in only two responders) and treatment-induced changes in leukocytosis were infrequent (data not shown).

**Treatment Effect on Histology, Cytogenetics, or JAK2V617F Allele Burden**

Follow-up bone marrow examination was available in four responders who completed 1 year of treatment with pomalidomide with or without prednisone. In three of these patients, overall bone marrow cellularity, degree of fibrosis, or cytogenetic findings did not change (Table 2; patients 5, 7, and 12). In one patient (Table 2; patient 6), bone marrow fibrosis decreased from grade 3+ to grade 1. This patient received pomalidomide 2 mg/d plus prednisone and also had a partial cytogenetic remission after 1 year of protocol therapy; number of metaphases with monosomy 7 and r(7) decreased from 18 of 20 to three of 20 metaphases examined. However, the patient developed, after 16 months of therapy, marked leukocytosis with eosinophilia, and pleural effusion. A repeat bone marrow examination at the time revealed monosomy 7 and r(7) in all metaphases.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>All Patients</th>
<th>Pomalidomide (2 mg/d) + Placebo</th>
<th>Pomalidomide (2 mg/d) + Prednisone</th>
<th>Pomalidomide (0.5 mg/d) + Prednisone</th>
<th>Prednisone + Placebo</th>
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</thead>
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<tr>
<td>No. of patients</td>
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<td>22</td>
<td>19</td>
<td>22</td>
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<td>Median age, years</td>
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<td>67</td>
<td>70</td>
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<td>68</td>
<td>17</td>
<td>59</td>
<td>67</td>
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<td>2</td>
<td>15</td>
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<td>4</td>
<td>2</td>
<td>2</td>
<td>2</td>
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<tr>
<td>Post-PV</td>
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<td>0.6</td>
<td>0.11</td>
<td>1.1</td>
<td>1.7</td>
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<td>Previous therapy*</td>
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<td>0.04-6.27</td>
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<td>0.01-10.9</td>
<td>0.05-14.3</td>
</tr>
<tr>
<td>Median disease duration, years</td>
<td>65</td>
<td>77</td>
<td>17</td>
<td>59</td>
<td>76</td>
</tr>
<tr>
<td>Median leukocyte count, ×10^9/L</td>
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<td>8.2</td>
<td>6.4</td>
<td>7.3</td>
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<td>Leukocyte count</td>
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<td>3.1-50.4</td>
<td>3.2-50</td>
<td>3.1-20.5</td>
<td>2.7-25.8</td>
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<tr>
<td>Median platelet count, ×10^9/L</td>
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<td>190</td>
<td>232</td>
<td>209</td>
<td>201</td>
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<tr>
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<td>52</td>
<td>14</td>
<td>36</td>
<td>52</td>
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<tr>
<td>Abnormal cytogenetics†</td>
<td>21</td>
<td>47</td>
<td>5</td>
<td>12</td>
<td>6</td>
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</table>

**NOTE.** Please see text for additional information regarding treatment dose and schedule for both pomalidomide and prednisone. Abbreviations: MF, myelofibrosis; ET, essential thrombocythemia; PV, polycythemia vera.

*Please note that the number of patients does not always add up since some patients received both cytotoxic and non-cytotoxic therapy.

†Cytogenetic information was available in only 47 patients.

This study did not include patients with platelet counts of lower than 50 × 10^9/L or absolute neutrophil counts of lower than 1 × 10^9/L; therefore, the drug's effect on clinically relevant thrombocytopenia or neutropenia could not be accurately assessed. However, a greater than 50% increase in platelet count was recorded in six (43%) of 14 patients who received pomalidomide (0.5 or 2 mg/d) with or without prednisone and had baseline platelet counts in the range of 50 to 100 × 10^9/L (baseline platelet counts of 64 to 86 × 10^9/L increased to peak levels of 148 to 293 × 10^9/L). Unlike our previous experience with lenalidomide, serum lactate dehydrogenase level was seldom affected by pomalidomide with or without prednisone therapy (normalized in only two responders) and treatment-induced changes in leukocytosis were infrequent (data not shown).
examined. Granulocyte DNA-derived JAK2 V617F allele burden was measured in four responding patients at baseline and after variable follow-up time; in one of these patients who showed an increase in her hemoglobin to 16 g/dL (Table 2; patient 6), mutant allele burden increased from 18% to 48% after 1 year of treatment; patient 7 from 21% to 3.3% after 1 year of treatment; patient 8 from 9% to 9% after 5 months of treatment; patient 9 from 3% to 3.3% after 1 year of treatment; patient 7 from 21% to 3% after 1 year of treatment; patient 12 from 18% to 48% after 1 year of treatment; patient 13 from 18% to 48% after 1 year of treatment; patient 14 from 18% to 48% after 1 year of treatment; patient 15 from 18% to 48% after 1 year of treatment; patient 16 from 18% to 48% after 1 year of treatment; patient 17 from 18% to 48% after 1 year of treatment; patient 18 from 18% to 48% after 1 year of treatment; patient 19 from 18% to 48% after 1 year of treatment; patient 20 from 18% to 48% after 1 year of treatment).

### Treatment Toxicity

At the time of this writing, all patients still on-study have completed six cycles of treatment. Twenty-two patients (26%) discontinued treatment before the completion of three cycles because of, according to documents provided by the treating physician, lack of efficacy/progressive disease in nine patients, adverse events in six patients, withdrawal of consent in four patients and death unrelated to drug therapy in three patients. A total of eight deaths were documented during protocol therapy and were attributed to progressive...
This study identifies pomalidomide, at a dose level of 0.5 to 2 mg/d, with or without a short course of concomitant prednisone therapy, as a well-tolerated drug in patients with myelofibrosis. The study also demonstrates the drug’s erythropoietic activity, especially in the absence of leukocytosis. The observed anemia response rates of up to 40% are particularly notable considering the study’s utilization of the strict IWG-MRT criteria for response and the fact that 81% of the responders were RBC transfusion dependent at baseline. By comparison, using single-agent thalidomide or lenalidomide in myelofibrosis, we have previously reported anemia response rates of 20% and 22%, respectively.12,18 The overall findings from this study also suggest that pomalidomide at the lower dose level (0.5 mg/d), combined with a short course of prednisone therapy, might be as effective as the drug at the higher dose level (2 mg/d), and possibly less toxic. Furthermore, the responses seen at the lower pomalidomide dose level were maintained for several months beyond the discontinuation of concomitant prednisone therapy and are thus likely the result of pomalidomide and not prednisone effect. However, this study was not designed or adequately powered to either compare the different treatment arms or clarify the additional value of prednisone during pomalidomide therapy in myelofibrosis. After all, it is possible that prednisone can modify the effect of IMiDs immunomodulatory compounds by suppressing inflammatory stimuli. Therefore, additional studies are required to validate single-agent pomalidomide activity at 0.5 mg/d.

The underlying mechanism for the salutary effect of pomalidomide for myelofibrosis-associated anemia is not readily apparent and the available data on the drug’s effect on normal erythropoiesis are not always consistent. For example, a recent study showed that both pomalidomide and lenalidomide favorably modulated human erythropoiesis by promoting survival of erythroid progenitors and enhancing fetal hemoglobin expression.25 These observations are somewhat at odds with a previous study where pomalidomide suppressed erythroid colony formation and instead increased the frequency of myeloid colonies.26 Regardless, these in vitro observations do not necessarily recapitulate in vivo drug effects that take into account the presence of clonal erythropoiesis and an aberrant cytokine milieu. Pomalidomide is known to upregulate interferon-γ, interleukin (IL) -2, IL-5, and IL-10 and downregulate TNF-α, IL-1β, IL-11, and IL-12 and the composite effect of such cytokine modulation might be favorable in terms of ineffective erythropoiesis and over-ride any direct...
In this regard, it is important to mention that the anti-TNF-α activity of pomalidomide is much more potent than that of both thalidomide and lenalidomide; TNF-α markedly and directly inhibits human erythropoiesis. Furthermore, it is possible that IMiDs immunomodulatory compounds promote opposing effects on normal and clonal cells. The latter scenario provides an explanation for lenalidomide’s effect in restoring normal blood counts while eradicating the del(5q) clone in patients with myelodysplastic syndrome and for our observation of a patient who experienced a robust increase in hemoglobin level while achieving a partial cytogentic remission.

For evaluating predictors of anemia response, we considered all patients receiving pomalidomide with or without prednisone (n = 63). Multiple parameters including myelofibrosis subtype, JAK2V617F mutational status, cytogentic findings, leukocyte and platelet counts, spleen size, duration of disease, disease stage, and presence or absence of RBC transfusion history at baseline. On multivariable analysis, only leukocytosis, which was often associated with marked splenomegaly, came out as a significant negative predictor of response. It is possible that anemia in myelofibrosis patients with leukocytosis or marked splenomegaly is etiologically different and less amenable to treatment with IMiDs immunomodulatory compound (ie, it might reflect more a clone-intrinsic rather than cytokine-mediated ineffective erythropoiesis). It is also possible that the benefit of pomalidomide in patients with leukocytosis or marked splenomegaly was undermined by the dynamics of their disease progression, which might have necessitated intervention with alternative therapy and premature interruption of the study drug; unlike our previous experience with lenalidomide, and to some degree with thalidomide, pomalidomide had little effect on splenomegaly. Therefore, considering the fact that the drug exerted minimal myelosuppression, it is reasonable to consider additional studies that allow combination therapy with pomalidomide and a cytoreductive drug.

We were particularly encouraged by the relatively low incidence of grade ≥ 3 neutropenia and thrombocytopenia associated with pomalidomide therapy: 5% and 9% at the lower 0.5 mg/d dose level and 9% to 16% and 14% to 16% at the higher 2 mg/d dose levels. These figures compare favorably to those seen in our previous study of lenalidomide therapy (10 mg/d) in myelofibrosis; 40% and 24%, respectively. However, it is possible that higher doses of pomalidomide could produce a similar degree of myelosuppression, and in this regard, it is important to recall that the drug causes dose-dependent myelosuppression in multiple myeloma. Moreover, it has been shown that increased JAK2V617F allele burden. Finally, additional studies are needed to evaluate safety and efficacy of the drug at the lower dose level (eg, 0.5 mg/d) in patients with baseline platelet counts of lower than 50 × 10^9/L and absolute neutrophil count of lower than 1 × 10^9/L.

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**Consultant or Advisory Role:** B. Nebiyou Bekele, Celgene Corporation (C)
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