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Recombinant Interferon-Beta1a Use in Six Patients with Myeloproliferative Neoplasms: A First Impression

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Dear Editor;

Recombinant and pegylated interferon-alpha2 (IFN-α2) have a long history of off-label use in patients with Philadelphia-negative chronic myeloproliferative neoplasms (MPNs).[1] While preclinical evidence suggests potential anti-cancer activity for rIFN-β1a (recombinant interferon-beta1a)[2], clinical experience with this agent in MPNs is lacking, with no prior reports of its use in this setting to the best of our knowledge. rIFN-β1a, while a well-established therapy for multiple sclerosis, remains understudied in MPNs. Here, we present our initial observations on the safety profile of rIFN-β1a in six MPN patients who transitioned to this therapy.

Six MPN patients, previously treated with off-label pegylated r-IFNα-2a due to unavailability, transitioned to off-label r-IFNβ-1a and were retrospectively evaluated. Patients were followed for a median of 35.3 months after MPN diagnosis (30.8 months under r-IFNα-2a and 16.8 months under r-IFNβ-1a). Demographic, clinical, and laboratory characteristics are summarized in the Table 1.

Table 1. Demographic, Clinical, and Laboratory Characteristics of Patients

	1	2	3	4	5	6
Age/Gender	46/F	45/M	47/M	41/F	44/M	34/F
Diagnosis	PV	PV	ET	ET	PV	ET
Mutation	JAK2	Triple negative	JAK2	CALR Type 1	JAK2	CALR Type 1
Risk Score[a]	Low	Low	Low	Low	Low	Low
The Initiation of IFN-Beta1a						
Hb (g/dL)	13.3	13.2	14.7	11.5	15.3	12.4
Htc (%)	41.6	44.3	42.8	34.5	45.3	38.2
Leu (x10^3/mm^3)	5500	11700	5900	9400	4600	8900
Plt (x10^3/mm^3)	454	272	419	774	151	606
LDH (U/L)	237	191	241	313	229	263
JAK-2 allele (%)	9.8		8		10	
Latest Visit of IFN-Beta 1a						
Hb (g/dL)	12.9	12.9	16.4	10.9	16.9	11.7
Htc (%)	38.7	41.8	50.1	32.7	51.1	37.6
Leu (x10^3/mm^3)	7570	10700	11500	13250	10300	6500
Plt (x10^3/mm^3)	666	395	721	756	510	639
LDH (U/L)	203	173	303	309	178	318
JAK2 allele (%)	3.6		1.7		0.6	
Adverse Event						
Myalgia	Grade 2	Grade 1	Grade 2	Grade 2	Grade 2	Grade 1

Hb, hemoglobin; htc, hematocrit; leu, leukocyte; plt, platelet; LDH, lactate dehydrogenase. [a]Age ≤ 60 y of age, platelets ≤ 1500 (×10^3/mm^3) and no prior majoLor thrombosis

All patients initiated r-IFNβ-1a at a dose of 44 mcg weekly. All patients reported myalgia on treatment days, requiring concomitant non-steroidal anti-inflammatory drug (NSAID) administration. This adverse event prevented dose escalation, and the initial dose remained unchanged. No other adverse events were observed,

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and no treatment discontinuations occurred due to adverse events. No new arterial or venous thrombotic events were observed during r-IFNβ-1a therapy.

Three of the six patients harbored the JAK2V617F mutation. Due to insufficient JAK2V617F measurements, assessment with an exponential response model was not feasible. However, a reduction in allele burden was observed in these three patients following the initiation of r-IFNβ-1a.

MPNs are characterized by a self-sustaining inflammatory cycle driving clonal expansion.[3,4], supporting the idea of early interferon intervention to halt disease progression.[5] While IFN-α and IFN-β share similar immunomodulatory mechanisms.[6], they also exhibit distinct characteristics, most notably IFN-β's higher receptor binding affinity.[7]

In this six-patient case series, r-IFNβ-1a demonstrated a manageable safety profile, with myalgia being the most common side effect, which limited dose escalation. While some patients showed a reduction in JAK2V617F allele burden, the small sample size prevents any definitive conclusions about its clinical significance. Further studies are needed to determine the optimal role of r-IFNβ-1a in the treatment of MPNs.

Conflict of Interest

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