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Real-World Phlebotomy (PHL) Burden and Treatment Gaps in US Patients with Polycythemia Vera (PV)

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Background

- Polycythemia vera (PV) is a myeloproliferative neoplasm associated with increased thrombotic risk and symptoms such as fatigue.[1]
- Treatment aims to maintain hematocrit (HCT) <45% using phlebotomy (PHL) and/or cytoreductive therapy (CRT) to reduce thrombotic risk.[2]
- Although PHL remains a cornerstone of PV management, patients may require frequent PHLs, often with inadequate HCT control,[3] and it may contribute to iron deficiency (ID), symptom burden, and treatment inconvenience.[4,5]
- Management of ID with iron supplementation is generally not recommended in PV, as replenishing iron stores can exacerbate erythrocytosis and increase HCT.[6]
- Real-world data describing PHL utilization, management changes, and PHL-related complications in PV across practice settings remain limited.

Objectives

- To assess PHL utilization, interruptions and change from PHL-based management including underlying reasons and associated ID, overall and by practice setting (community or academic).

Methods

- Retrospective US medical chart review of adults with newly diagnosed PV
- **Inclusion criteria**
  - Initial diagnosis of PV at any time between August 1, 2020 and July 31, 2023
  - Age ≥18 years at PV diagnosis
  - ≥24 months of follow-up available unless death occurred earlier.
- **Exclusion criteria**
  - History of acute myeloid leukemia, myelofibrosis, myelodysplastic syndrome, or stem cell transplantation prior to PV diagnosis
  - Participation in a clinical trial at any time during follow-up
- **Data collection**
  - Hematologists/oncologists from academic and community practices abstracted de-identified medical record data using a standardized electronic case report form (eCRF).
  - Data included patient characteristics, PHL and CRT utilization, treatment modifications, laboratory values, and reasons for treatment interruption or discontinuation.

Results

Patient Demographics & Clinical Characteristics

- The study included 208 patients with PV (37.5% female; median age, 63 years [IQR, 57.0-69.0]), including 108 (51.9%) treated in academic settings and 100 (48.1%) treated in community settings.
- Patients treated in community settings were older than those treated in academic settings (median age, 64.5 vs 61.5 years; p=0.042).
- While JAK2V617F positivity was common across both settings (85.6% overall), negative or unknown JAK2 mutation status was observed more frequently in community settings than academic settings (13% vs. 3.7%).
- Mean baseline HCT was 53.3 ± 5.1%; 35.6% of patients had missing baseline laboratory data. Mean baseline HCT was similar between academic and community settings.
- Geographic distribution differed by practice setting (p<0.001), with greater representation from the Northeast in academic practices (44.4%) and from the South (33.0%) and West (32.0%) in community practices.
- Community-treated patients had a higher comorbidity burden than academically treated patients (mean ± standard deviation Deyo-Charlson Comorbidity Index [DCCI], 1.3 ± 1.7 vs 0.8 ± 1.0; p=0.006).
- The most common comorbidities were hypertension (40.9%), diabetes without end-organ damage (22.1%), and chronic pulmonary disease (11.5%) (Table 1).

Table 1: Baseline Patient Demographic and Clinical Characteristics

Characteristic	Overall PV (n=208)	Academic (n=108)	Community (n=100)	p –value*
Female, n (%)	78 (37.5)	42 (38.9)	36 (36.0)	0.774
Age at index, median (IQR)	63.0 (57.0 - 69.0)	61.5 (55.7 - 67.0)	64.5 (59.0 - 70.0)	**0.042**
Race, n (%)				0.422
White	164 (78.8)	89 (82.4)	75 (75.0)	
Black/African American	27 (13.0)	11 (10.2)	16 (16.0)	
Asian	15 (7.2)	8 (7.4)	7 (7.0)	
Other race	2 (1.0)	(0.0)	2 (2.0)	
Geographic region, n (%)				**<0.001**
Northeast	68 (32.7)	48 (44.4)	20 (20.0)	
Midwest	35 (16.8)	20 (18.5)	15 (15.0)	
South	54 (26.0)	21 (19.4)	33 (33.0)	
West	51 (24.5)	19 (17.6)	32 (32.0)	
JAK2 mutation status, n (%)				0.076
JAK2V617F+	178 (85.6)	98 (90.7)	80 (80.0)	
Exon 12+	13 (6.3)	6 (5.6)	7 (7.0)	
Negative or unknown	17 (8.2)	4 (3.7)	13 (13.0)	
History of TEs, n (%)	13 (6.3)	7 (6.5)	6 (6.0)	1.000
History of major bleeding, n (%)	3 (1.4)	3 (2.8)	0 (0)	0.273
Deyo-CCI, mean ± SD	1.1 ± 1.4	0.8 ± 1.0	1.3 ± 1.7	**0.006**
Common comorbidities, n (%)				
Hypertension	85 (40.9)	44 (40.7)	41 (41.0)	1.000
Diabetes without end-organ damage	46 (22.1)	19 (17.6)	27 (27.0)	0.143
Chronic pulmonary disease	24 (11.5)	11 (10.2)	13 (13.0)	0.676
Laboratory values [a]				
HCT (%), mean ± SD	53.3 ± 5.1	53.7 ± 4.3	52.8 ± 5.9	0.295
WBC (10^3/µL), mean ± SD	15.2 ± 13.0	14.3 ± 11.2	16.5 ± 15.0	0.359
Platelets (10^3/µL), mean ± SD	404.7 ± 172.4	378.4 ± 180.6	439.3 ± 155.8	**0.038**
Missing baseline labs, n (%)	74 (35.6)	32 (29.6)	42 (42.0)	0.086

IQR, interquartile range; JAK2, Janus kinase 2; PV, polycythemia vera, SD, standard deviation; TE, thromboembolic events; CCI, Charlson-Comorbidity Index; HCT, hematocrit; WBC, white blood cells.*Bivariate comparisons were conducted using Chi-square/Fisher's exact tests (categorical) and t-tests/Wilcoxon rank-sum tests (continuous). [a] Baseline laboratory values were measured ±60 days of initial PV diagnosis.

Figure 1: Initial Planned Frequency of PHL

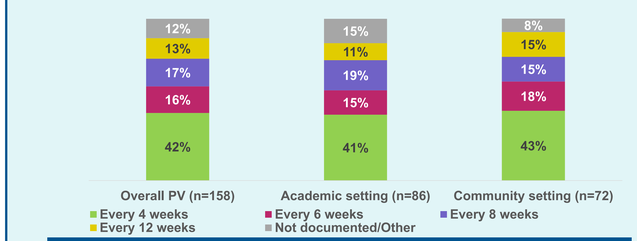
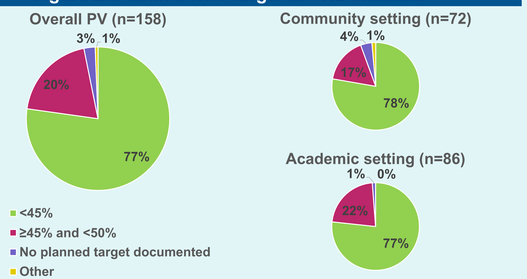


Figure 2: Planned HCT target at PHL initiation



HCT, hematocrit; PHL, phlebotomy; PV, polycythemia vera

Figure 3: Reasons for Pauses/Interruptions in PHL

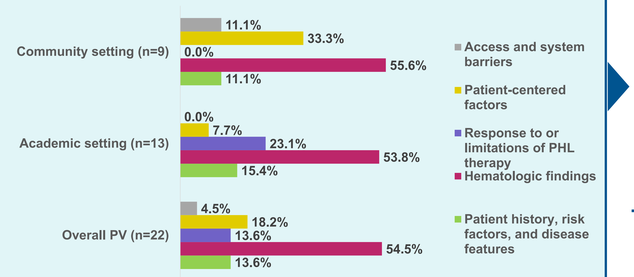
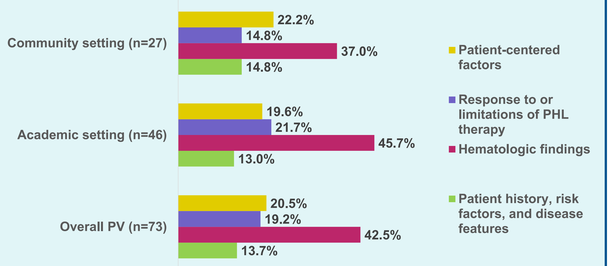


Figure 4: Reasons for Change of Management from PHL



Reason categories were defined as: **patient history, risk factors, and disease features** (e.g., thrombosis, splenomegaly, fatigue, cognitive impairment); **hematologic findings** (e.g., anemia, ID, leukocytosis, thrombocytosis, inadequate HCT control); **response to or limitations of PHL therapy** (e.g., intolerance, poor venous access, dehydration, dizziness, fainting, lack of symptom improvement); **patient-centered factors** (e.g., patient request for CRT, preference to discontinue or pause PHL, treatment burden, nonadherence); and **access/system barriers** (e.g., transportation challenges, insurance loss, or unsustainable out-of-pocket costs). Multiple reasons could be selected.

CRT, cytoreductive therapy; HCT, hematocrit; PHL, phlebotomy; PV, polycythemia vera

PHL Utilization (N=158)

- After a median follow-up of 2.3 years (IQR, 2.1-3.2), 158 patients (76.0%) received PHL, including 57.6% who received concurrent CRT.
- PHL use was similar across practice settings (community, 72.0%; academic, 76.0%; p=0.261), as was the mean PHL rate during periods without CRT (6.7 vs 7.0 procedures per patient-year; p=0.619).
- The most common planned PHL frequency was every 4 weeks (41.8%), followed by every 8 weeks (17.1%), every 6 weeks (16.5%), and every 12 weeks (12.7%); 12.0% had another or undocumented schedule (Figure 1).
- At PHL initiation, 77.2% of patients had an HCT target <45%, 19.6% had a target ≥45% to <50%, and 2.5% had no documented HCT target (Figure 2).
- During PHL treatment periods without cytoreductive therapy, 107 patients had an HCT measurement, and among those, 95.3% had at least one HCT >45%.

PHL Pauses/Interruptions (N=22)

- PHL pauses/interruptions occurred in 22 patients receiving PHL overall (13.9%), 9 (12.5%) in community settings only, and 13 (15.1%) in academic settings only.
- Overall, the most common reason for the first PHL pause/interruption was anemia (27.3%), ID (18.2%) and patient preference to pause or discontinue (13.6%) (**Figure 3**).

Change from PHL-based Management (N=73)

- Overall, 73 patients (35.1%) changed from PHL-based management. The most common reasons were inadequate HCT control (18%), patient request for CRT (14%), and patient request to stop PHL (12%) (Figure 4).
- At the time of change from PHL-based management, 47% were receiving concurrent CRT, 45% subsequently initiated a new CRT (most commonly hydroxyurea [42%], ruxolitinib [36%], or ropeginterferon alfa-2b [21%]), and 15% did not receive CRT.

Poor PHL Response/Tolerance (N=46)

- Poor PHL response/tolerance was observed in 46 (29.1%) PHL-treated patients, with a mean time to change in PHL-based management of 320 days.
- Following change in PHL-based management due to poor response/tolerance, 52.2% initiated CRT (hydroxyurea, 26.1%; ruxolitinib, 17.4%; ropeginterferon alfa-2b, 8.7%), while 47.8% had no subsequent treatment observed.

Table 2: PHL Related ID Among PHL Treated Patients

Measure	Overall PV (n=158)	Academic (n=86)	Community (n=72)	p-value
ID related to PHL, n (%)				0.085
Yes	32 (20.3)	23 (26.7)	9 (12.5)	
No	98 (62.0)	49 (57.0)	49 (68.1)	
Unknown/not assessed	28 (17.7)	14 (16.3)	14 (19.4)	
Time to ID onset from PHL initiation (in days), Median (95% CI)	555 (309 - NA)	385 (185 - NA)	634 (555 - NA)	0.400
Action taken for ID, n (%)				
Hold PHL	11 (34.4)	8 (34.8)	3 (33.3)	1.000
Switch to CRT				0.107
Hydroxyurea	10 (31.2)	9 (39.1)	1 (11.1)	
Ruxolitinib	1 (3.1)	0 (0.0)	1 (11.1)	
Iron supplementation	12 (37.5)	9 (39.1)	3 (33.3)	1.000
None	6 (18.8)	2 (8.7)	4 (44.4)	
Symptoms from ID, n (%)	32 (20.3)	23 (26.7)	9 (12.5)	0.085
Anemia	10 (31.2)	7 (30.4)	3 (33.3)	1.000
Cognitive impairment	5 (15.6)	3 (13.0)	2 (22.2)	0.919
Fatigue	25 (78.1)	20 (87.0)	5 (55.6)	0.145
Dizziness	12 (37.5)	10 (43.5)	2 (22.2)	0.477
Itching/pruritus	5 (15.6)	3 (13.0)	2 (22.2)	0.919
None	5 (15.6)	1 (4.3)	4 (44.4)	**0.023**

CI, confidence interval. Time from ID onset from PHL was estimated using Kaplan-Meier methods from time from PHL initiation to onset, censoring patients at loss to follow-up or discontinuation. Bivariate comparisons were conducted using Chi-square/Fisher's exact tests (categorical) and t-tests/Wilcoxon rank-sum tests (continuous).

PHL-related ID (N=32)

- PHL-related ID occurred in 20.3% of PHL-treated patients (academic, 26.7%; community, 12.5%; p=0.085).
- Fatigue was reported in 78.1% of patients during the first ID episode.
- Management of the first ID episode most commonly included iron supplementation (37.5%) and pausing PHL (34.4%), with similar patterns across practice settings (Table 2).

Conclusions

- PHL remains widely used in routine PV care across clinical practice settings, with high reliance and frequent management changes despite available CRT options.
- Substantial PHL burden and inadequate HCT control as a driver of management change were observed.
- PHL-related ID was common and frequently associated with fatigue and other symptoms, highlighting the clinical burden of ongoing PHL reliance in routine PV management.
- Despite current literature recommending against iron supplementation,[5] a notable portion of patients received iron supplementation in response to PHL-related ID.
- These findings highlight a need for more effective, better-tolerated treatments that reduce PHL reliance, achieve and maintain HCT control, address the burden of PHL-related iron deficiency, and support consistent care across settings.

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Disclosures

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