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# Basophilia and eosinophilia in primary myelofibrosis: phenotype, genotype, and prognostic correlates



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**Dear Editor,**

Primary myelofibrosis (PMF) is a myeloid neoplasm that is currently classified in the category of *JAK2* mutation-prevalent myeloproliferative neoplasms (JAK2-MPNs) [1]; other members of JAK2-MPNs include essential thrombocythemia (ET) and polycythemia vera (PV). JAK2-MPNs are characterized molecularly by JAK-STAT activating mutations, involving *JAK2*, *CALR*, and *MPL* genes, and morphologically by trilineage myeloid proliferation in the bone marrow (BM) that is accentuated by megakaryocyte proliferation and atypia [2]. Peripheral blood (PB) manifestations of JAK2-MPNs include leukocytosis, thrombocytosis, and/or erythrocytosis while other disease features include splenomegaly, thrombosis, bleeding, microvascular disturbances, pruritus, and constitutional symptoms. Patients with MPN are at risk for premature death and disease progression into a fibrotic or leukemic disease phase [3]. Disease complications in JAK2-MPNs are most severe in PMF where median survival is estimated at 4.4 years and leukemic progression at 9%, at a median follow-up of 3.2 years [4].

Leukocytosis, in general, has long been identified/suspected as a risk factor for a number of disease complications in JAK2-MPNs including overall and leukemia-free survival [5, 6], disease progression [7, 8], thrombosis risk [9–11], and extramedullary hematopoiesis [12]. Considering the multicomponent nature of leukocytes, more recent studies in JAK2-MPNs have appropriately looked into the differential prognostic impact of absolute neutrophil (ANC) [13–15], monocyte (AMC) [13, 16–19], and lymphocyte (ALC) counts [13–15]. By comparison, fewer studies have reported on the prognostic contribution of absolute basophil (ABC) or eosinophil (AEC) counts in JAK2-MPNs, in general, and in PMF, in particular, not associated with tyrosine kinase fusion genes [20–24]. On the other hand, the prognostic relevance of basophilia in chronic myeloid leukemia (CML) is well established and is taken under consideration in defining accelerated phase CML [25, 26]. In the current study, we utilized a large Mayo Clinic database of patients with PMF in order to describe the prevalence and the clinical, molecular, and prognostic correlates of ABC and AEC.

The current retrospective study was conducted under an institutional review board (Mayo Clinic -IRB: 12-003574) approved minimum risk protocol that allowed retrospective collection and analysis of data from patient records. Mutations/variants were screened by multi-gene next-generation sequencing in accordance with institutional protocols for clinical use. Study patients were retrospectively identified, from Mayo Clinic databases of patients with PMF, based on availability of information on ABC and AEC, at time of diagnosis of first referral. Cytogenetic results were reported according to the International System for Human Cytogenetic Nomenclature [27]. Diagnostic criteria were according to the International Consensus Criteria (ICC) [1]. Statistical analyses considered clinical and laboratory data collected at the time of initial diagnosis/referral, confirmed by bone marrow examination. Receiver operating characteristic (ROC) curve analysis, for predicting survival at the timepoint of 5 years, was utilized to determine the optimal cutoff points for ABC and AEC. Cox regression analysis was applied to identify risk factors for overall (OS) and leukemia-free (LFS) survival. The Kaplan–Meier method was used to construct time-to-event curves, which were compared by the log-rank test. Statistical analyses were conducted using JMP Pro 17.0.0 software (SAS Institute, Cary, NC, USA).

We identified a cohort of 411 patients with PMF in whom baseline ABC and AEC data were available at time of diagnosis/first referral (median age 64 years, range 22–87; 65% males). Patient characteristics are outlined in Table 1. Risk stratification by the dynamic international prognostic scoring system (DIPSS)-plus model [28] was 11% low, 17% intermediate-1, 39% intermediate-2, and 32% high. Risk distributions by exclusively genetics-based IPSS (GIPSS) [29] and mutation and karyotype enhanced IPSS (MIPSSv2) [30] are also outlined in Table 1. Median (range) values were ABC $0.13 \times 10^9$/L (0-23.9), AEC $0.12 \times 10^9$/L (0-19.6), hemoglobin 11.3 g/dL (7.9-16.1), leukocyte count $9.6 \times 10^9$/L (1.0-218.5), and platelet count $212 \times 10^9$/L (11-2466). Driver mutation distribution was *JAK2* 60%, *CALR* 23% and *MPL* 7%. NGS information was available in variable fractions of patients (Supplementary Table 1) and most frequent other mutations included *ASXL1* 40%, *TET2* 18%, and *SRSF2* 15%.

ROC analysis-determined optimal cutoff values for ABC and AEC were $0.8 \times 10^9$/L (12% of patients above the threshold) and $0.6 \times 10^9$/L (13% of patients above the threshold), respectively. Furthermore, to examine the additional prognostic impact of "marked" basophilia, we also considered an operational ABC cutoff value of $2 \times 10^9$/L, as at least 3 x upper limit of the normal reference range (Table 1 and Supplementary Table 1). ABC and AEC were highly correlated ($p < 0.01$) with a correlation coefficient of 0.91 (95% CI: 0.89–0.92); 24 (46%) of 52 patients with an AEC > $0.6 \times 10^9$/L displayed an ABC > $0.8 \times 10^9$/L while 24 of 49 (49%) patients with an ABC > $0.8 \times 10^9$/L displayed an AEC > $0.6 \times 10^9$/L (Fisher's exact test, $p < 0.01$). It was, therefore, not surprising to see significant association between both ABC > $0.8 \times 10^9$/L and AEC > $0.6 \times 10^9$/L and higher leukocyte count, and higher peripheral blood blast percentage (Table 1). In addition, ABC > $0.8 \times 10^9$/L was associated with older age and higher disease stage measured by DIPSS-plus. In terms of molecular associations, AEC > $0.6 \times 10^9$/L appeared to cluster with *JAK2* mutation, and both AEC > $0.6 \times 10^9$/L and ABC > $0.8 \times 10^9$/L were less likely to be associated with Type 1/like *CALR* mutation (Table 1); also, a significant association was noted between ABC > $0.8 \times 10^9$/L and *ASXL1* mutation ($p = 0.03$) and between AEC > $0.6 \times 10^9$/L and *SH2B3* mutation ($p = 0.04$; Supplementary Table 2). Similar analyses using the ABC threshold of $2 \times 10^9$/L did not reveal any molecular association (Supplementary Table 1)

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**Table 1.** Demographic, clinical and laboratory characteristics of 411 patients with primary myelofibrosis stratified by absolute basophil count (ABC) and absolute eosinophil count (AEC).

| | All patients | Basophils ≤0.8 X10$^9$/L | Basophils > 0.8 X10$^9$/L | *P* value | Basophils < 2 X10$^9$/L | Basophils ≥ 2 X10$^9$/L | *P* value | Eosinophils ≤0.6 X10$^9$/L | Eosinophil >0.6 X10$^9$/L | *P* value |
|---|---|---|---|---|---|---|---|---|---|---|
| **Number (%)** | 411 | 362 (88) | 49 (12) | | 396 (96) | 15 (4) | | 359 (87) | 52 (13) | |
| **Age (years, median)** | 64 | 63 | 67 | **0.01** | 64 | 66 | 0.2 | 64 | 65 | 0.2 |
| **Range** | 22–87 | 22–84 | 46–87 | | 22–87 | 49–72 | | 22–87 | 35–81 | |
| **Female, *n* (%)** | 145 (35) | 132 (36) | 13 (27) | 0.2 | 142 (36) | 3 (20) | 0.2 | 129 (36) | 16 (31) | 0.5 |
| **Male, *n* (%)** | 266 (65) | 230 (64) | 36 (73) | | 254 (64) | 12 (80) | | 230 (64) | 36 (69) | |
| **Palpable splenomegaly, *n* (%)** | 301 (75) | 263 (74) | 38 (79) | 0.4 | 291 (75) | 10 (71) | 0.8 | 265 (75) | 36 (72) | 0.6 |
| **Hemoglobin, g/dL; median** | 11.3 | 11.2 | 11.5 | 0.9 | 11.3 | 10.7 | 0.6 | 11.2 | 11.6 | 0.06 |
| **Range (evaluable *n* = 332)** | 7.9–16.1 | 7.9–16.1 | 8.2–14.9 | | 7.9–16.1 | 8.2–12.8 | | 7.9–16.0 | 8.2–16.1 | |
| **While blood cell, x10$^9$/L; median** | 9.6 | 8.4 | 31 | **< 0.01** | 9.1 | 61 | **< 0.01** | 8.3 | 30.3 | **< 0.01** |
| **Range(evaluable *n* = 404)** | 1.0–218.5 | 1.0–120.6 | 3.9–218.5 | | 1.0–120.6 | 7.9–218.5 | | 1.0–112.4 | 4.8–218.5 | |
| **Absolute Basophil count,** | 0.13 | 0.1 | 1.5 | | 0.1 | 3.4 | | 0.1 | 0.7 | |
| **X10$^9$/L, median (Range)** | (0–23.9) | 0–0.8 | (0.84–23.9) | | (0–1.8) | (2–23.9) | | (0–2.9) | (0–23.9) | |
| **Absolute Eosinophil count,** | 0.12 | 0.1 | 0.6 | | 0.1 | 2.9 | | 0.09 | 1.3 | |
| **x10$^9$/L, median (Range)** | (0–19.6) | (0–2.7) | (0–19.6) | | (0–3.4) | (0–19.6) | | (0–0.6) | (0.6–19.6) | |
| **Platelets, x10$^9$/L; median** | 212 | 213 | 207 | 0.4 | 213 | 165 | 0.2 | 206 | 277 | 0.06 |
| **Range** | 11–2466 | 11–2282 | 14–2466 | | 11–2466 | 14–885 | | 11–2466 | 14–885 | |
| **Peripheral blasts, %; median** | 1 | 1 | 2 | **< 0.01** | 1 | 1 | 0.2 | 1 | 1 | **0.02** |
| **Range** | 0–15 | 0–15 | 0–13 | | 0–15 | 0–13 | | 0–15 | 0–13 | |
| Peripheral blasts ≥ **2%, *n* (%)** | 131 (32) | 104 (29) | 27 (55) | | 124 (31) | 7 (47) | | 107 (30) | 24 (46) | |
| **Lactate dehydrogenase U/L, median** | 516 | 511 | 669 | **0.03** | 516 | 1148 | **0.02** | 515 | 565 | 0.4 |
| **Range (evaluable *n* = 225)** | 136–1961 | 136–1901 | 230–1961 | | 136–1901 | 294–1961 | | 136–1433 | 138–1961 | |
| **DIPPS-Plus** | | | | | | | | | | |
| **Low, *n* (%)** | 47 (11) | 47 (13) | 0 (0) | | 47 (12) | 0 (0) | | 41 (11) | 6 (11.5) | |
| **Intermediate-1, *n* (%)** | 69 (17) | 68 (19) | 1 (2) | **< 0.01** | 68 (17) | 1 (7) | **0.02** | 63 (18) | 6 (11.5) | 0.3 |
| **Intermediate-2, *n* (%)** | 162 (39) | 140 (39) | 22 (45) | | 158 (40) | 4 (27) | | 144 (40) | 18 (35) | |
| **High, *n* (%)** | 133 (32) | 107 (29) | 26 (53) | | 123 (31) | 10 (67) | | 111 (31) | 22 (42) | |

**Table 1.** continued

| | All patients | Basophils ≤0.8 X10$^9$/L | Basophils > 0.8 X10$^9$/L | *P* value | Basophils < 2 X10$^9$/L | Basophils ≥ 2 X10$^9$/L | *P* value | Eosinophils ≤0.6 X10$^9$/L | Eosinophil >0.6 X10$^9$/L | *P* value |
|---|---|---|---|---|---|---|---|---|---|---|
| **GIPSS (evaluable *N* = 305)** | | | | | | | | | | |
| **Low, *n* (%)** | 31 (9) | 30 (10) | 1 (3) | | 31 (9) | 0 (0) | | 20 (10) | 1 (2) | |
| **Intermediate, *n* (%)** | 132 (38) | 122 (40) | 10 (25) | **0.04** | 130 (39) | 2 (13) | **0.03** | 119 (39) | 13 (31) | 0.06 |
| **High, *n* (%)** | 138 (40) | 116 (38) | 22 (55) | | 128 (39) | 10 (67) | | 114 (38) | 24 (57) | |
| **Very high risk, *n* (%)** | 44 (13) | 37 (12) | 7 (17) | | 41 (12) | 3 (20) | | 40 (13) | 4 (10) | |
| **MIPPS70+ V 2.0 (evaluable *n* = 341)** | | | | **< 0.01** | | | **< 0.01** | | | 0.1 |
| **Low, n (%)** | 71 (21) | 69 (23) | 2 (5) | | 71 (22) | 0 (0) | | 65 (22) | 6 (14) | |
| **Intermediate-1, *n* %)** | 63 (19) | 61 (20 | 2 (5) | | 63 (19) | 0(0) | | 59 (20) | 4 (10) | |
| **Intermediate-2, *n* (%)** | 158 (46) | 132 (44) | 26 (65) | | 147 (45) | 11 (73) | | 135 (45) | 23 (55) | |
| **High, n (%)** | 49 (14) | 39 (13) | 10 (25) | | 45 (14) | 4 (27) | | 40 (13) | 9 (21) | |
| ***JAK2 V617F* mutated, *n* (%)** | 249 (60.0) | 215 (59) | 34 (69) | 0.2 | 239 (60) | 10 (67) | 0.6 | 209 (58) | 40 (77) | **< 0.01** |
| ***MPL* mutated, *n* (%)** | 29 (7) | 28 (8) | 1 (2) | 0.09 | 28 (7) | 1 (7) | 1.0 | 27 (7) | 2 (4) | 0.3 |
| *CALR* **mutated, *n* (%)** | 90 (23) | 74 (23) | 6 (12) | 0.06 | 89 (22) | 1 (7) | 0.1 | 87(23) | 3 (5) | **< 0.01** |
| **Type 1/like *CALR* mutated, *n* (%)** | 73 (18) | 69 (19) | 4 (8) | **0.04** | 72 (18) | 1 (7) | 0.2 | 71 (20) | 2 (4) | **< 0.01** |
| **Follow up in months,** | | | | | | | | | | |
| **median** | 52 | 60 | 26 | **< 0.01** | 58 | 24 | **< 0.01** | 59 | 34 | **< 0.01** |
| **Range** | 0–453 | 0–453 | 2–197 | | 0–453 | 2-53 | | 0–453 | 2–284 | |
| **Allogeneic stem cell transplants; *n* (%)** | 28 (7) | 26 (7) | 2 (4) | 0.4 | 27 (7) | 1 (7) | 1.0 | 25 (7) | 3 (6) | 0.7 |
| **Leukemic transformations; *n* (%)** | 45 (11) | 40 (11) | 5 (10) | 0.9 | 42 (11) | 3 (20) | 0.3 | 40 (11) | 5 (10) | 0.7 |
| **Deaths; *n* (%)** | 335 (82) | 287 (79) | 48 (98) | **< 0.01** | 320 (81) | 15 (100) | **0.01** | 288 (80) | 47 (90) | 0.06 |

Bold indicates statistically significant difference

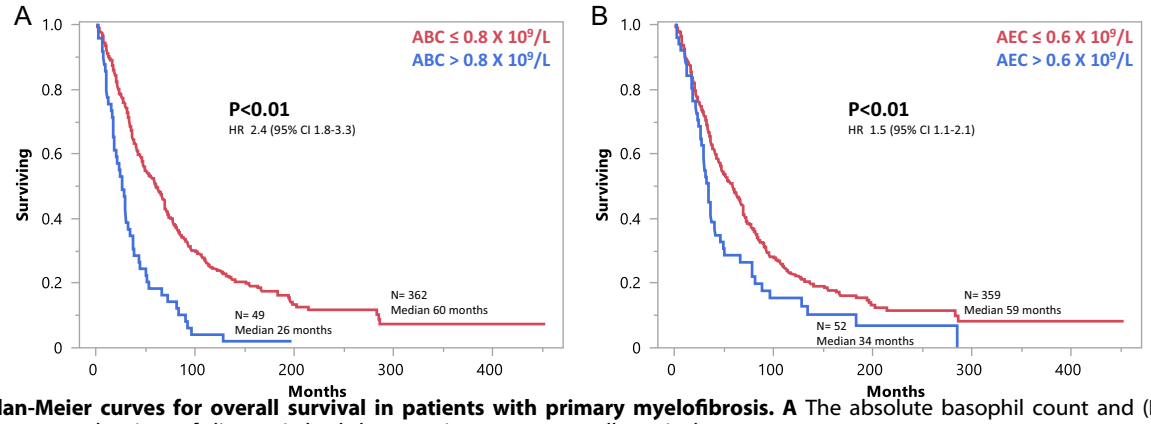


**Fig. 1 Kaplan-Meier curves for overall survival in patients with primary myelofibrosis. A** The absolute basophil count and (**B**) absolute eosinophil count at the time of diagnosis both have an impact on overall survival.

while the clinical associations were similar to those using the lower threshold of $0.8 \times 10^9$/L (Table 1).

Median follow-up was 52 months (range 0–453). During this period, 335 (82%) deaths, 45 (11%) leukemic transformations (LT), and 28 (7%) ASCTs were documented. Kaplan-Meier assessed median OS was 52 months (95% CI 45–63). In univariate analysis, OS was inferior with ABC > $0.8 \times 10^9$/L (median 26 vs. 60 months; HR 2.4; $p < 0.01$; Fig. 1a) and with AEC > $0.6 \times 10^9$/L (median 34 vs. 59 months; HR 1.5; $p < 0.01$; Fig. 1b). In multivariable analysis that included both ABC > $0.8 \times 10^9$/L and AEC > $0.6 \times 10^9$/L, the former ($p < 0.01$) but not the latter ($p = 0.33$) sustained significance. In addition, ABC > $0.8 \times 10^9$/L remained significant in multivariable analysis against DIPSS-plus (p < 0.01), GIPSS ($p < 0.01$), and MIPSSv2 ($p < 0.01$). ABC > $0.8 \times 10^9$/L remained significant ($p < 0.01$) in yet another multivariable analysis that included *ASXL1*, *SRSF2*, and *U2AF1*Q157 mutations and unfavorable karyotype; similar results were obtained when ABC > $0.8 \times 10^9$/L was substituted by ABC ≥ $2.0 \times 10^9$/L. In regard to LFS, univariate analysis showed an association with ABC ≥ $2.0 \times 10^9$/L (HR 3.7, 95% CI 1.1–12.4; $p = 0.03$) but not ABC > $0.8 \times 10^9$/L ($p = 0.3$) or AEC > $0.6 \times 10^9$/L ($p = 0.7$). Furthermore, the significant association displayed by ABC ≥ $2.0 \times 10^9$/L, in regard to LFS, was lost during multivariable analysis that included PB blasts ≥2% ($p = 0.1$), *SRSF2* (p = 0.2) or *ASXL1* ($p = 0.08$) mutation, or unfavorable karyotype ($p = 0.1$).

The current study suggests that basophilia in PMF, but not eosinophilia, is closely aligned with high-risk disease and might be detrimental to OS, but not necessarily to LFS. We are intrigued by the consistent independent effect on OS, in the context of contemporary risk models for PMF. It has been postulated that TGF-β secreted by megakaryocytes promotes basophil differentiation and proliferation via IL-3, as well as increased fibrosis, making basophilia a potential marker of advanced disease [21]. Our observations are somewhat consistent with a recent report from Yuen et al. [21] that included 195 patients with a spectrum of myeloproliferative neoplasms (MPNs), including 45 patients with overt myelofibrosis (MF) and 16 with pre-fibrotic MF. In the particular study [21], the overall prevalence of basophilia (defined by an ABC of > $0.3 \times 10^9$/L) was 22% and was higher in primary or secondary MF (35%), compared to ET/PV (8%); basophilia was associated with older age and higher leukocyte count, more frequent *JAK2* and less frequent *CALR* mutations, as was the case in the current study. More importantly, patients with basophilia also displayed significantly shorter OS and LFS, even after exclusion of patients with ET or PV [21]; in multivariable analysis that was restricted to patients with PMF ($N = 61$), the authors were able to show an adverse impact from basophilia, on both OS and LFS, independent of MIPSSv2 [21].

Two other studies have also reported on the adverse impact of basophilia in PMF [20, 23]. In one retrospective study of 58 cases [20], the authors used ROC analysis to define basophilia at an ABC of > $0.1 \times 10^9$/L and showed a DIPSS [31]-independent adverse impact on OS. In the second study [23], the authors identified 32 patients with acute myeloid leukemia (AML) arising from a previous diagnosis of MF and compared them to a control group of MF patients who did not develop AML and showed persistent basophilia and more frequent *CALR* mutation in the former. Taken together, the observations from the current study and those of the aforementioned reports warrant additional studies to prospectively clarify the prognostic impact of basophilia in PMF and related MPNs and standardize the prognostically optimal ABC cutoff level and examine the additional impact from PB basophil percentage.

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## DATA AVAILABILITY

By email request to the corresponding author.

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## AUTHOR CONTRIBUTIONS

SD, SF, PF, and AT were involved in study design, gathering and analysis of data; NG, DD, and AT participated in patient care; SD and AT wrote the paper.

## COMPETING INTERESTS

AT and NG are members of the editorial board for BCJ.

## ETHICAL APPROVAL

This study was approved by the Institutional Review Board of the Mayo Clinic (IRB protocol number: 12-003574). All methods were performed in accordance with the Declaration of Helsinki, the relevant guidelines, and regulations.

## ADDITIONAL INFORMATION

**Supplementary information** The online version contains supplementary material available at https://doi.org/10.1038/s41408-025-01285-x.

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