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Blood 142 (2023) 6422–6423

The 65th ASH Annual Meeting Abstracts

ONLINE PUBLICATION ONLY

634.MYELOPROLIFERATIVE SYNDROMES: CLINICAL AND EPIDEMIOLOGICAL

The Diagnostic Value of Circulating Blasts in Patients with Myeloproliferative Neoplasms in Accelerated or Blast Phase

Abdulraheem Yacoub, MD[1], *Shirene Philipose*[2]

[1]The University of Kansas Medical Center, Leawood, KS
[2]the university of Kansas Medical Center, Kansas City, KS

Introduction: Myeloproliferative neoplasms (MPNs) are progressive cancers with variable propensity to transform to accelerated phase and blast phase (APBP), an acute leukemia-equivalent state. MPN in APBP is associated with very poor prognosis with shortened survival and is notorious for poor response to therapy. The only curative therapy is allogeneic stem cell transplant.
Blast phase is defined by 20% blasts or higher in the bone marrow biopsy (BMBx) or peripheral blood (PB), while accelerated phase is defined by blast percentage 10% or higher. The diagnosis, and response to therapy often requires serial BMBx and PB testing. There is recognition of excessive circulating PB blasts in MPNs that occasionally exceeds blast count in BMBs, especially in fibrotic bone marrow. The aim of this study is to investigate the correlation between PB and BMBx blasts, and if PB testing might be a more convenient and lest costly marker for diagnosis and response assessment of MPN in APBP.
Methods: This is a cross sectional study, based on single institution retrospective chart review. Subjects included all available subjects over the age of 18, diagnosed with APBP MPN between January 1, 2017, to July 1, 2022. We collected serial blast percentages in simultaneously collected BMBx and PB samples, assessed by the same hematopathologist. We also collected baseline disease characteristics upon transformation to APBP. This is reported descriptively, and statistically analyzed for significance using a paired t-test.
Results: 15 subjects and 54 paired BP and BMBx blast assessments were identified. Median age was 59 (range 30 -71) and median duration since MPN diagnosis was 48 months (1-420). Median OS of the cohort since APBP diagnosis was 10 months. BMBx fibrosis was Grade II-III in 13/15 patients. Cytogenetic and mutation profiles, and therapies delivered are listed on Table 1. Based on evaluation of the results, there was no statistical correlation between blasts in the PB and BMBx both prior to therapy, and during therapy (detailed biostatistical analysis will be provided during ASH presentation)
Conclusions: Patients with APBP MPN undergo frequent diagnostic testing, including a bone marrow biopsy, to evaluate the percentage of blasts in the bone marrow. Quantification of blast count in peripheral blood should not be as a standalone diagnostic lab test for APBP MPN. BMBx remains essential for this diagnosis, response assessment, as well as cytogenetic and molecular profiling. The outcomes on APBP remain poor despite utilization of modern targeted therapeutics.

Disclosures Yacoub: *Apellis:* Consultancy; *Acceleron Pharma, Inc.:* Consultancy; *CTI Pharma:* Consultancy; *Protagonist Therapeutics, Inc.:* Consultancy; *Servier:* Consultancy; *Gilead:* Consultancy; *Novartis:* Consultancy; *Pfizer:* Consultancy; *Pharmaessentia:* Consultancy; *Incyte Corporation:* Consultancy; *Notable Labs:* Consultancy; *AbbVie Inc.:* Consultancy; *AbbVie, Acceleron, Apellis, CTI Pharma, Gilead, Incyte, Notable Labs, Novartis, Pfizer, PharmaEssentia, Servier.:* Consultancy.

https://doi.org/10.1182/blood-2023-184761

Table 1

Age at Diagnosis of APBP	Preceding MPN	Duration of MPN prior to APBP (months)	Cytogenetics	Pathological mutations on NGS	Grade of fibrosis	Treatment offered after transformation	Overall Survival
57	PV	15	complex including -5q	*P53 , DNMT3A, JAK2*	MF-3	Venetoclax/Azacytidine	2
66	PV	57	Nomral Karyotype	*KRAS, WT1, JAK2, RUNX1*	MF-2	Decitabine/Venetoclax; Mylotarg	28
39	ET	7	Nomral Karyotype	*ASXL1, CBL, SETPB1, ZRSR2*	NA	Decitabine/Venetoclax; (7+3); FLAG IDA; Azacytidine; Mylotarg; investigational CAR-T; Cladrbine, and LDAC	26
67	ET	36	complex	*IDH2, P53, SRSF2, CALR; TP53, CEBPA*	MF-3	Decitabine/Venetoclax; Enasidinib	23
65	PMF	180	47XY, +9[6]	*JAK2*	MF 2/3	NA	2
77	ET	2	Nomral Karyotype	*JAK2, SRSF2, RUNX1*	MF-2	Decitabine; Ivosidenib; Glasdigib and LDAC; Cladribine and LDAC Hydroxyurea,	33
55	PMF	233	Nomral Karyotype	*CALR*	MF-3	Decitabine; Mylotarg; Decitabine/ Venetoclax	21
55	PMF	0	Nomral Karyotype	*JAK2, U2AF1, ASXL1, STAG2*	MF-2	Decitabine Allo SCT	7
38	ET	420	Nomral Karyotype	*ASXL1, JAK2*	MF-2	Decitabine	10
52	ET	1	Complex	*P53 , JAK2 , EZH2, CTNNA1, PTPN11, KMT2C, NRAS*	MF-2	Decitabine/venetoclax;	8
76	PMF	60	+8, chr 6 abnormalities	*IDH1, JAK2*	MF-3	Decitabine Isovidinib	5
61	PV	180	46XX, t(2;8)(p13;q22)	*JAK2, GATA2, U2AF1*	MF-2	Decitabine / ruxolitinib	12
72	PV	36	45XX -7[20]	*IDH1, SRSF2, JAK2, PMS2, GATA2,*	MF-3	Azacytidine/ivosedinib	4
63	ET	48	47 XX +8 [4]/ 46XX [16]	*ASXL1, EZH2*	MF-1	Azacytidine; Decitabine/venetoclax ALLO SCT	24
30	ET	120	Complex	*JAK2, P53*	MF-2	Decitabine; Cladribine / LDAC; LDAC / venetoclax	7

Figure 1