RESEARCH LETTER

blood advances

TO THE EDITOR:

Genetic features and outcomes of allogeneic transplantation in patients with *WT1*-mutated myeloid neoplasms

Anmol Baranwal,[1] Rami Basmaci,[1] Rong He,[2] David Viswanatha,[2] Patricia Greipp,[2] Hemant S. Murthy,[3] James Foran,[3] Jeanne Palmer,[4] William J. Hogan,[1] Mark R. Litzow,[1] Mehrdad Hefazi,[1] Abhishek Mangaonkar,[1] Mithun Vinod Shah,[1] Aref Al-Kali,[1] and Hassan B. Alkhateeb[1]

[1]Division of Hematology and [2]Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MN; [3]Division of Hematology, Mayo Clinic, Jacksonville, FL; and [4]Division of Hematology, Mayo Clinic, Phoenix, AZ

The Wilms tumor 1 (*WT1*) gene, located at locus 11p13, is a tumor suppressor gene that encodes a zinc-finger transcription factor.[1,2] The presence of *WT1* gene mutation (m*WT1*) among adult patients with acute myeloid leukemia (AML) has been associated with poor prognoses.[1,3] Outcomes of allogeneic stem cell transplantation (alloSCT) in patients with m*WT1* AML have been explored in only a few studies limited by small sample size and inclusion of other gene mutations.[4-7] The goal of this study was to determine factors affecting post-alloSCT survival in patients with m*WT1* myeloid neoplasms.

We retrospectively reviewed patients with m*WT1* myeloid neoplasms, as defined by the World Health Organization.[8] The study was approved by the Mayo Clinic institutional review board and conducted in accordance with the Declaration of Helsinki. Patients with ≥2 *WT1* mutations were deemed to have multihit *WT1* (mh*WT1*) mutations. Given the limited sample size, we decided a priori upon variables deemed important from previous transplantation studies[9-14] to be included in stepwise regression analysis and arrived at the final multivariate model. Please refer to supplemental Methods for a detailed description of methods.

A total of 6887 patients were tested, and 75 (1.1%) patients were found to harbor a *WT1* mutation. Fifty-six (74.7%) had AML, 7 (9.3%) had myelodysplastic syndrome (MDS), 6 (8%) had mixed phenotypic acute leukemia (MPAL), and 6 (8%) patients had other myeloid neoplasms. Median age at diagnosis was 60 years (interquartile range [IQR], 42-67 years). Among patients with MDS, 4 (57.1%) had MDS with increased blasts (MDS-IB): 3 (42.9%) had MDS-IB2 and 1 (14.3%) had MDS-IB1. Median overall survival (OS) for the entire cohort was 1.9 years (95% confidence interval [CI], 1.45-2.42).

A total of 33 (44%) patients (21 [63.6%] males) underwent alloSCT at a median age of 44 years (IQR, 33-62 years). Twenty-five (75.8%) patients had AML, 3 (9.1%) had MDS, 2 (6.1%) had MPAL, and 3 (9.1%) had other myeloid neoplasms (supplemental Table 1). The median time to alloSCT after diagnosis was 6 months (IQR, 4-16 months). Twenty-seven (81.8%) patients were in complete remission (CR)/CR with incomplete count recovery at the time of alloSCT, 7 (21.2%) of whom were in second CR or beyond, whereas 5 (18.2%) patients, including 2 with AML and 1 with MPAL, had active disease. Post-alloSCT maintenance therapy was used in 13 (39.4%) patients, 8 (61.5%) of whom had a *FLT3* mutation (supplemental Table 2; supplemental Figure 1).

Five (15.2%) patients only had a *WT1* mutation, whereas 28 (84.8%) had at least 1 other comutated gene (Figure 1A). Twenty-one (63.6%) patients were found to have WT1 at initial diagnosis, whereas 12 (36.4%) of the patients were found to have a *WT1* mutation at first relapse or later (supplemental Table 3). The median number of comutations in the 28 patients was 2 (range, 1-5). The most frequently comutated gene was *FLT3* (n = 15, 45.4%): 12 (80%) had FLT3–internal tandem duplication and

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The data sets generated and analyzed in this study may be obtained upon reasonable request from the corresponding author, Hassan B. Alkhateeb (alkhateeb.hassan@mayo.edu).

The full-text version of this article contains a data supplement.

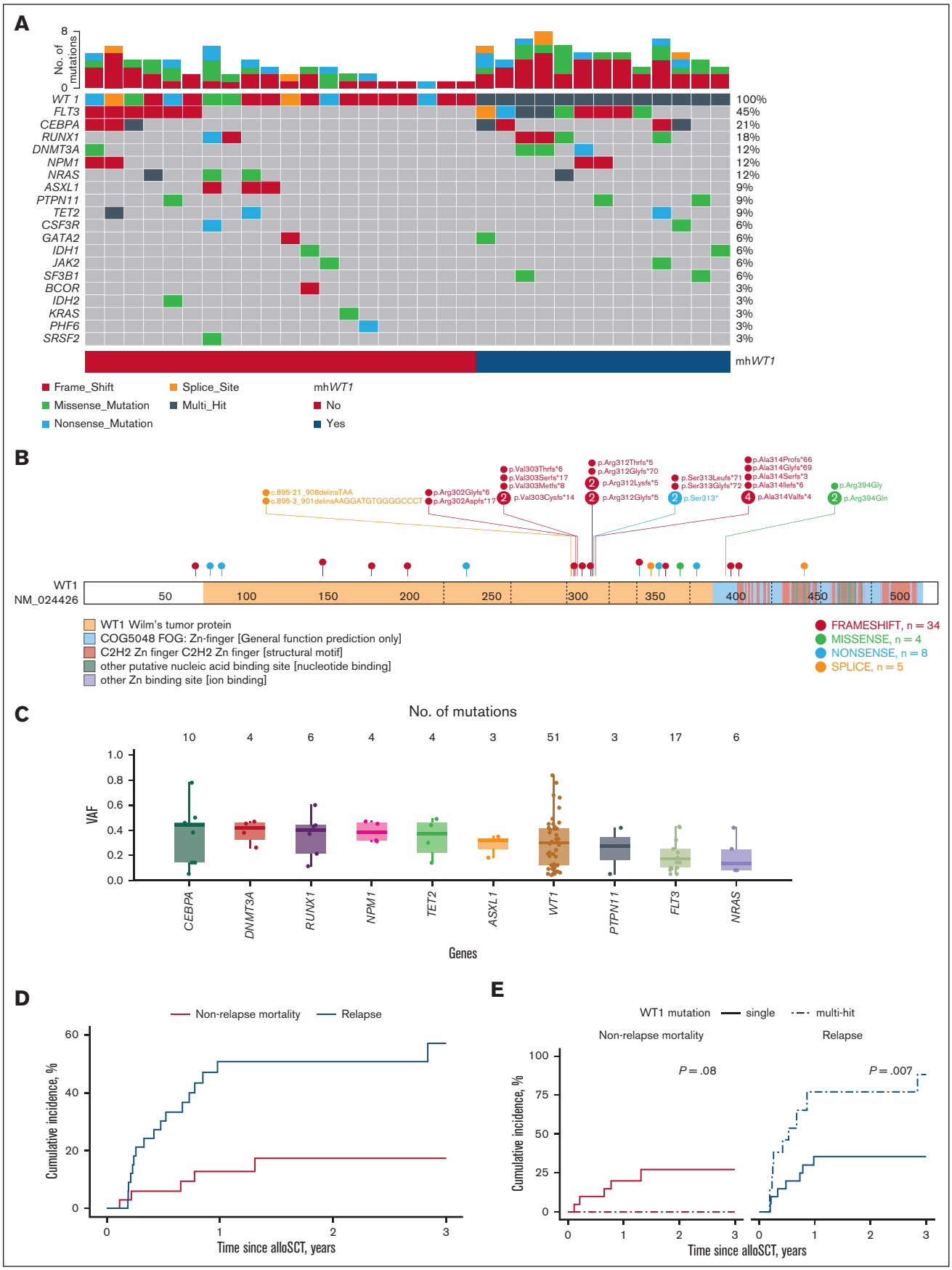

Figure 1.

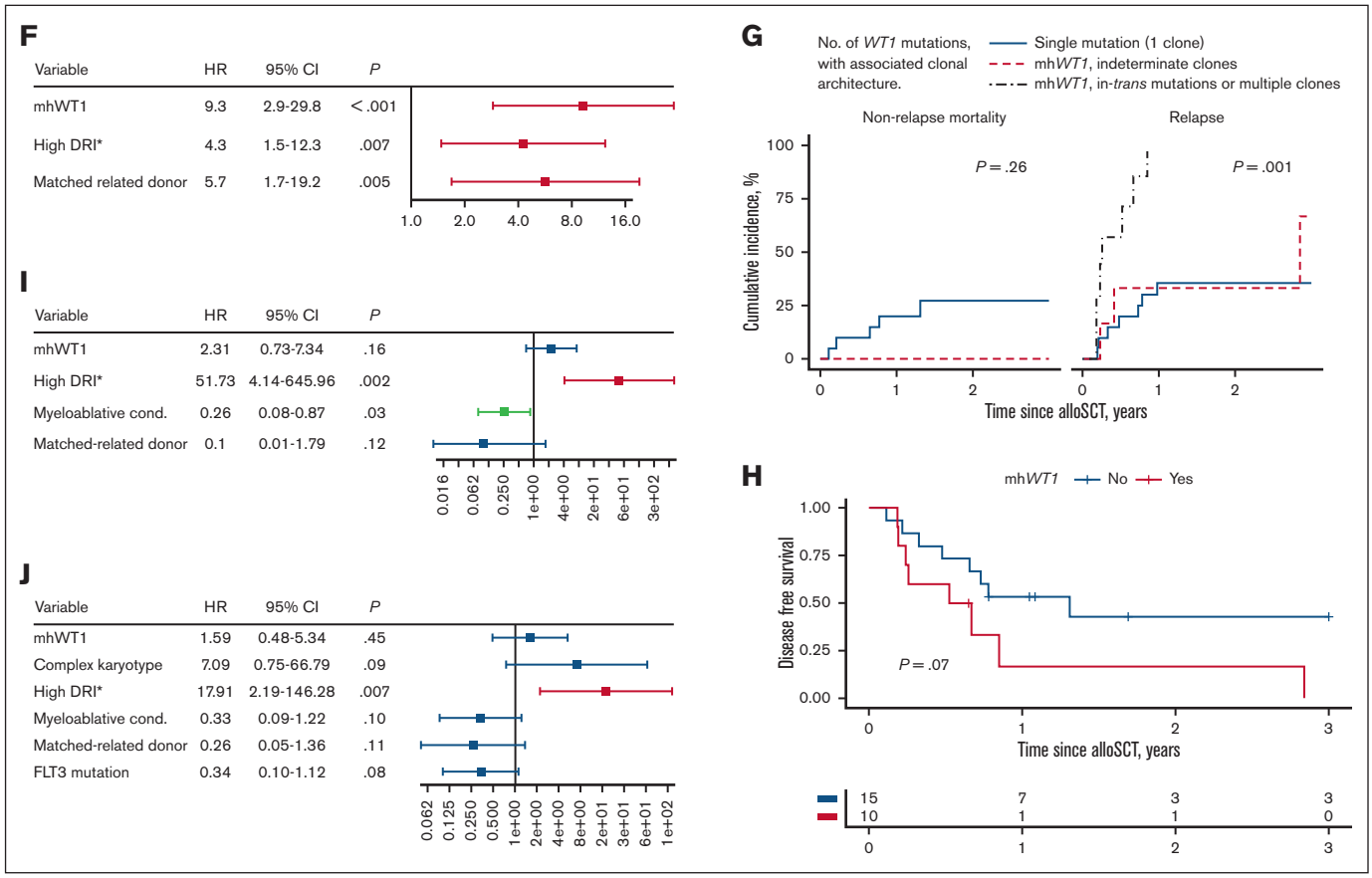

Figure 1 (continued) Genetic landscape and clinical outcomes of patients with *WT1*-mutated myeloid neoplasms undergoing alloSCT. (A) Oncoplot depicting comutations associated with *WT1*. Thirteen of 33 patients had mh*WT1*. (B) Lollipop plot showing position of mutations among 33 patients. Two hot spots were observed: codons 301-303 and codons 312-314. (C) Variant allele frequency (VAF) of top 10 mutated genes. (D) Post-alloSCT non-relapse mortality (NRM) and RI of the entire cohort. (E) Post-alloSCT NRM and RI stratified by single or mh*WT1* mutations. (F) Multivariate competing risk regression analysis for relapse among patients undergoing alloSCT. (G) Cumulative incidence of NRM and relapse stratified by *WT1* mutations and associated clonal architecture. (H) DFS after alloSCT, stratified by mh*WT1*. (I) Multivariate Cox proportional hazard analysis for 3-year DFS after transplantation in patients with m*WT1* AML. (J) Multivariate Cox proportional hazard analysis for 3-year OS after transplantation. *None of the patients had a very high DRI.

3 (20%) had *FLT3*–tyrosine kinase domain mutations. Several studies have shown that m*WT1* AML is associated with *FLT3* mutations.[15-17] High-risk comutations such as *BCOR* or *TP53* were rare (1 [3.3%] patient each). Thirteen (39.4%) patients had mh*WT1* (Figure 1A; Table 1). Two hot-spot regions were found: codons 301 to 303 (7 [21.2%] patients), and codons 312 to 314 (13 [42.4%] patients; Figure 1B). Median *WT1* variant allele frequency for patients undergoing alloSCT was 30% (IQR, 12%-42%; Figure 1C).

The cumulative relapse incidence (RI) was 21.1% at 100 days, 50.7% at 1 year, and 57.1% at 3 years after alloSCT (Figure 1D). Patients with mh*WT1* had higher post-alloSCT RI at 100 days (38.5% vs 10%), 1 year (76.9% vs 35.6%), and 3 years (88.5% vs 35.6%; $P = .007$; Figure 1E).

In multivariate analysis, mh*WT1* (hazard ratio [HR], 9.3; 95% CI, 2.9-29.8; $P < .001$), matched-related donor, and high disease risk index (DRI) were associated with an increased risk of posttransplant relapse (Figure 1F). We then studied the impact of mh*WT1* mutations in *cis* vs in *trans*/multiple clones on relapse. Of the 13 patients with mh*WT1*, 7 (53.8%) had *WT1* mutations either in *trans* or had multiple *WT1* clones, whereas the clonal architecture could not be determined in 6 (46.2%) patients with mh*WT1*. Patients with mutations in *trans*/multiple clones had a significantly higher RI than patients with mh*WT1* with indeterminate clonal architecture, or a single *WT1* mutation (1-year RI, 100% vs 33.3% vs 35.6%, $P = .001$; and 3-year RI, 100% vs 66.7% vs 35.6%, $P = .001$; supplemental Figure 1G).

Among patients with AML, those with mh*WT1* had an inferior 3-year disease-free survival (DFS; 3-year DFS, 0% vs 42.7%; $P = .07$; Figure 1H). Data on multiparameter flow cytometry–based minimal residual disease (MRD) testing were available in 14 (56%) patients with AML, 8 (57.1%) of whom were MRD negative (supplemental Table 4). Patients with MRD positive or unknown status before transplantation had a significantly higher relapse rate than patients who were MRD negative at 1 year (66.7% vs 63.6% vs 0.00%; $P = .03$) and 3 years (not applicable [NA] vs 72.7% vs 0.00%; $P = .02$) after alloSCT (supplemental Figure 2). Multivariate analysis showed that a high DRI was associated with an inferior DFS (HR, 51.73; 95% CI, 4.14-645.96; $P = .002$). Although mh*WT1* was associated with

Table 1. Characteristics and outcomes of patients with m*WT1* stratified by single *WT1* or mh*WT1*

Variable	mh*WT1* No (n = 20)	mh*WT1* Yes (n = 13)	*P* value
Age at diagnosis, y			
Median (min, max)	46.8 (18.4, 67.5)	42.7 (26.7, 71.8)	.85
Age at alloSCT, y			
Median (min, max)	48.0 (18.7, 68.0)	44.1 (27.5, 72.2)	.8
Sex			
Female	9 (45.0%)	3 (23.1%)	.36
Male	11 (55.0%)	10 (76.9%)	
Ethnicity			
Caucasian	17 (85.0%)	11 (84.6%)	1
Other	3 (15.0%)	2 (15.4%)	
***WT1* first detected**			
Initial diagnosis	14 (70.0%)	7 (53.8%)	.57
Relapse	6 (30.0%)	6 (46.2%)	
Disease characteristics			
Disease			
AML	15 (75%)	10 (76.9%)	.39
MDS	1 (5%)	2 (15.4%)	
MPAL	1 (5%)	1 (7.7%)	
Others	3 (15%)	0 (0%)	
Hemoglobin ≥ 10 g/dL at diagnosis			
No	17 (85.0%)	11 (84.6%)	1
Yes	2 (10.0%)	1 (7.7%)	
Missing	1 (5.0%)	1 (7.7%)	
Platelets ≥ 100 × 10^3/μL at diagnosis			
No	12 (60.0%)	10 (76.9%)	.42
Yes	7 (35.0%)	2 (15.4%)	
Missing	1 (5.0%)	1 (7.7%)	
Abnormal karyotype at diagnosis			
No	8 (40.0%)	8 (61.5%)	.47
Yes	11 (55.0%)	5 (38.5%)	
Missing	1 (5.0%)	0 (0%)	
Monosomy 7 at diagnosis			
No	18 (90.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
Missing	1 (5.0%)	0 (0%)	
Complex karyotype at diagnosis			
No	16 (80.0%)	13 (100%)	.38
Yes	3 (15.0%)	0 (0%)	
Missing	1 (5.0%)	0 (0%)	
Monosomal karyotype at diagnosis			
No	18 (90.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
Missing	1 (5.0%)	0 (0%)	
max*WT1* VAF			
Median [min, max]	34.5 [4.00, 84.0]	33.0 [12.0, 49.0]	.66

ATG, anti-thymocyte globulin; BCNU, carmustine; Bu, busulfan; CRi, complete remission with incomplete count recovery; Cy, cyclophosphamide; Flu, fludarabine; GVHD, graft-versus-host disease; HCT-CI, hematopoietic stem cell transplant comorbidity index; max, maximum; Mel, melphalan; min, minimum; MRD, matched related donor; MMUD, mismatched unrelated donor; MUD, matched unrelated donor; PT, posttransplant; TBI, total body irradiation; TLI, total lymphoid irradiation; VAF, variant allele frequency.

Table 1 (continued)

Variable	mh*WT1* No (n = 20)	mh*WT1* Yes (n = 13)	*P* value
Isolated WT1 (no comutation)			
No	15 (75.0%)	13 (100%)	.14
Yes	5 (25.0%)	0 (0%)	
m*WT1* in codons 301-303			
No	19 (95.0%)	7 (53.8%)	.02
Yes	1 (5.0%)	6 (46.2%)	
m*WT1* in codons 312-314			
No	14 (70.0%)	5 (38.5%)	.15
Yes	6 (30.0%)	8 (61.5%)	
m*WT1* in zinc-finger motif			
No	17 (85.0%)	11 (84.6%)	1
Yes	3 (15.0%)	2 (15.4%)	
CR/CRi at alloSCT			
No	2 (10.0%)	3 (23.1%)	.64
Yes	17 (85.0%)	10 (76.9%)	
Missing	1 (5.0%)	0 (0%)	
DRI			
Low	3 (15%)	1 (7.7%)	.83
Intermediate	13 (65%)	9 (69.2%)	
High	3 (15%)	2 (15.4%)	
NA	1 (5%)	1 (7.7%)	
Comutations			
ASXL1			
No	17 (85.0%)	13 (100%)	.40
Yes	3 (15.0%)	0 (0%)	
BCOR			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
CEBPA			
No	17 (85.0%)	9 (69.2%)	.52
Yes	3 (15.0%)	4 (30.8%)	
CSF3R			
No	19 (95.0%)	12 (92.3%)	1
Yes	1 (5.0%)	1 (7.7%)	
DNMT3A			
No	19 (95.0%)	10 (76.9%)	.31
Yes	1 (5.0%)	3 (23.1%)	
FLT3			
No	14 (70.0%)	4 (30.8%)	.06
Yes	6 (30.0%)	9 (69.2%)	
IDH1			
No	19 (95.0%)	12 (92.3%)	1
Yes	1 (5.0%)	1 (7.7%)	
IDH2			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	

ATG, anti-thymocyte globulin; BCNU, carmustine; Bu, busulfan; CRi, complete remission with incomplete count recovery; Cy, cyclophosphamide; Flu, fludarabine; GVHD, graft-versus-host disease; HCT-CI, hematopoietic stem cell transplant comorbidity index; max, maximum; Mel, melphalan; min, minimum; MRD, matched related donor; MMUD, mismatched unrelated donor; MUD, matched unrelated donor; PT, posttransplant; TBI, total body irradiation; TLI, total lymphoid irradiation; VAF, variant allele frequency.

Table 1 (continued)

Variable	mh*WT1* No (n = 20)	mh*WT1* Yes (n = 13)	*P* value
GATA2			
No	19 (95.0%)	12 (92.3%)	1
Yes	1 (5.0%)	1 (7.7%)	
JAK2			
No	19 (95.0%)	12 (92.3%)	1
Yes	1 (5.0%)	1 (7.7%)	
KRAS			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
NPM1			
No	18 (90.0%)	11 (84.6%)	1
Yes	2 (10.0%)	2 (15.4%)	
NRAS			
No	17 (85.0%)	12 (92.3%)	.93
Yes	3 (15.0%)	1 (7.7%)	
PHF6			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
PTPN11			
No	19 (95.0%)	11 (84.6%)	.69
Yes	1 (5.0%)	2 (15.4%)	
RUNX1			
No	18 (90.0%)	9 (69.2%)	.29
Yes	2 (10.0%)	4 (30.8%)	
SF3B1			
No	20 (100%)	11 (84.6%)	.29
Yes	0 (0%)	2 (15.4%)	
SRSF2			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
TET2			
No	18 (90.0%)	12 (92.3%)	1
Yes	2 (10.0%)	1 (7.7%)	
TP53			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
ZRSR2			
No	19 (95.0%)	13 (100%)	1
Yes	1 (5.0%)	0 (0%)	
Transplantation characteristics and outcomes			
HCT-CI ≥ 3			
No	11 (55.0%)	9 (69.2%)	.65
Yes	9 (45.0%)	4 (30.8%)	

ATG, anti-thymocyte globulin; BCNU, carmustine; Bu, busulfan; CRi, complete remission with incomplete count recovery; Cy, cyclophosphamide; Flu, fludarabine; GVHD, graft-versus-host disease; HCT-CI, hematopoietic stem cell transplant comorbidity index; max, maximum; Mel, melphalan; min, minimum; MRD, matched related donor; MMUD, mismatched unrelated donor; MUD, matched unrelated donor; PT, posttransplant; TBI, total body irradiation; TLI, total lymphoid irradiation; VAF, variant allele frequency.

Table 1 (continued)

Variable	mh*WT1* No (n = 20)	mh*WT1* Yes (n = 13)	*P* value
Conditioning intensity			
Myeloablative	12 (60.0%)	8 (61.5%)	1
Reduced intensity	7 (35.0%)	5 (38.5%)	
Missing	1 (5.0%)	0 (0%)	
Conditioning			
Bu/Cy	4 (20.0%)	1 (7.7%)	NA
Bu/Flu	4 (20.0%)	3 (23.1%)	
Bu/Flu/ATG	1 (5.0%)	0 (0%)	
Cy/Flu/thiotepa/TLI	2 (10.0%)	0 (0%)	
Cy/TBI	2 (10.0%)	2 (15.4%)	
Flu/Mel	4 (20.0%)	4 (30.8%)	
Flu/TBI	2 (10.0%)	1 (7.7%)	
Bu/Cy/ATG	0 (0%)	1 (7.7%)	
Flu/BCNU/Mel	1 (5%)	1 (7.7%)	
Graft source			
Peripheral blood	18 (90.0%)	13 (100%)	1
Bone marrow	1 (5.0%)	0 (0%)	
Missing	1 (5.0%)	0 (0%)	
Donor type			
MRD	5 (25.0%)	1 (7.7%)	.36
MUD	11 (55.0%)	9 (69.2%)	
MMUD	0 (0%)	1 (7.7%)	
Haploidentical	4 (20.0%)	2 (15.4%)	
Major/bidirectional ABO mismatch			
No	15 (75.0%)	10 (76.9%)	1
Yes	4 (20.0%)	3 (23.1%)	
Missing	1 (5.0%)	0 (0%)	
GVHD prophylaxis			
CD34 selection	1 (5.0%)	0 (0%)	.16
Tacrolimus + methotrexate (± ATG)	13 (65%)	9 (69.3%)	
Cyclosporine + methotrexate	2 (10.0%)	1 (7.7%)	
Tacrolimus + mycophenolate	3 (15.0%)	0 (0%)	
PT-Cy based	0 (0%)	3 (23.1%)	
None	1 (5.0%)	0 (0%)	
Grade 2-4 acute GVHD			
No	17 (85.0%)	10 (76.9%)	.9
Yes	3 (15.0%)	3 (23.1%)	
Grade 3-4 acute GVHD			
No	13 (65.0%)	12 (92.3%)	.17
Yes	7 (35.0%)	1 (7.7%)	
Moderate/severe chronic GVHD			
No	18 (90.0%)	11 (84.6%)	1
Yes	2 (10.0%)	2 (15.4%)	

ATG, anti-thymocyte globulin; BCNU, carmustine; Bu, busulfan; CRi, complete remission with incomplete count recovery; Cy, cyclophosphamide; Flu, fludarabine; GVHD, graft-versus-host disease; HCT-CI, hematopoietic stem cell transplant comorbidity index; max, maximum; Mel, melphalan; min, minimum; MRD, matched related donor; MMUD, mismatched unrelated donor; MUD, matched unrelated donor; PT, posttransplant; TBI, total body irradiation; TLI, total lymphoid irradiation; VAF, variant allele frequency.

an inferior DFS, it was not statistically significant (HR, 2.31; 95% CI, 0.73-7.34; $P = .16$; Figure 1I).

Median follow-up after alloSCT was 3.6 years (95% CI, 1.69-NA). Median OS for the entire cohort was 1.31 years (95% CI, 1.12-NA) and was similar to that of patients with m*WT1* AML undergoing alloSCT (median OS, 1.45 years; 95% CI, 1.12-NA; $P = .81$; supplemental Figure 3). Post-alloSCT 3-year OS was comparable among patients with AML vs those with non-AML disease (median, 1.45 vs 1.2 years; $P = .57$). Multivariate analysis confirmed that a high DRI was associated with a worse 3-year OS (HR, 17.91; 95% CI, 2.19-146.28; $P = .007$), whereas mh*WT1* was not associated with OS (HR, 1.59; 95% CI, 0.48-5.34; $P = .45$; Figure 1J).

Our study shows that the majority of patients with m*WT1* myeloid malignancies have either acute leukemia or MDS-IB. These findings have been reported previously.[18,19] We found a high post-alloSCT RI in this subset of patients. Luskin et al evaluated 112 patients with AML, 8 of whom had m*WT1*, and found that m*WT1* was associated with an increased post-alloSCT relapse (HR, 2.07; $P = .07$).[6] Similarly, Quek et al also reported that m*WT1* was associated with an increased risk of relapse (HR, 4.81; $P = .018$).[7] Most importantly, we found that mh*WT1* was associated with a high relapse rate, particularly among those with mutations in *trans* or with multiple *WT1* clones. Further studies involving single-cell sequencing will help confirm this finding.

Although mh*WT1* was not associated with an inferior post-alloSCT OS, there was a trend toward poor DFS. Likely, the early relapse detection through MRD and molecular-based studies and improved postrelapse treatment strategies influenced postrelapse survival. Post-alloSCT maintenance therapy was associated with a numerically superior 3-year post-alloSCT OS (median, 21 vs 11 months; $P = .16$) and DFS (median, 13 vs 7 months; $P = .39$; supplemental Figure 4). We did not find any specific comutation confounding the effect of mh*WT1* on post-alloSCT relapse (supplemental Table 5). Post-alloSCT survival was similar among patients with *WT1* mutation detected at initial diagnosis vs at relapse (median OS, 1.31 vs 1.23 years; $P = .61$).

Some of the limitations of our study include its retrospective nature and the small sample size.

In conclusion, this is, to our knowledge, the first study evaluating posttransplant outcomes in patients with *WT1*-associated myeloid malignancies. Our study shows that patients with *WT1* mutation are at a high risk of posttransplant relapse, particularly patients with mh*WT1* and, most importantly, those with mh*WT1* mutations in *trans* or multiple *WT1* clones. Post-alloSCT survival is poor, predominantly driven by relapse. Larger studies are needed to validate these findings.

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Contribution: H.B.A. conceptualized the study; A.B. and H.B.A. contributed to study design, analyzed data, and wrote the manuscript; R.B. contributed to data acquisition and reviewed the manuscript; R.H., D.V., and P.G. provided cytogenetics and molecular data; H.S.M., J.F., J.P., W.J.H., M.R.L., M.H., A.M., M.V.S., and A.A.-K. recruited patients; and all authors reviewed the manuscript and approved the final version.

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ORCID profiles: A.B., 0000-0002-3432-0489; R.H., 0000-0001-6116-8163; P.G., 0000-0002-5536-9011; J.P., 0000-0002-2185-1504; W.J.H., 0000-0002-5841-4105; M.R.L., 0000-0002-9816-6302; M.V.S., 0000-0002-5359-336X; A.A.-K., 0000-0002-0824-3715.

Correspondence: Hassan B. Alkhateeb, Mayo Clinic, Division of Hematology, 200 1st St SW, Rochester, MN 55905; email: alkhateeb.hassan@mayo.edu.

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