

Early post-transplant molecular residual disease predicts outcomes in myelodysplastic neoplasms and myelodysplastic neoplasms/acute myeloid leukemia undergoing allogeneic transplantation

Hsin-An Hou

hsinanhou@ntu.edu.tw

National Taiwan University Hospital <https://orcid.org/0000-0003-2780-4845>

Wan-Hsuan Lee

National Taiwan University Hospital <https://orcid.org/0000-0003-4795-9844>

Xavier Cheng-Hong Tsai

National Taiwan University Hospital

Chien-Chin Lin

National Taiwan University Hospital

Feng-Ming Tien

National Taiwan University Hospital <https://orcid.org/0000-0003-4513-2472>

Min-Yen lo

National Taiwan University Hospital

Yu-Sung Chang

National Taiwan University Hospital <https://orcid.org/0009-0000-4571-3328>

Tai-Chung Tseng

National Taiwan University Hospital

Tai-Chung Huang

National Taiwan University Hospital <https://orcid.org/0000-0002-1625-7295>

Bor-Sheng Ko

National Taiwan University Hospital <https://orcid.org/0000-0002-7965-7579>

Ming Yao

National Taiwan University

Yen-Ling Peng

National Taiwan University Hospital

Mei-Hsuan Tseng

National Taiwan University Hospital

Yuan-Yeh Kuo

National Taiwan University Hospital <https://orcid.org/0000-0002-6090-9000>

Ming-Chih Liu

National Taiwan University Hospital

Sze-Hwei Lee

Graduate Institute of Clinical Medicine, College of Medicine, National Taiwan University

Chi-Yuan Yao

National Taiwan University

Chang-Tsu Yuan

National Taiwan University Hospital

Shan-Chi Yu

National Taiwan University Hospital

Hwei-Fang Tien

NTUH <https://orcid.org/0000-0002-1384-5593>

Wen-Chien Chou

National Taiwan University Hospital <https://orcid.org/0000-0003-2967-698X>

Article

Keywords: myelodysplastic syndromes/neoplasms, allogeneic hematopoietic stem cell transplantation, next-generation sequencing, measurable residual disease

Posted Date: June 5th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9867217/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Additional Declarations: There is **NO** conflict of interest to disclose.

Abstract

Allogeneic hematopoietic stem cell transplantation (allo-HSCT) remains the only curative therapy for myelodysplastic neoplasms/syndromes (MDS), but post-transplant relapse continues to limit long-term survival. Reliable identification of patients at high relapse risk remains lacking. A cohort of 192 patients with MDS or MDS/acute myeloid leukemia undergoing allo-HSCT was analyzed to assess the prognostic significance of measurable residual disease (MRD) by next-generation sequencing (NGS) and multiparameter flow cytometry (MFC). MRD was assessed longitudinally at predefined time points; NGS-MRD positivity was defined as persistence of disease-associated mutations (excluding *DNMT3A*, *TET2*, and *ASXL1*) at a variant allele frequency > 0.01%. At 1 month post-transplantation, NGS-MRD positivity was detected in 25.9% of patients and was strongly associated with higher relapse risk and inferior survival outcomes. The 6-month cumulative incidence of relapse was significantly higher in MRD-positive vs. MRD-negative patients (64.7% vs. 23.5%), with markedly shorter relapse-free survival (5.3 vs. 85.3 months) and overall survival (13.4 vs. 73.7 months) (all $P < 0.05$). In multivariable analysis, 1-month NGS-MRD positivity remained an independent relapse and mortality predictor. MFC-MRD demonstrated prognostic value only at later time points. Early post-transplantation NGS-MRD may be a dominant prognostic biomarker and support its integration into MRD-guided, risk-adapted post-transplantation management strategies.

Introduction

Myelodysplastic syndromes/neoplasms (MDS) exhibit marked clinical and biological heterogeneity and carry a high risk of transformation to acute myeloid leukemia (AML) (1, 2). Although allogeneic hematopoietic stem cell transplantation (allo-HSCT) remains the only curative modality, post-transplant relapse, with cumulative incidence rates ranging from 15% to 60%, persists as the primary barrier to long-term survival and accounts for the majority of treatment failures (3–6).

Currently, considerable controversy remains regarding the optimal disease status and prognostic relevance of the bone marrow (BM) blast percentage before allo-HSCT (7, 8). Furthermore, no clear consensus exists on how pre-transplant treatment strategies, including the increasing use of hypomethylating agents (HMAs) and BCL-2 inhibitors, affect clonal architecture or subsequent transplant outcomes (8–10). Although the Molecular International Prognostic Scoring System (IPSS-M) has enhanced baseline risk stratification (11–14), its prognostic utility after transplantation remains constrained by its inherently static design (12, 15). Consequently, it fails to reflect the dynamic clearance of malignant clones throughout the transplant trajectory.

The inability to reliably identify patients at the highest risk of post-transplant relapse limits the use of timely, risk-adapted interventions, such as donor lymphocyte infusion (DLI) or maintenance strategies. Concurrently, it may expose low-risk individuals to unnecessary treatment-related toxicities. Molecular measurable residual disease (MRD) assessment using high-throughput sequencing provides a more advanced approach than conventional morphology and flow cytometry (16). Nonetheless, studies

assessing peri-transplant MRD in relation to transplant outcomes, using both molecular approaches and flow cytometry, remain limited. Consequently, accurately identifying patients at high risk of relapse and implementing timely, risk-adapted therapeutic interventions remains an unresolved clinical challenge.

This study aimed to address these critical knowledge gaps by systematically evaluating the clinical significance of post-transplant molecular MRD. Specifically, we examined how pre-transplant disease status and peri-transplant MRD, determined by both next-generation sequencing (NGS) and flow cytometry, collectively influence transplant outcomes. By establishing a temporal framework for MRD assessment, we aimed to define clinically actionable strategies to optimize pre-transplant management and identify the optimal window for post-transplant intervention.

Materials and Methods

Patient cohort

Overall, 1,694 patients were initially screened; 1,399 patients who did not undergo allo-HSCT were excluded. An additional 81 patients were excluded because of missing NGS data ($n = 59$) or the absence of detectable somatic mutations at diagnosis ($n = 22$), and 15 were excluded because cryopreserved BM samples were unavailable at 1 month post-transplantation. The final study cohort comprised 192 patients (Supplemental Fig. 1). All patients underwent allo-HSCT at the National Taiwan University (NTU) Hospital (NTUH) or NTU Cancer Center and harbored detectable somatic mutations at diagnosis, as determined by deep targeted sequencing.

Clinical variables were collected, including age at transplantation, sex, disease classification according to the French–American–British (FAB) classification, the 2016 and 2022 World Health Organization (WHO) classifications, and the International Consensus Classification (ICC), as well as risk stratification using the revised IPSS (IPSS-R) and IPSS-M. Variables also included molecular characteristics at diagnosis; type of and response to pre-transplant therapy; conditioning regimen; donor type; graft source; graft-versus-host disease (GVHD); DLI; post-transplant maintenance therapy; relapse; non-relapse mortality (NRM); relapse-free survival (RFS); and overall survival (OS).

The study was approved by the Research Ethics Committee of NTUH. Written informed consent was obtained from all participants in accordance with the Declaration of Helsinki (approval numbers: 201709072RINC and 202207050RINB).

BM assessment and cytogenetic analysis

BM cellularity and fibrosis grade were assessed using reticulin staining and were independently confirmed by board-certified hematopathologists (17). Cytogenetic studies were performed on BM aspirates and interpreted according to the International System for Human Cytogenetic Nomenclature (11, 18).

MRD assessment by NGS and multiparameter flow cytometry (MFC)

Baseline molecular profiling of BM samples obtained at diagnosis was performed using the TruSight myeloid sequencing panel (Illumina, San Diego, CA, USA) on the HiSeq platform (Illumina, San Diego, CA, USA). Alterations in 54 genes relevant to myeloid neoplasms were analyzed (Supplemental Table 1) (11, 19, 20). Pathogenic and likely pathogenic somatic variants were identified and used as molecular markers for MRD tracking. MRD was evaluated early post-transplant (30 days after allo-HSCT). NGS-based MRD positivity was defined as the persistence or re-emergence of disease-associated mutations with a variant allele frequency (VAF) > 0.01%. Only mutations already present at diagnosis were considered for NGS-MRD evaluation. The median sequencing depth at diagnosis was approximately 10,000×, and a similar depth was achieved at the MRD time points to ensure high sensitivity for low-frequency variant detection. To detect MRD, error-corrected genomic DNA sequencing was performed using a customized Qiagen myeloid panel (Hilden, Germany) with unique molecular identifiers covering 42 recurrently mutated genes. Libraries were prepared according to the manufacturer's protocol and sequenced on the Illumina NovaSeq platform. Raw data were processed using CLC Genomics Workbench software. Variants were called using tools optimized for low-frequency detection and annotated using COSMIC, dbSNP, and ClinVar; functional predictions were generated using PolyPhen-2 and SIFT. Error suppression was performed using a position-specific background model derived from the normal controls. Variants required three supporting reads. For conventional variant calling, variants were required to exceed the assay-specific limit of detection (approximately 0.2–0.5%). However, for MRD assessment, previously identified disease-associated mutations were tracked using ultra-deep error-suppressed sequencing, allowing detection down to a VAF of 0.01%. Germline variants were filtered using population databases and VAF patterns without matched normal samples. Donor-derived clonal hematopoiesis was not assessed. MRD tracking included only mutations detected at diagnosis. Low-VAF variants (< 1%) were included if they were reproducible. MRD positivity was defined as the presence of non-DTA mutations, with additional analyses including all mutations.

MFC was performed on BM aspirates using standardized antibody panels, longitudinally at prespecified time points, including pre-transplant, early post-transplant (30 days after allo-HSCT), and subsequent follow-up periods (60 and 90 days after allo-HSCT). Flow cytometry MRD was defined as the presence of phenotypically abnormal myeloid populations distinct from normal hematopoietic patterns. MFC-based MRD positivity was defined as $\geq 0.1\%$ LAIP/DfN-positive cells among total CD45 + cells. MRD negativity required acquisition of at least 5×10^5 CD45 + events, allowing reliable assessment down to 0.1%.

Statistical analysis

The Mann-Whitney U test was used for continuous variables, and Fisher's exact test or the χ^2 test was used for categorical variables. OS was calculated from the date of allo-HSCT to the last follow-up or death from any cause. RFS was calculated from the date of allo-HSCT to relapse, last follow-up, or death, whichever occurred first. Relapse and NRM were analyzed as competing-risk endpoints. The cumulative incidence of relapse (CIR) was estimated from the date of allo-HSCT using the cumulative incidence

function, with NRM treated as a competing event. Differences between groups were compared using Gray's test. Landmark analyses were performed at 1, 3, and 6 months, excluding patients who relapsed or died before each landmark time point.

Survival outcomes were estimated using the Kaplan–Meier method and compared using the log-rank test. Univariable and multivariable Cox proportional hazards regression models were fitted to evaluate variables associated with clinical outcomes, adjusting for relevant clinical and transplant-related covariates. Variables with clinical relevance or statistical significance in the univariable analysis were included in the multivariable model. To assess whether the prognostic impact of post-transplant MRD was driven by AML-like disease biology, a sensitivity analysis was performed, excluding patients who fulfilled the AML criteria based on the 2016 and 2022 WHO classifications or ICC before transplantation. Associations between post-transplant MRD status, RFS, and OS were then re-evaluated using the same statistical models as in the primary analysis.

All *P* values were two-sided and were considered statistically significant at $P < 0.05$. Analyses were performed using IBM SPSS Statistics v23 for Windows and R v4.5.1 for Windows.

Results

Demographic features and treatment characteristics

Diagnostic classifications according to the FAB, 2016 and 2022 WHO classifications, and ICC are summarized in Supplemental Table 2. The cohort included 23 patients with refractory anemia (RA), seven with RA with ring sideroblasts, 107 with RA with excess blasts, and 55 with RA with excess blasts in transformation (RAEB-T), as defined by the FAB classification. Discrepancies in AML classification arose from differences in cytogenetic and molecular definitions, as well as blast thresholds, between the 2022 WHO and ICC systems, particularly in cases involving *NPM1* mutations, MDS-related gene mutations or cytogenetic abnormalities, *CEBPA* mutations, and *NUP98* rearrangements. Overall, 10–14% of the patients were classified as having AML with MDS-related features, depending on the classification system (Supplemental Table 2). Four patients harbored biallelic *TP53* mutations. The genetic landscape is shown in Supplemental Table 3.

Patient characteristics are summarized in Table 1. The median age at diagnosis was 51 years, and 59.9% of patients were male. At diagnosis, 63% and 68% of patients were classified as high- or very high-risk according to IPSS-R and IPSS-M, respectively. One-third of patients (30.7%) underwent allo-HSCT after disease progression. Pre-transplant IPSS-R assessment was available for 80% of patients ($n = 153$); among these, 58% ($n = 89$) were downstaged, whereas 9% ($n = 14$) were upstaged before allo-HSCT. Upfront allo-HSCT was performed in 46% of patients, while 7% received HMA in combination with venetoclax before transplantation. Overall, 33% of patients achieved complete remission (CR), CR with limited count recovery, or CR with partial hematologic recovery, whereas approximately half exhibited no response to prior therapy.

Table 1
Demographics

Variables	Number (%) (n = 192)	Variables	Number (%) (n = 192)
Sex		No response	98 (51.0)
Female	77 (40.1)	PD	20 (10.4)
Male	115 (59.9)	Not evaluable	3 (1.6)
Age*	51 (18–72)	Pre-transplant BM blast*	3.8 (0-24.5)
Laboratory data*		Positive pre-transplant MRD by MFC	79 (60.3)
WBC, ×10 ⁹ /L	2.91 (0.6–125.0)	Donor	
ANC, ×10 ⁹ /L	1.01 (0.05–31.4)	MSD	81 (42.2)
Hb, g/dL	8.3 (3.2–14.0)	MUD	43 (22.4)
Platelet, ×10 ⁹ /L	73 (3-911)	MMSD	3 (1.6)
BM blast (%)	10 (0–29)	URD	27 (14.1)
PB blast (%)	1 (0–29)	Haploidentical	38 (19.8)
IPSS-R (at diagnosis)		Stem cell source	
Very low	3 (1.6)	PB	164 (85.4)
Low	21 (10.9)	BM	9 (4.7)
Intermediate	49 (25.5)	BM + PB	19 (9.9)
High	59 (30.7)	Conditioning	
Very high	60 (31.3)	MAC	50 (26.0)
IPSS-R (pre-transplant)	n = 153	RIC	142 (74.0)
Very low	27 (17.6)	Post-transplant	
*Median (range).			
†Other treatment: include low dose cytarabine, rabbit-derived anti-thymocyte globulin, cyclosporine, danazol, eltrombopag, erythropoietin-stimulating agents, thalidomide, and steroid			
‡Response was assessed according to the 2023 International Working Group criteria.			
§Three for virus infection. Two for poor engraftment. One for post-transplant pure red cell aplasia			

Variables	Number (%) (n = 192)	Variables	Number (%) (n = 192)
Low	42 (27.5)	positive 1-month MRD by FRC	29/144 (20.1)
Intermediate	32 (20.9)	positive 1-month MRD by NGS	28/108 (25.9)
High	39 (25.5)	3-month relapse rate	11/177 (6.2)
Very high	13 (8.5)	6-month relapse rate	28/158 (17.7)
IPSS-M (at diagnosis)	n = 177	12-month relapse rate	40/127 (31.5)
Very low	4 (2.3)	24-month relapse rate	51/103 (49.5)
Low	16 (9.0)	Acute graft-versus-host disease	n = 180
Moderate low	13 (7.3)	Grade 1–2	69 (38.3)
Moderate high	24 (13.6)	Grade 3–4	39 (21.7)
High	40 (22.6)	Chronic graft-versus-host disease	n = 176
Very high	80 (45.2)	Mild	63 (35.8)
Initial treatment		Moderate	25 (14.2)
Upfront HSCT	88 (45.8)	Severe	18 (10.2)
Intensive chemotherapy	49 (25.5)	Donor lymphocyte infusion	n = 169
HMA + venetoclax	13 (6.8)	prophylaxis	11 (6.5)
HMA monotherapy	98 (51.0)	mixed chimerism	8 (4.7)
Clinical trial	5 (2.6)	relapsed	24 (14.2)
Other treatment [†]	14 (7.3)	others [§]	6 (3.6)
Disease status at transplant [‡]		Post-transplant maintenance therapy	n = 17
CR + CRh+CR _L	63 (32.8)	HMA monotherapy	7 (4.1%)
*Median (range).			
[†] Other treatment: include low dose cytarabine, rabbit-derived anti-thymocyte globulin, cyclosporine, danazol, eltrombopag, erythropoietin-stimulating agents, thalidomide, and steroid			
[‡] Response was assessed according to the 2023 International Working Group criteria.			
[§] Three for virus infection. Two for poor engraftment. One for post-transplant pure red cell aplasia			

Variables	Number (%) (n = 192)	Variables	Number (%) (n = 192)
PR	7 (3.6)	HMA and venetoclax	10 (5.8%)
HI	1 (0.5)		
*Median (range).			
†Other treatment: include low dose cytarabine, rabbit-derived anti-thymocyte globulin, cyclosporine, danazol, eltrombopag, erythropoietin-stimulating agents, thalidomide, and steroid			
‡Response was assessed according to the 2023 International Working Group criteria.			
§Three for virus infection. Two for poor engraftment. One for post-transplant pure red cell aplasia			

Regarding transplant characteristics, 42% of patients received grafts from matched sibling donors, and 20% underwent haploidentical transplantation. Reduced-intensity conditioning (RIC) was used in most cases (74%). At 1 month post-transplantation, MRD positivity was detected in 20% of patients by flow cytometry and in 25.9% by NGS. Acute GVHD occurred in 60% of patients, with 22% classified as grades 3–4. Chronic GVHD was observed in 60% of patients; among these, half of them (50%) had mild-to-moderate disease. DLI was administered to 14.2% of patients for relapse and to 6.5% for prophylaxis. Post-transplant maintenance therapy with HMA, with or without venetoclax, was delivered to a limited subset of patients (8.9%).

Outcomes

During a median follow-up of 93.7 months, the median OS from the date of HSCT was 73.7 months. After allo-HSCT, the 2- and 5-year RFS rates were 71.5% and 64.8%, respectively. The posttransplant relapse rates at 3, 6, 12, and 24 months were 6.2%, 17.7%, 31.5%, and 49.5%, respectively (Table 1). The cumulative incidence of NRM was 19.3%, with similar rates between myeloablative conditioning (MAC) and RIC regimens (18.0% vs. 19.7%).

Pre-transplant disease characteristics, MRD assessment, and clinical outcomes

First, we assessed the impact of pre-transplant disease characteristics on post-transplant outcomes. Pre-transplant disease status was not statistically significantly associated with relapse incidence, RFS, or OS in this cohort (Supplemental Fig. 2). Conversely, pre-transplant BM blast percentage emerged as a significant prognostic factor. Patients with a pre-transplant BM blast count of < 5% exhibited a significantly longer median RFS than those with 5% or greater blasts (109.6 vs. 20.5 months, *P* = 0.011). A trend toward improved median OS after HSCT was also observed in the lower-blast group, although

this was not statistically significant (not reached vs. 38.2 months, $P = 0.05$) (Supplemental Fig. 3). Pre-transplant MRD assessed using MFC was not significantly associated with any clinical outcome measure, including relapse incidence, RFS, or OS (Supplemental Fig. 4).

Post-transplant MRD assessment and clinical outcomes

Next, we evaluated the prognostic impact of MRD assessed by MFC and NGS at different post-transplant time points. Post-transplant MRD assessment by MFC was performed 1, 3, and 6 months after allo-HSCT. Prognostic significance was observed exclusively at the 3- and 6-month time points. MFC-based MRD positivity at 3 months was associated with a markedly higher relapse rate, as well as significantly shorter median RFS (4.4 vs. 156.7 months) and median OS (11.5 months vs. not reached), compared with MRD-negative patients (Figs. 1d-1f). Similarly, MFC-based MRD positivity at 6 months predicted significantly worse median RFS (6.8 months vs. not reached) and median OS (18.1 months vs. not reached). All comparisons were statistically significant ($P < 0.01$) (Figs. 1g-1i). Conversely, MFC-based MRD positivity at 1 month post-transplant did not demonstrate predictive value for any of the evaluated outcomes (Fig. 1a-1c).

Subsequently, we examined molecular MRD assessed by NGS at 1 month post-transplant. NGS-based MRD positivity was associated with a higher relapse rate and shorter RFS (Supplemental Fig. 5). Notably, when mutations in *DNMT3A*, *TET2*, and *ASXL1* (DTA mutations), which persist in clonal hematopoiesis without necessarily indicating residual disease, were excluded from the analysis, the survival differences between MRD-positive and MRD-negative patients became substantially more pronounced (Fig. 2). Among patients classified as NGS MRD-positive after DTA mutation exclusion, the 6-month CIR was 64.7% compared with 23.5% in the MRD-negative group. Furthermore, MRD-positive patients demonstrated significantly shorter median RFS (5.3 vs. 85.3 months) and median OS (13.4 vs. 73.7 months) after allo-HSCT than their MRD-negative counterparts (all $P < 0.05$).

Combined MFC- and NGS-based MRD assessment at 1 month post-transplant

To further refine post-transplant risk stratification, we evaluated the combined prognostic utility of MFC- and NGS-based MRD (excluding DTA mutations) at 1 month after allo-HSCT. This integrated strategy identified distinct risk groups based on dual MRD status.

Patients with NGS-based MRD negativity had the lowest CIR, regardless of concurrent MFC-based MRD status. Conversely, NGS-based MRD positivity was associated with the highest relapse rates, independent of MFC-based MRD status. The 6-month CIR was 9.2% for patients who were double-negative (both NGS- and MFC-based MRD-negative), compared with 85.7% for those who were double-positive (Fig. 3a).

Regarding survival outcomes, patients with double-negative MRD status achieved median RFS and OS of 63.3 and 79.3 months, respectively, whereas those with double-positive MRD had markedly inferior outcomes, with median RFS and OS of 4.4 and 12.7 months, respectively (Fig. 3b and 3c). Consistent with the CIR findings, NGS-based MRD positivity, irrespective of MFC-based MRD status, was associated with the shortest RFS among all subgroups. Notably, patients with NGS-based MRD negativity but MFC-based MRD positivity ($n = 13$) demonstrated a numerically shorter median RFS and significantly shorter OS than did the double-negative group (RFS: 28.4 vs. 63.3 months, $P = 0.430$; OS: 7.9 vs. 79.3 months, $P = 0.027$).

Subgroup analysis by IPSS-R risk category

To evaluate whether the prognostic impact of post-transplantation NGS-based MRD differed by disease risk, we performed subgroup analyses stratified by the IPSS-R score at diagnosis and pre-transplantation. Among patients classified as very low-, low-, or intermediate-risk by IPSS-R at diagnosis, post-transplant NGS-based MRD positivity at 1 month (excluding DTA mutations) was not significantly associated with CIR, RFS, or OS (Supplemental Fig. 6). Conversely, among patients with high- or very high-risk IPSS-R at diagnosis, NGS-based MRD positivity at 1 month post-transplant was significantly associated with markedly higher relapse rates and inferior survival outcomes. The 6-month CIR was 66.7% in MRD-positive patients compared with 5.5% in MRD-negative patients, with correspondingly shorter median RFS (4.4 vs. 41.7 months) and median OS (13.4 vs. 55.2 months); all comparisons reached statistical significance (all $P < 0.05$) (Supplemental Fig. 7).

When patients were stratified by pre-transplant IPSS-R status, NGS-based MRD negativity at 1 month post-transplantation retained prognostic value in both lower- and higher-risk groups. MRD-negative patients consistently demonstrated superior outcomes, irrespective of pre-transplant risk category (Supplemental Figs. 8 and 9).

Univariable and multivariable analysis

Univariable analysis incorporating clinically relevant factors identified age; IPSS-M and IPSS-R at diagnosis; pre-transplant BM blast percentage; 1-month post-transplant NGS-based MRD status; and the occurrence of acute and chronic GVHD as factors significantly associated with clinical outcomes (Supplemental Table 4).

In multivariable analysis, 1-month post-transplant NGS-based MRD positivity (excluding DTA mutations) remained a significant, independent adverse prognostic marker. This finding conferred a more than 10-fold increased risk of relapse or treatment failure (hazard ratio [HR] 10.2, 95% confidence interval [CI] 2.8–36.8 for RFS) and a five-fold increased risk of death (HR 5.5, 95% CI 2.0–14.6 for OS) (Table 2). After excluding patients who met the AML criteria before transplantation, post-transplant NGS-based MRD positivity remained significantly associated with inferior RFS, consistent with the findings of primary analysis (Supplemental Table 5).

Table 2
Multivariable Cox regression analysis for relapse-free survival (RFS) and overall survival (OS)

Variables (N = 192)	RFS		OS	
	HR (95% CI)	P value	HR (95% CI)	P value
Age*	0.10 (0.95–1.04)	0.827	1.04 (0.99–1.09)	0.093
IPSS-M (at diagnosis)		0.988		0.937
Very low/low	Reference	-	Reference	-
Moderate low	0.91 (0.06–14.66)	0.946	0.73 (0.08–6.59)	0.778
Moderate high	0.96 (0.08–12.22)	0.974	0.52 (0.07–3.77)	0.515
High	0.70 (0.05–9.65)	0.792	0.64 (0.11–3.65)	0.613
Very high	1.02 (0.09–11.52)	0.990	0.53 (0.11–2.63)	0.438
Pre-transplant BM blast				
<5%	Reference	-	Reference	-
5-<30%	1.86 (0.68–5.10)	0.229	1.07 (0.42–2.72)	0.891
Post 1-month MRD by MFC	0.75 (0.25–2.27)	0.615	0.87 (0.30–2.53)	0.802
Post 1-month MRD by NGS†	10.22 (2.84–36.82)	< 0.001	5.46 (2.04–14.62)	0.001
Conditioning regimen				
RIC	Reference	-	Reference	-
MAC	0.67 (0.13–3.55)	0.636	0.55 (0.16–1.87)	0.338
Acute GVHD		0.785		0.531
Grade 0	Reference	-	Reference	-
Grade 1–2	0.88 (0.29–2.71)	0.826	1.88 (0.60–5.90)	0.278
Grade 3–4	0.64 (0.18–2.30)	0.490	1.50 (0.54–4.15)	0.439
P values of < 0.05 are statistically significant.				
*As continuous variables analysis.				
†DNMT3A, TET2, ASXL1 mutations were excluded.				
Abbreviations: BM, bone marrow; CI, confidence interval; GVHD, graft-versus-host disease; HR, hazard ratio; IPSS-M, Molecular International Prognostic Scoring System; MRD, measurable residual disease; MAC, myeloablative conditioning; NGS, next generation sequencing; RIC, reduced intensity conditioning				

Variables (N = 192)	RFS		OS	
	HR (95% CI)	P value	HR (95% CI)	P value
Chronic GVHD		0.240		0.063
no cGVHD	Reference	-	Reference	-
mild	0.82 (0.23–2.30)	0.580	0.34 (0.11–1.04)	0.058
moderate	0.23 (0.05–1.00)	0.050	0.24 (0.07–0.81)	0.021
severe	0.55 (0.14–2.13)	0.384	0.26 (0.05–1.38)	0.113
P values of < 0.05 are statistically significant.				
*As continuous variables analysis.				
†DNMT3A, TET2, ASXL1 mutations were excluded.				
Abbreviations: BM, bone marrow; CI, confidence interval; GVHD, graft-versus-host disease; HR, hazard ratio; IPSS-M, Molecular International Prognostic Scoring System; MRD, measurable residual disease; MAC, myeloablative conditioning; NGS, next generation sequencing; RIC, reduced intensity conditioning				

Furthermore, moderate chronic GVHD was identified as a protective factor, associated with a reduced risk of relapse and prolonged OS, consistent with a graft-versus-leukemia effect (Table 2).

Prognostic impact of conditioning intensity

In the overall cohort, MAC was associated with numerically longer RFS and OS than RIC (median RFS: 109.6 vs. 40.1 months, $P = 0.050$; median OS: 153.5 vs. 50.1 months, $P = 0.165$; Supplemental Fig. 10). To further investigate the interaction between pre-transplant disease burden and conditioning intensity, we performed a stratified analysis comparing MAC and RIC across subgroups defined by pre-transplant BM blast percentage. Among patients with a pre-transplant BM blast percentage > 5%, those who received MAC demonstrated a trend toward superior outcomes compared with those receiving RIC, with median RFS of 136.2 versus 14.5 months ($P = 0.055$) and median OS of 153.5 versus 24.1 months ($P = 0.105$) (Fig. 4). In contrast, among patients with a pre-transplant BM blast percentage < 5%, no additional survival benefit was observed with MAC over RIC (Supplemental Fig. 11).

Discussion

In this retrospective analysis of 192 patients with MDS and RAEB-T who underwent allo-HSCT, we evaluated diagnostic classification, clinical presentation, risk stratification according to IPSS-R and IPSS-M, pre-transplant disease status, and treatment regimens. We also analyzed the prognostic implications of pre-transplant disease status, BM blast percentage, and MRD assessed using MFC. Importantly, post-transplant assessments demonstrated that MRD evaluated using NGS at 1 month after allo-HSCT provides critical prognostic information. To our knowledge, this study is among the first to integrate

IPSS-M molecular risk stratification, pre-transplant MFC-based MRD, and post-transplant 1-month MFC-based and NGS-based MRD within a single, comprehensive framework for patients with MDS and RAEB-T undergoing allo-HSCT. By showing that NGS-MRD at 1 month post-transplant independently identifies patients at high risk of relapse, our work addresses a critical unmet need in post-transplant surveillance and may support the feasibility of early, NGS-guided preemptive intervention.

Allo-HSCT remains the only potentially curative treatment for patients with MDS, particularly those classified as higher risk by IPSS-R or IPSS-M (10, 21). Nonetheless, the role of pre-transplant bridging therapy in achieving optimal disease control, reducing transplant-related morbidity, and improving post-transplant outcomes remains controversial.

HMA has served as the backbone of pre-transplant cyto-reduction for the past two decades, with the AZA-001 (22) and D-0007 studies (23) establishing HMA as standard therapy for higher-risk MDS. Nonetheless, HMA-based combinations have yielded limited survival benefits. The ENHANCE trial (magrolimab plus azacitidine) was terminated early because of futility and safety concerns (24), whereas the VERONA trial (azacitidine plus venetoclax) improved response rates without an OS benefit (25). Although HMA plus venetoclax may increase transplant eligibility (10, 26–29), the optimal pre-transplant strategy remains controversial. Retrospective studies have reported no significant differences in post-transplant outcomes, and analyses have suggested that the choice of pre-transplantation regimen is associated with only modest, and frequently non-significant, differences in post-transplantation outcomes (9, 30).

Beyond pre-transplant bridging therapy, emerging evidence suggests that molecular risk downstaging after pre-transplant treatment does not translate into improved post-transplant outcomes. An EBMT registry study retrospectively analyzed 1,482 patients with MDS who had paired IPSS-R scores at diagnosis and at transplantation and demonstrated that IPSS-R improvement after HMA therapy or other treatments conferred no post-transplant survival benefit (31). This observation was subsequently extended to the molecular era by Aguirre et al. in a recent multicenter analysis of 176 HMA-treated patients with MDS undergoing allo-HSCT, in which dynamic IPSS-M reassessment at transplantation did not improve prognostic performance over baseline IPSS-M at diagnosis; apparent IPSS-M improvement yielded no gain in survival or relapse relative to unchanged risk (15). Consistent with the results of these studies, our cohort also showed that pre-transplant therapy, IPSS-R downstaging before transplantation, and response to pre-transplant treatment were not significantly associated with post-transplant outcomes.

The prognostic impact of the pre-transplant BM blast percentage on allo-HSCT outcomes in MDS remains controversial. Trottier et al. reported that > 10% pre-transplant blasts were independently associated with worse OS and higher NRM (32), whereas Schroeder et al. found no significant effect of blast percentage (> 10% vs < 10%) on OS or RFS in high-risk patients undergoing upfront transplantation (30). In our cohort, a pre-transplant BM blast percentage < 5% was associated with significantly superior RFS and a trend toward improved OS. The lack of a significant association between pre-transplant

disease status and outcomes may reflect the heterogeneity of disease-status categories and the limited sample size of this cohort. Conventional disease status may not adequately capture the actual residual disease burden at transplantation, whereas the pre-transplant BM blast percentage represents a more direct, quantitative measure of disease activity. This may explain why BM blast percentage, but not disease status, demonstrated prognostic significance for RFS. Importantly, in patients with a pre-transplant BM blast percentage > 5% prior to transplantation, MAC was associated with numerically longer RFS and OS than RIC, although these comparisons did not reach statistical significance. These exploratory findings suggest a possible interaction between disease burden and conditioning intensity that warrants further validation. This discordance across studies suggests that the pre-transplant blast percentage may serve as a surrogate for the underlying disease biology rather than as an independently modifiable prognostic factor.

Two landmark studies established the prognostic value of post-transplant NGS-based MRD in patients with MDS. Duncavage et al. demonstrated that persistence of mutations at day + 30 post-HCT was strongly associated with disease progression and inferior survival (33). Tobiasson et al. subsequently found, in a prospective cohort of 266 patients with MDS, that MRD positivity preceded relapse by a median of 71 days and was independently associated with markedly shorter RFS and OS (34). Building on this framework, our study showed that longitudinal post-transplant MRD assessment using MFC had time-dependent prognostic significance. In contrast, NGS-based MRD identified patients at high risk of relapse earlier and was more consistently predictive than MFC-MRD, emerging as the primary determinant of post-transplant prognosis in multivariable analyses. These findings suggest that 1-month post-transplantation NGS-MRD is the dominant molecular biomarker for early relapse risk stratification in patients with MDS.

For post-transplant maintenance therapy, the RELAZA-2 trial (35) demonstrated that MRD-guided preemptive azacitidine could delay or prevent overt hematologic relapse in a subset of patients. Conversely, a randomized study by Oran et al. 2020 phase III (36) showed that azacitidine maintenance, used as a prophylactic strategy irrespective of MRD status, did not confer a significant survival benefit. Therefore, MRD-guided post-transplant strategies may reduce unnecessary treatment-related toxicity while enabling identification of patients most likely to benefit from the intervention. Our study shows that NGS-based MRD may offer a critical window for preemptive interventions that MFC-based surveillance alone may not reliably provide.

This study has some limitations. First, its retrospective, single-center design may introduce selection bias and may limit generalizability to broader populations. Second, the final cohort comprised 192 patients, reduced from 1,694 screened based on the eligibility criteria, which may limit statistical power for subgroup analyses, particularly in the NGS-MRD-negative/MFC-MRD-positive subgroup and lower-risk IPSS-R categories. Additionally, only a limited number of patients received post-transplant maintenance therapy, and the use of DLI varied considerably across the cohorts. Consequently, the independent impact of these interventions on transplant outcomes could not be comprehensively evaluated, and their potential interaction with MRD status remains to be clarified in future prospective studies. Third,

although pragmatically justified, excluding DTA mutations as surrogates for clonal hematopoiesis may overlook their prognostic relevance in certain clinical contexts and warrants further investigation. Finally, heterogeneity in conditioning regimens, donor types, and post-transplant interventions across the study period may have independently influenced the outcomes. A prospective multicenter validation using standardized MRD protocols is required to confirm these findings.

To our knowledge, this study is the first comprehensive evaluation of both MFC- and NGS-based MRD in patients with MDS who underwent allo-HSCT. We demonstrated that 1-month post-transplant NGS-based MRD positivity is a strong, independent predictor of relapse and poorer survival. Notably, pre-transplant treatment strategies did not emerge as significant determinants of transplant outcomes, suggesting that timely post-transplant identification of high-risk patients and early, risk-adapted interventions may represent the most effective strategy for improving long-term outcomes. A prospective multicenter validation is warranted to establish standardized MRD-guided protocols and optimize outcomes in this challenging population.

Abbreviations

BM	bone marrow
CR	complete remission
CR _L	CR with limited count recovery
CRh	CR with partial hematologic recovery
MRD	measurable residual disease
MFC	multiparameter flow cytometry
MSD	matched sibling donor
MUD	matched unrelated donor
MMSD	mismatched sibling donor
MAC	myeloablative conditioning
NGS	next generation sequencing
RIC	reduced intensity conditioning

Hb
hemoglobin
HMA
hypomethylating agent
HSCT
allogeneic hematopoietic stem cell transplantation
HI
hematological improvement
URD
mismatched unrelated donor
IPSS-R
revised international prognostic scoring system
IPSS-M
molecular international prognostic scoring system
PR
partial response
PD
progressive disease
PB
peripheral blood
WBC
white blood cell count

Declarations

Competing Interests

The authors declare that they have no conflicts of interest.

Funding

This work was partially supported by grants from the Ministry of Science and Technology, Taiwan (MOST 109-2314-B-002-213); the National Science and Technology Council, Taiwan (NSTC 114-2314-B-002-286-MY2, 111-2314-B-002-122-MY3, 114-2314-B-002-256-MY3, and 114-2314-B-002-256-MY3); and the Ministry of Health and Welfare, Taiwan (MOHW 107-TDU-B-211-114009, 111-TDU-B-221-114001, and 115-TDU-B-221-114004). The funding agencies had no role in the study design, data acquisition, data analysis, manuscript preparation, or decision to submit the manuscript for publication.

Author contributions

WHL was responsible for data collection and management, statistical analysis and interpretation, literature review, and manuscript drafting. XCHT, YYK, and MHT conducted next-generation sequencing data collection and interpretation. MCL performed cytogenetic analyses. TCT, MY, SHL, YSC, CYY, TCH, BSK, CCL, FMT, MYL, YLP, WCC, MY, and HFT contributed to data collection and management. SCY and CTY performed the grading of bone marrow fibrosis. WCC coordinated its execution. HAH conceived and designed the study, coordinated its execution, interpreted the data, and contributed to manuscript writing and revision. All the authors have reviewed and approved the final manuscript.

Acknowledgments

We acknowledge the services provided by the Department of Laboratory Medicine, Medical Research, National Taiwan University Hospital, and the Tai-Chen Cell Therapy Center. We also thank the DNA Sequencing Core of the First Core Laboratory, National Taiwan University College of Medicine, Taiwan, for their invaluable assistance.

Data Availability Statement

The datasets generated and/or analyzed in this study are available from the corresponding author upon reasonable request.

References

1. Karel D, Valburg C, Woddor N, Nava VE, Aggarwal A. Myelodysplastic Neoplasms (MDS): The Current and Future Treatment Landscape. *Curr Oncol*. 2024;31(4):1971–93.
2. Tefferi A, Vardiman JW. Myelodysplastic syndromes. *N Engl J Med*. 2009;361(19):1872–85.
3. Lim Z, Brand R, Martino R, van Biezen A, Finke J, Bacigalupo A, et al. Allogeneic hematopoietic stem-cell transplantation for patients 50 years or older with myelodysplastic syndromes or secondary acute myeloid leukemia. *J Clin Oncol*. 2010;28(3):405–11.
4. Deeg HJ, Scott BL, Fang M, Shulman HM, Gyurkocza B, Myerson D, et al. Five-group cytogenetic risk classification, monosomal karyotype, and outcome after hematopoietic cell transplantation for MDS or acute leukemia evolving from MDS. *Blood*. 2012;120(7):1398–408.
5. Yeung CCS, Gerds AT, Fang M, Scott BL, Flowers MED, Gooley T, et al. Relapse after Allogeneic Hematopoietic Cell Transplantation for Myelodysplastic Syndromes: Analysis of Late Relapse Using Comparative Karyotype and Chromosome Genome Array Testing. *Biol Blood Marrow Transplant*. 2015;21(9):1565–75.
6. Mina A, Greenberg PL, Deeg HJ. How I reduce and treat posttransplant relapse of MDS. *Blood*. 2024;143(14):1344–54.

7. Busca A, Pecoraro C, Giaccone L, Bruno B, Allione B, Corsetti MT, et al. Allogeneic stem cell transplant for adults with myelodysplastic syndromes: relevance of pre-transplant disease status. *Leuk Lymphoma*. 2014;55(4):863–9.
8. Chen Y, Huang F, Xuan L, Zhang Y, Fan Z, Xu N, et al. Upfront transplantation may have better outcomes than pretransplant cytoreductive therapy for treating patients with MDS-EB-1 or MDS-EB-2. *Int J Cancer*. 2021;149(5):1109–20.
9. Potter VT, Iacobelli S, van Biezen A, Maertens J, Bourhis JH, Passweg JR, et al. Comparison of Intensive Chemotherapy and Hypomethylating Agents before Allogeneic Stem Cell Transplantation for Advanced Myelodysplastic Syndromes: A Study of the Myelodysplastic Syndrome Subcommittee of the Chronic Malignancies Working Party of the European Society for Blood and Marrow Transplant Research. *Biol Blood Marrow Transplant*. 2016;22(9):1615–20.
10. Lee WH, Lin CC, Tsai XC, Tien FM, Lo MY, Kuo YY, et al. Epidemiology, Treatment Outcomes, and Prognosis of Myelodysplastic Syndromes/Neoplasms in Taiwan: Real-World Insights and Trends. *Clin Lymphoma Myeloma Leuk*. 2025;25(6):e336-e47.e14.
11. Lee WH, Tsai MT, Tsai CH, Tien FM, Lo MY, Tseng MH, et al. Validation of the molecular international prognostic scoring system in patients with myelodysplastic syndromes defined by international consensus classification. *Blood Cancer J*. 2023;13(1):120.
12. Bernard E, Tuechler H, Greenberg PL, Hassserjian RP, Arango Ossa JE, Nannya Y, et al. Molecular International Prognostic Scoring System for Myelodysplastic Syndromes. *NEJM Evid*. 2022;1(7):EVIDoa2200008.
13. Sauta E, Robin M, Bersanelli M, Travaglino E, Meggendorfer M, Zhao LP, et al. Real-World Validation of Molecular International Prognostic Scoring System for Myelodysplastic Syndromes. *J Clin Oncol*. 2023;41(15):2827–42.
14. Aguirre LE, Al Ali N, Sallman DA, Ball S, Jain AG, Chan O, et al. Assessment and validation of the molecular international prognostic scoring system for myelodysplastic syndromes. *Leukemia*. 2023;37(7):1530–9.
15. Aguirre LE, Kim HT, Elmariah H, Frumm SM, Kelkar AH, Ho VT, et al. Baseline IPSS-M vs Pretransplant Risk Downstaging as Prognostic Determinants in MDS Undergoing Allogeneic Transplantation. *Blood Adv*. 2026.
16. Hou C, Zhou L, Yang M, Jiang S, Shen H, Zhu M, et al. The Prognostic Value of Early Detection of Minimal Residual Disease as Defined by Flow Cytometry and Gene Mutation Clearance for Myelodysplastic Syndrome Patients After Myeloablative Allogeneic Hematopoietic Stem-Cell Transplantation. *Front Oncol*. 2021;11:700234.
17. Lee WH, Lin CC, Tsai CH, Tien FM, Lo MY, Tseng MH, et al. Comparison of the 2022 world health organization classification and international consensus classification in myelodysplastic syndromes/neoplasms. *Blood Cancer J*. 2024;14(1):57.
18. International Standing Committee on Human Cytogenomic Nomenclature M-JJHRJMSSKG. ISCN 2020 an International System for Human Cytogenomic Nomenclature (2020): recommendations of

- the International Standing Committee on Human Cytogenomic Nomenclature including revised sequence-based cytogenomic nomenclature developed in collaboration with the Human Genome Variation Society (HGVS) Sequence Variant Description Working Group²⁰²⁰.
19. Grimwade D, Ivey A, Huntly BJ. Molecular landscape of acute myeloid leukemia in younger adults and its clinical relevance. *Blood*. 2016;127(1):29–41.
 20. Lee WH, Lin CC, Tsai CH, Tseng MH, Kuo YY, Liu MC, et al. Effect of mutation allele frequency on the risk stratification of myelodysplastic syndrome patients. *Am J Hematol*. 2022;97(12):1589–98.
 21. Gagelmann N, Kröger N. Allogeneic Stem Cell Transplant for Myelofibrosis and Myelodysplastic Syndromes: A Contemporary Review. *Am J Hematol*. 2025;100 Suppl 4(Suppl 4):16–29.
 22. Fenaux P, Mufti GJ, Hellstrom-Lindberg E, Santini V, Finelli C, Giagounidis A, et al. Efficacy of azacitidine compared with that of conventional care regimens in the treatment of higher-risk myelodysplastic syndromes: a randomised, open-label, phase III study. *Lancet Oncol*. 2009;10(3):223–32.
 23. Kantarjian H, Issa JP, Rosenfeld CS, Bennett JM, Albitar M, DiPersio J, et al. Decitabine improves patient outcomes in myelodysplastic syndromes: results of a phase III randomized study. *Cancer*. 2006;106(8):1794–803.
 24. Sallman DA, Al Malki MM, Asch AS, Wang ES, Jurcic JG, Bradley TJ, et al. Magrolimab in Combination With Azacitidine in Patients With Higher-Risk Myelodysplastic Syndromes: Final Results of a Phase Ib Study. *J Clin Oncol*. 2023;41(15):2815–26.
 25. Garcia-Manero G, Platzbecker U, Fenaux P, Roboz GJ, Fong CY, Hus M, et al. MDS-1497: Primary Analysis of the Randomized, Phase 3 VERONA Study of Venetoclax With Azacitidine Versus Placebo With Azacitidine in Patients With Treatment-Naïve, Intermediate and Higher-Risk Myelodysplastic Syndromes. *Clinical Lymphoma Myeloma and Leukemia*. 2025;25:S644-S5.
 26. Bataller A, Sasaki K, Urrutia S, Montalban-Bravo G, Bazinet A, Chien K, et al. Oral decitabine cedazuridine with and without venetoclax in higher-risk myelodysplastic syndromes or chronic myelomonocytic leukemia: a propensity score-matched study. *Blood Cancer J*. 2025;15(1):50.
 27. Gangat N, McCullough K, Johnson I, Al-Kali A, Begna KH, Patnaik MM, et al. Real-world experience with venetoclax and hypomethylating agents in myelodysplastic syndromes with excess blasts. *Am J Hematol*. 2022;97(6):E214-e6.
 28. Zugasti I, Lopez-Guerra M, Castaño-Díez S, Esteban D, Avendaño A, Pomares H, et al. Hypomethylating agents plus venetoclax for high-risk MDS and CMML as bridge therapy to transplant: a GESMD study. *Exp Hematol Oncol*. 2025;14(1):61.
 29. Garcia JS, Platzbecker U, Odenike O, Fleming S, Fong CY, Borate U, et al. Efficacy and safety of venetoclax plus azacitidine for patients with treatment-naïve high-risk myelodysplastic syndromes. *Blood*. 2025;145(11):1126–35.
 30. Schroeder T, Wegener N, Lauseker M, Rautenberg C, Nachtkamp K, Schuler E, et al. Comparison between Upfront Transplantation and different Pretransplant Cytoréductive Treatment Approaches

- in Patients with High-Risk Myelodysplastic Syndrome and Secondary Acute Myelogenous Leukemia. *Biol Blood Marrow Transplant*. 2019;25(8):1550–9.
31. Scheid C, Eikema DJ, van Gelder M, Salmenniemi U, Maertens J, Passweg J, et al. Does IPSS-R downstaging before transplantation improve the prognosis of patients with myelodysplastic neoplasms? *Blood*. 2024;144(4):445–56.
 32. Trottier BJ, Sachs Z, DeFor TE, Shune L, Dolan M, Weisdorf DJ, et al. Novel disease burden assessment predicts allogeneic transplantation outcomes in myelodysplastic syndrome. *Bone Marrow Transplant*. 2016;51(2):199–204.
 33. Duncavage EJ, Jacoby MA, Chang GS, Miller CA, Edwin N, Shao J, et al. Mutation Clearance after Transplantation for Myelodysplastic Syndrome. *N Engl J Med*. 2018;379(11):1028–41.
 34. Tobiasson M, Pandzic T, Illman J, Nilsson L, Weström S, Ejerblad E, et al. Patient-Specific Measurable Residual Disease Markers Predict Outcome in Patients With Myelodysplastic Syndrome and Related Diseases After Hematopoietic Stem-Cell Transplantation. *J Clin Oncol*. 2024;42(12):1378–90.
 35. Platzbecker AS, Georgi JA, Middeke JM, Sockel K, Wehner R, Herbst R, et al. Azacitidine to treat measurable residual disease in patients with MDS/AML: final long-term results of the RELAZA2 trial. *Blood*. 2026;147(10):1098–110.
 36. Oran B, de Lima M, Garcia-Manero G, Thall PF, Lin R, Popat U, et al. A phase 3 randomized study of 5-azacitidine maintenance vs observation after transplant in high-risk AML and MDS patients. *Blood Adv*. 2020;4(21):5580–8.

Figures

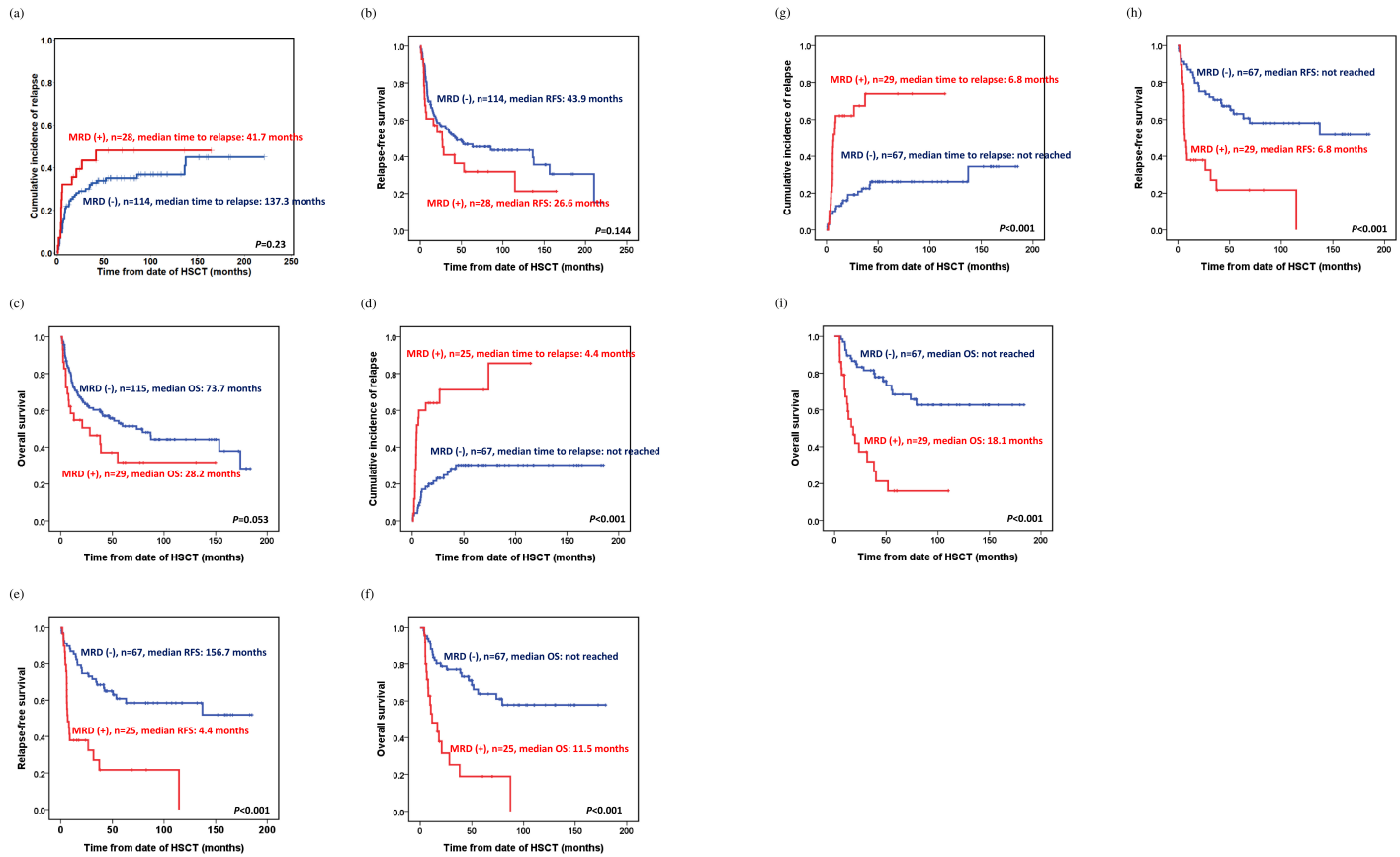


Figure 1

Time to disease relapse, relapse-free survival (RFS) and overall survival (OS) in patients with myelodysplastic syndromes/neoplasms undergoing allogeneic hematopoietic stem cell transplantation (HSCT) according to multiparametric flow cytometry–based measurable residual disease (MRD) status at 1 month (a, b, c), 3 month (d, e, f) and 6 month (g, h, i) post-transplant

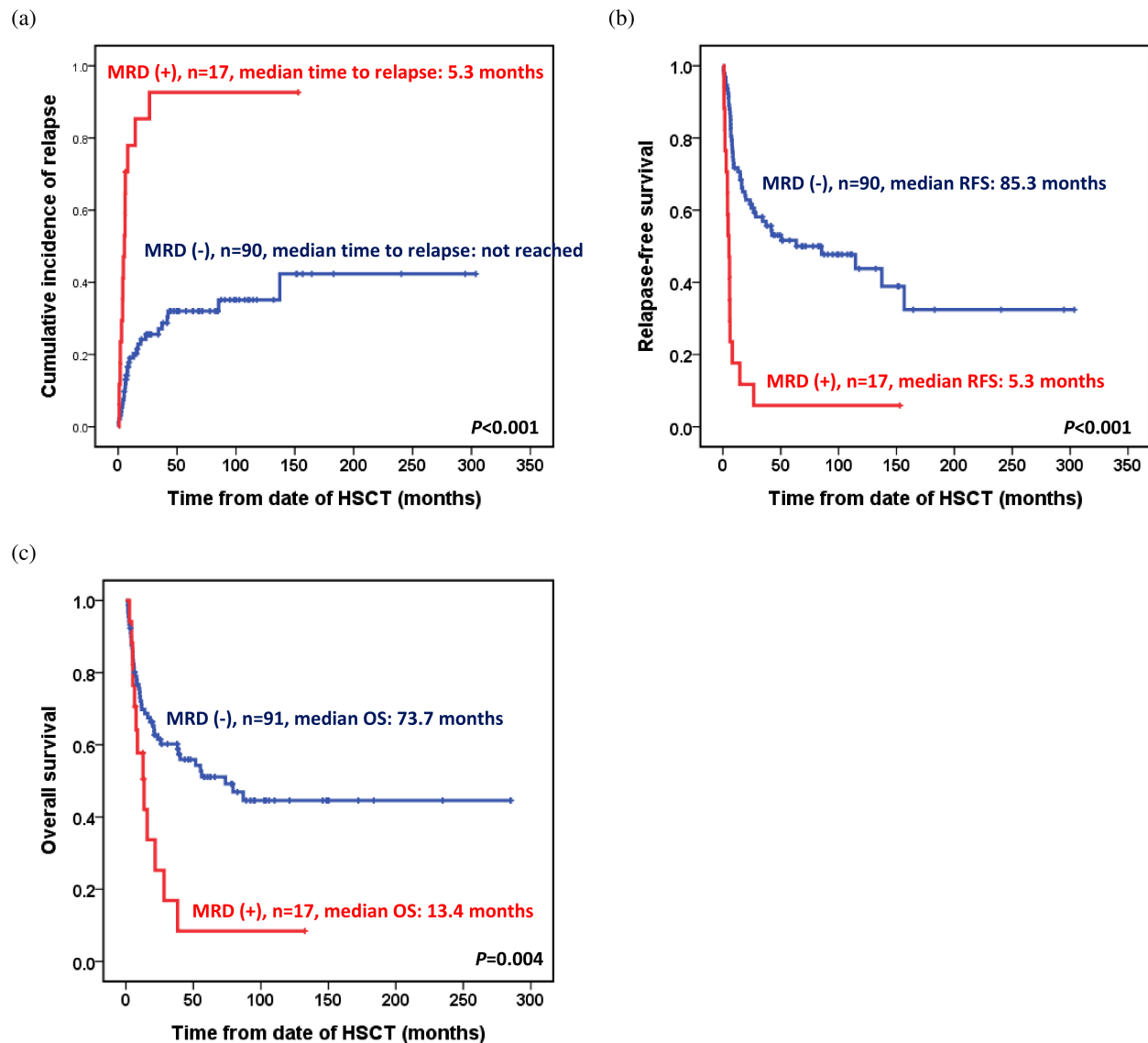


Figure 2

Time to disease relapse (a), relapse-free survival (RFS) (b) and overall survival (OS) (c) in patients with myelodysplastic syndromes/neoplasms undergoing allogeneic hematopoietic stem cell transplantation (HSCT) according to next-generation sequencing–based measurable residual disease (MRD) status at 1 month post-transplant. MRD assessment excluded mutations in *DNMT3A*, *TET2*, and *ASXL1*.

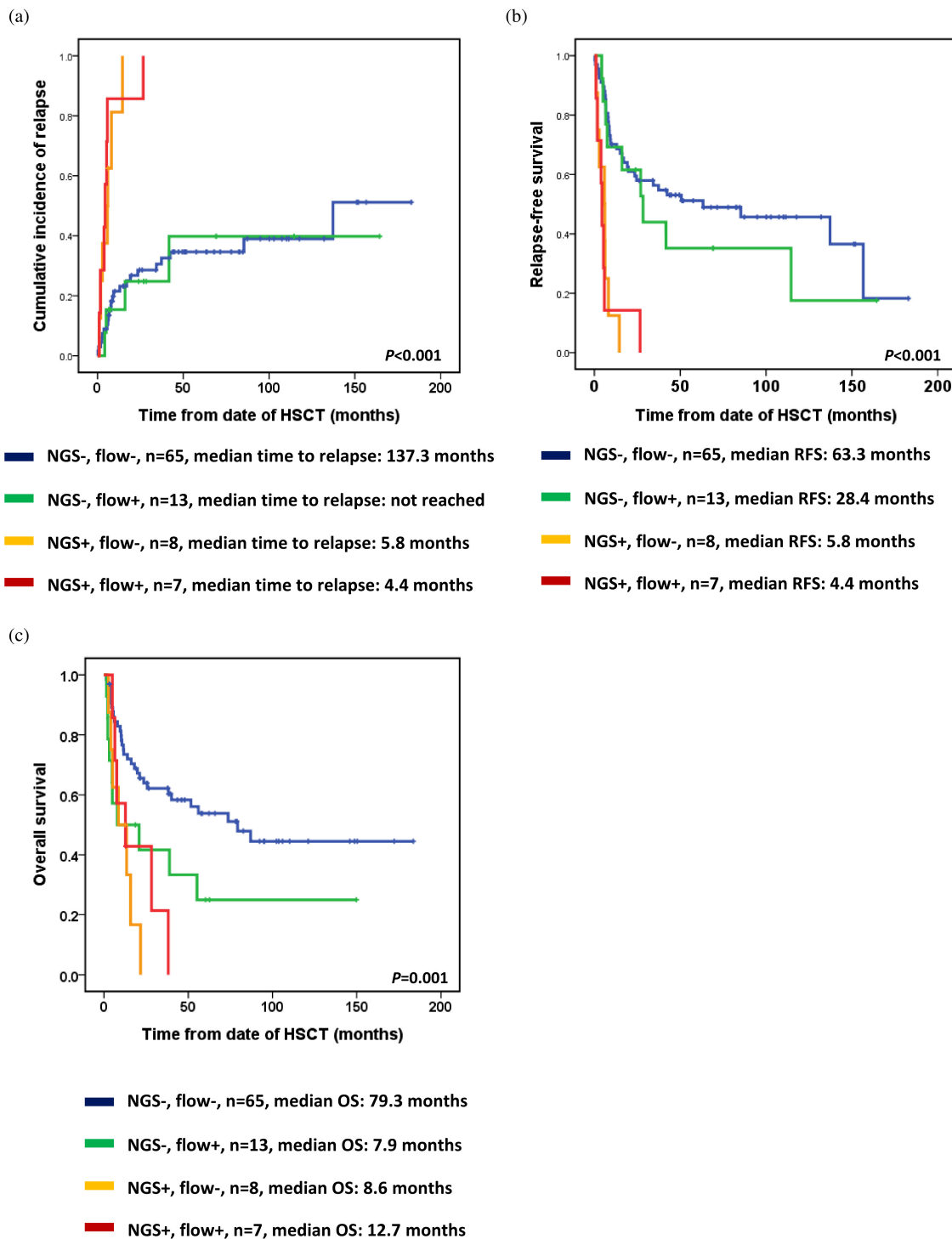
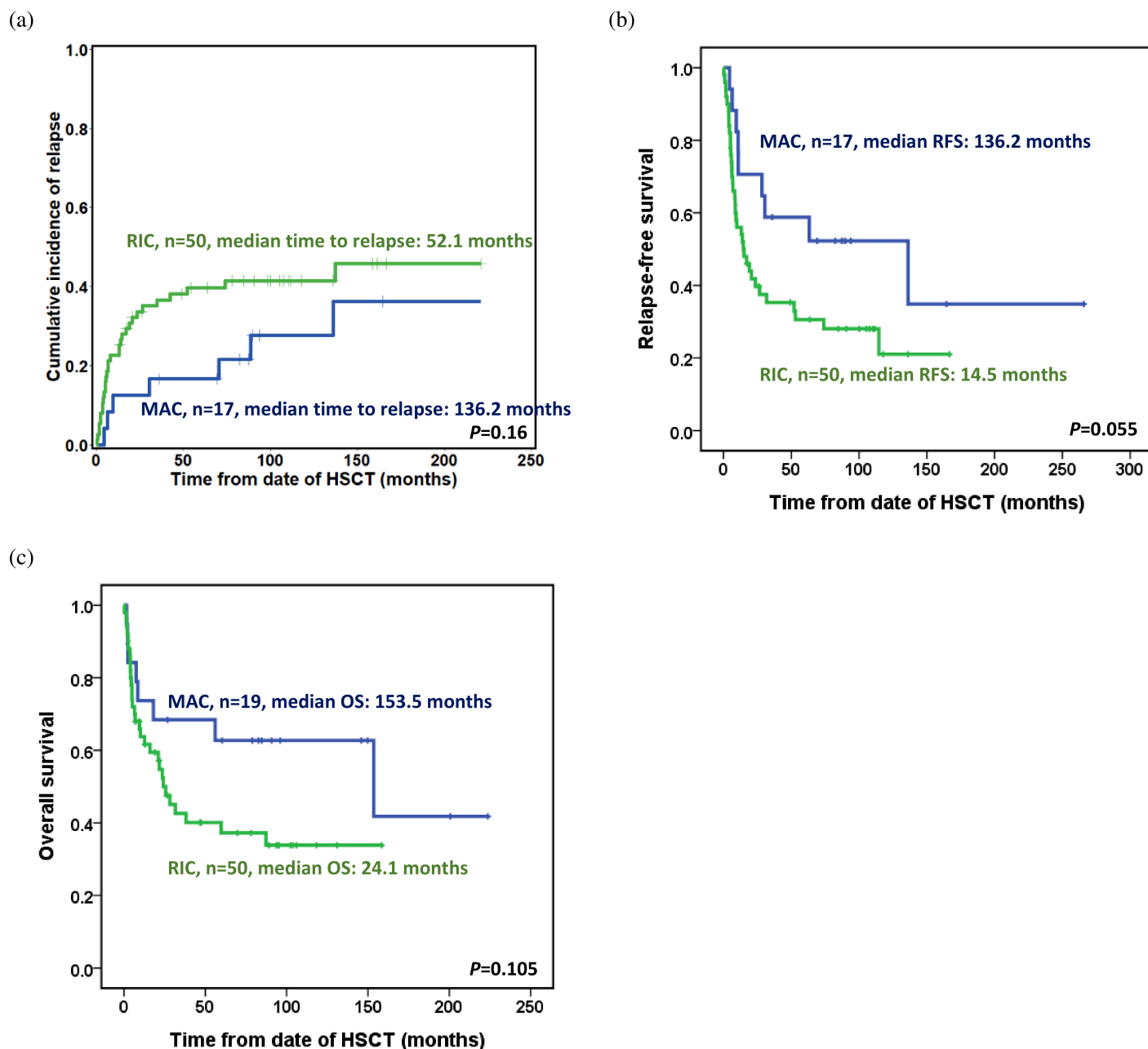


Figure 3

Time to disease relapse (a), relapse-free survival (RFS) (b) and overall survival (c) in patients with myelodysplastic syndromes/neoplasms undergoing allogeneic hematopoietic stem cell transplantation (HSCT) according to multiparametric flow cytometry-based and next-generation sequencing-based measurable residual disease (MRD) status at 1 month post-transplant. MRD assessment excluded mutations in *DNMT3A*, *TET2*, and *ASXL1*.



Abbreviation: MAC, myeloablative conditioning; RIC, reduced intensity conditioning

Figure 4

Time to relapse (a), relapse-free survival (RFS) (b), and overall survival (OS) (c) in patients with myelodysplastic syndromes/neoplasms undergoing allogeneic hematopoietic stem cell transplantation (HSCT) with pre-transplant bone marrow blasts 5-30%, stratified by conditioning regimen.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [HSCTSupplemental2060528.docx](#)