Outcomes of patients diagnosed with myelodysplastic syndrome or acute myeloid leukemia following solid organ transplantation

The incidence of solid organ transplantation (SOT) in the United States has dramatically increased over the past decade.[1] Immunosuppression is required to prevent transplant rejection and maintenance of allografts, but also increases the risk of developing secondary malignancies.[2] Following SOT, there is a 2.06-fold increased cancer mortality risk compared to the general population.[3] Skin cancers are overall the most common malignancies reported after SOT while post-transplantation lymphoproliferative disorders (PTLD) are the most common hematologic malignancy.[4,5] In contrast, post-SOT myeloid neoplasms are less common and incompletely understood. In this study, we analyzed the clinicopathologic characteristics and clinical outcomes of patients who received SOT and subsequently developed myelodysplastic syndrome (MDS) or acute myeloid leukemia (AML).

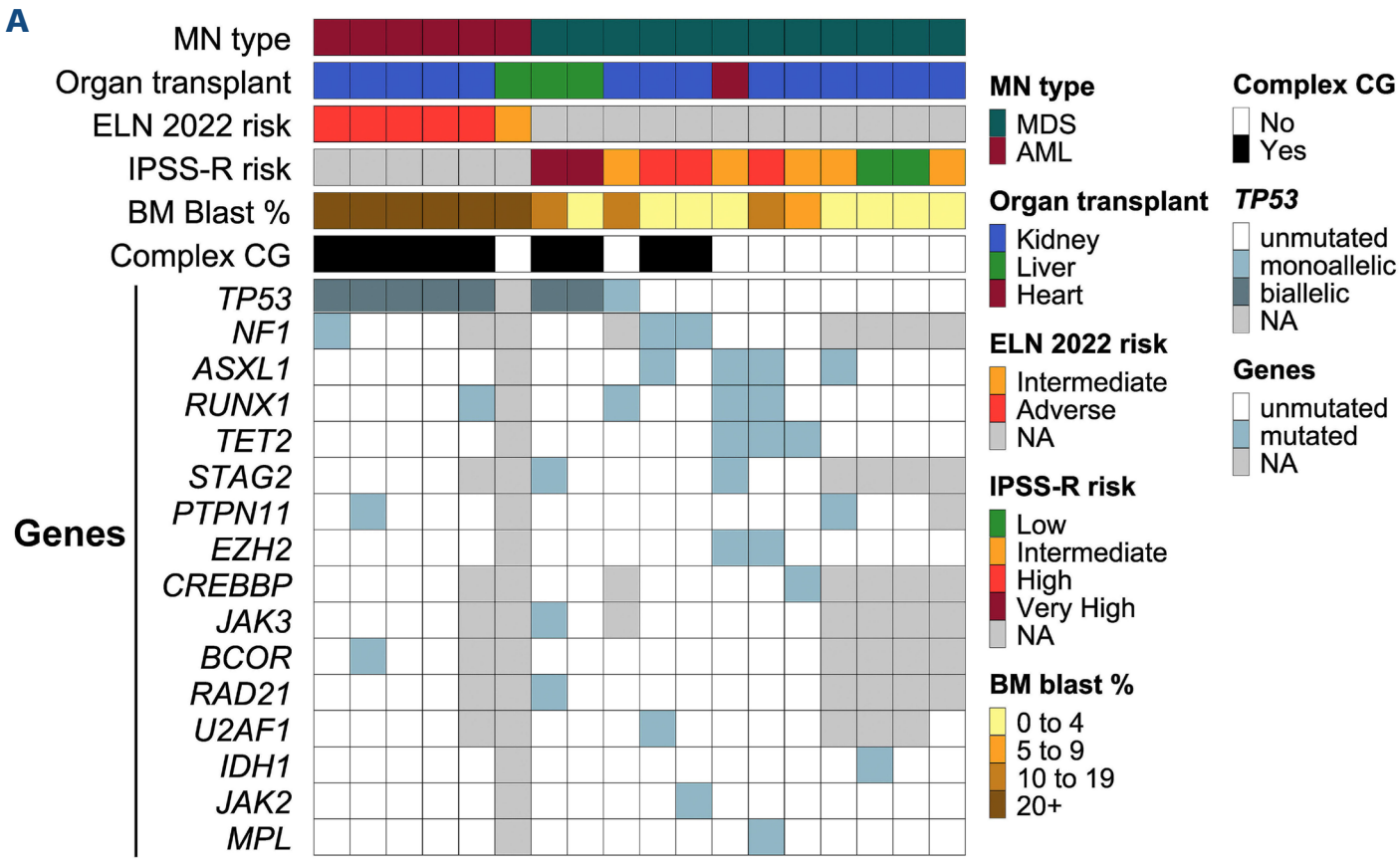
We conducted a single-center retrospective study spanning 2015-2025 at the University of Texas MD Anderson Cancer Center in Houston, TX, USA. We included patients with a history of SOT who were subsequently diagnosed with MDS or AML (WHO 2016). For the patients with AML, risk stratification and response assessment were performed according to the European LeukemiaNet 2022 (ELN 2022) guidelines.[6] For MDS, risk stratification was conducted using both the revised International Prognostic Scoring System (IPSS-R) and the molecular International Prognostic Scoring System (IPSS-M) when possible.[7,8] Higher risk MDS (HR-MDS) was defined as an IPSS-R score >3.5 points. Treatment responses in HR-MDS were assessed according to the International Working Group (IWG) 2023 criteria.[9] Next-generation sequencing (NGS) data were collected when available and NGS was performed using a 28- or 81-gene panel. Overall survival (OS) was defined as the time from diagnosis to death from any cause and was estimated using the Kaplan-Meier method. This study was approved by the University of Texas MD Anderson Institutional Review Board (IRB) and conducted in accordance with the principles of the Declaration of Helsinki.

We identified 18 patients who developed MDS (N=12, 67%) or AML (N=6, 33%) following SOT. The median age was 67 years (range: 50-77 years) in MDS and 58 years (range: 33-66 years) in AML. Baseline patient characteristics are shown in Table 1 and Figure 1A. SOT types were kidney in 14 (78%) patients, liver in 3 (17%), and heart in one (6%). Underlying reasons for kidney transplantation were glomerulonephritis (N=5), renal cell carcinoma (N=3), lupus nephritis (N=2), AL amyloidosis (N=1), diabetic nephropathy (N=1), congenital hypoplasia of kidneys (N=1), and chronic kidney disease of unknown cause (N=1). Reasons for liver transplantation

Table 1. Baseline patient characteristics.

Characteristics	MDS N=12	AML N=6
Median age, years, (range)	67 (50-77)	58 (33-66)
Male, N (%)	8 (67)	3 (50)
Median time from transplant to MN, years (range)	8.6 (1-43.7)	8.3 (1.1-31.4)
Organ transplant type, N (%)		
Kidney	9 (75)	5 (83)
Liver	2 (17)	1 (17)
Heart	1 (8)	0 (0)
Median BM blasts (%) (range)	2 (1- 17)	26 (20-40)
Median Hb, g/dL (range)	8.8 (7.1-11.8)	9.15 (7.4-12.5)
Median Plt, x10^9/L (range)	99 (30-453)	59 (34-137)
Median ANC, x10^9/L (range)	1.86 (0.81-6.00)	0.93 (0.00-2.80)
IPSS-R risk, N (%)		
Low	2 (17)	-
Intermediate	5 (42)	-
High	3 (25)	-
Very high	2 (17)	-
IPSS-M risk, N (%)		
Low	1/7 (14)	-
Moderate low	0/7 (0)	-
Moderate high	0/7 (0)	-
High	3/7 (43)	-
Very high	3/7 (43)	-
ELN 2022 risk, N (%)		
Intermediate	-	1 (17)
Adverse	-	5 (83)
Cytogenetic risk,* N (%)		
Good/Favorable	6 (50)	0 (0)
Intermediate/Intermediate	1 (8)	1 (17)
Poor/Adverse	2 (17)	5 (83)
Very poor	3 (25)	-
Complex cytogenetics, N (%)	4 (33)	5 (83)
TP53 status, N (%)		
Wild-type	9 (75)	0/5 (0)
Monoallelic mutated	1 (8)	0/5 (0)
Biallelic mutated	2 (17)	5/5 (100)

*Cytogenetic risk according to the Revised International Prognostic Scoring System (IPSS-R) / European LeukemiaNet (ELN) 2022. AML: acute myeloid leukemia; ANC: absolute neutrophil count; BM: bone marrow; ELN: European LeukemiaNet; Hb: hemoglobin; IPSS-M: molecular International Prognostic Scoring System; MDS: myelodysplastic syndrome; MN: myeloid neoplasm; N: number; Plt: platelets.



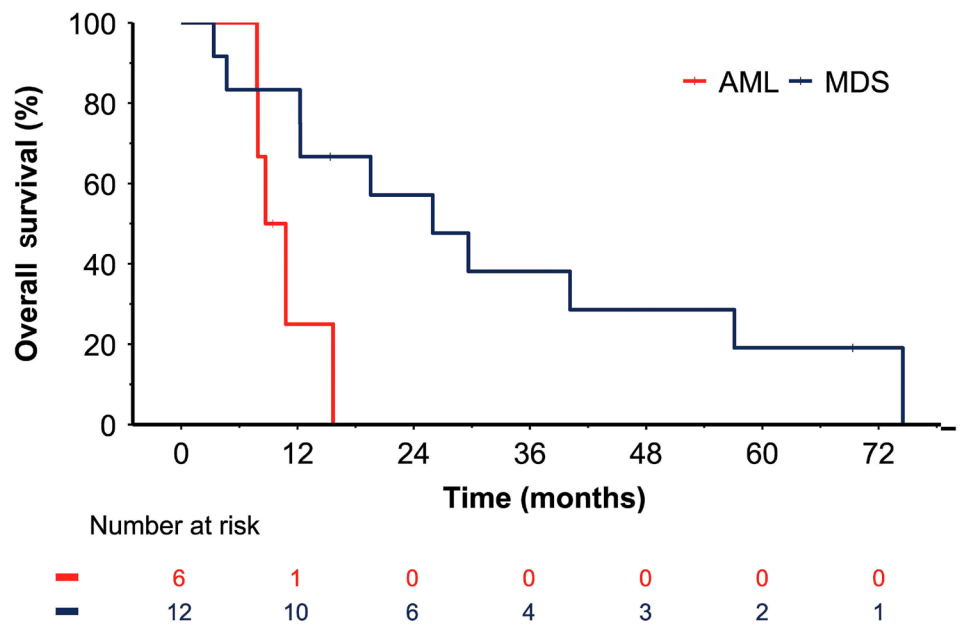


Figure 1. Characteristics and outcomes of post solid organ transplantation myeloid neoplasms. (A) Oncoprint of baseline characteristics. Biallelic *TP53*-mutated status was defined as a single *TP53* mutation with variant allele frequency >50%, two *TP53* mutations, or a *TP53* mutation with concurrent deletion of chromosome 17p. (B) Kaplan-Meier estimates of overall survival in patients with myelodysplastic syndrome (MDS) and acute myeloid leukemia (AML) after solid organ transplantation. BM: bone marrow; CG: cytogenetics; ELN: European LeukemiaNet; IPSS-R: Revised International Prognostic Scoring System; MN: myeloid neoplasm.

were metabolic dysfunction-associated steatohepatitis (MASH)-related cirrhosis (N=2) and hepatocellular carcinoma (N=1). One patient in the MDS cohort had received a heart transplantation for non-ischemic cardiomyopathy. The median latency period between the index SOT and initial myeloid neoplasm was 8.6 years (range: 1-43.7 years) in MDS and 8.3 years (range: 1.1-31.4 years) in AML.

This cohort was characterized by enrichment in higher risk disease features. Of the patients with MDS, 9/12 (75%) were HR-MDS by IPSS-R and 6/7 (86%) with 81-gene NGS data were high or very high risk by IPSS-M. In the patients with AML, 5/6 (83%) were adverse risk by ELN 2022. The most common mutations in the overall cohort were *TP53* (8/17, 47%), *NF1* (3/11, 27%), *ASXL1* (4/17, 24%), and *RUNX1* (4/17, 24%). Of the *TP53*-mutated patients (N=8), one (13%) was monoallelic mutated and 7 (88%) were biallelic mutated. Notably, all patients with AML who had test results available were biallelic *TP53*-mutated. The incidence of complex cytogenetics was 4/12 (33%) in MDS and 5/6 (83%) in AML (Table 1, Figure 1A). No patient in this cohort had *TERT* or *TERC* mutations.

Treatment outcome details are shown in Table 2. Frontline treatment regimens in the patients with MDS were hypomethylating agent (HMA)-based combinations in 7 (58%) patients, erythroid stimulating agents (ESA) in 2 (17%), luspatercept in one (8%), lenalidomide in one (8%), and observation alone in one (8%). Of these, only 4 patients were response evaluable as per the IWG 2023. The overall response rate in MDS was 50% (2/4 patients), including one complete remission (CR) and one CR with limited count recovery (CR_L), which occurred in HMA-treated patients. In AML, 4 (67%) patients received HMA-based therapy, one (17%)

received cladribine, low-dose cytarabine, and venetoclax, and one (17%) received intensive chemotherapy ("7 + 3"). The overall response rate in AML was 33% (2/6 patients), comprising one CR with "7+3" and one CR with incomplete platelet recovery (CRp) with decitabine + vosaroxin. Two (17%) patients in the MDS group and one (17%) patient in the AML group underwent allogeneic stem cell transplantation (SCT).

The survival outcomes were generally poor. With a median

Table 2. Treatments and patient outcomes.

ID	Age/ Sex	Organ transplant	Diagnosis	IPSS-R/ ELN 2022 risk	Cytogenetics	Mutations	Therapy	Response	SCT	Outcome
1	70/M	Kidney	MDS	Low	Normal	Negative	Darbepoetin alfa	-	No	Death from unknown cause
2	52/M	Kidney	MDS	Low	del(20q)	*IDH1*	Decitabine	-	No	Death from unknown cause
3	72/M	Kidney	MDS	Intermediate	del(7q)	*ASXL1, PTPN11*	None	-	No	Death from unknown cause
4	50/F	Liver	MDS	Very high	Complex	*JAK3, RAD21, STAG2, TP53* (bi)	Azacitidine + ruxolitinib	NR	Yes	Did not engraft post-SCT and progressed to AML. Death with refractory disease
5	77/F	Liver	MDS	Very high	Complex	*TP53* (bi)	Decitabine	NR	No	Death with refractory disease
6	58/F	Kidney	MDS	High	Complex	*JAK2, NF1*	Azacitidine	-	No	Death with refractory disease
7	52/F	Kidney	MDS	Intermediate	del(5q)	Negative	Lenalidomide	-	No	Alive at 5.8 years
8	62/M	Kidney	MDS	Intermediate	Normal	*RUNX1, TP53* (mono)	Decitabine + venetoclax	CR	Yes	Death from secondary graft failure on SCT D+138
9	69/M	Kidney	MDS	Intermediate	Normal	*CREBBP, TET2*	Darbepoetin alfa	-	No	Death from prostate cancer
10	75/M	Heart	MDS	Intermediate	add(21q)	*ASXL1, EZH2, RUNX1, STAG2, TET2*	Azacitidine	-	No	Death with refractory disease
11	65/M	Kidney	MDS	High	Normal	*ASXL1, EZH2, MPL, RUNX1, TET2*	Decitabine/ cedazuridine	CRL	No	Alive at 1.3 years
12	75/M	Kidney	MDS	High	Complex	*ASXL1, NF1, U2AF1*	Luspatercept	-	No	Death with refractory disease
13	66/F	Kidney	AML	Adverse	Complex	*TP53* (bi), *RUNX1*	Decitabine + vosaroxin	CRp	Yes	Death in remission from pneumonia on SCT D+65
14	61/M	Liver	AML	Intermediate	Normal	NA	7 + 3	CR	No	Death following relapse
15	33/M	Kidney	AML	Adverse	Complex	*TP53 (bi)*	Decitabine	NR	No	Death with refractory disease
16	52/F	Kidney	AML	Adverse	Complex	*NF1, TP53* (bi)	Azacitidine + venetoclax	NR	No	Death with refractory disease
17	54/F	Kidney	AML	Adverse	Complex	*TP53* (bi)	Azacitidine + anti-CD47	NR	No	Lost to follow-up
18	62/M	Kidney	AML	Adverse	Complex	*BCOR, PTPN11, TP53* (bi)	Cladribine + low-dose cytarabine + venetoclax	NR	No	Death with refractory disease

AML: acute myeloid leukemia; bi: biallelic; CR: complete remission; CRL: complete remission with limited count recovery; CRp: complete remission with incomplete platelet recovery; D: day; ELN: European LeukemiaNet; IPSS-R: revised International Prognostic Scoring System; MDS: myelodysplastic syndrome; mono: monoallelic; NA: not available; NR: no response; SCT: stem cell transplantation.

follow-up time of 69.3 months, the median OS from diagnosis was 15.7 months for the full cohort (1-year OS: 67%; 2-year OS: 14%). The median OS was 26 months (1-year: 83%; 5-year: 19%) in the patients with MDS and 9.7 months (1-year: 25%; 5-year: 0%) in the patients with AML (Figure 1B). The median OS was 19.6 months in HR-MDS *versus* 74.5 months in lower risk MDS (P=0.03). When stratified by *TP53* status, patients with *TP53* wild-type MDS had a median OS of 40.1 months *versus* 12.3 months in *TP53* mutated MDS (P=0.007). In the patients with MDS who received HMA-based therapy, the median OS from start of therapy was 25.1 months.

Toxicity from anti-leukemic therapy is a major concern in solid organ recipients due to ongoing use of immunosuppressive medications and potential fragile transplanted organ function. Infections were the most common cause of death in this cohort. Specifically, 8/12 (67%) deaths with known cause were due to infections (5 from pneumonia, 1 from urinary tract infection, and 2 from septic shock). In the patients with renal transplants (N=14), the median baseline creatinine was 1.2 (range: 0.7-3.7). Two renal transplant recipients (17%) experienced creatinine elevations of ≥30% from baseline while on therapy. These patients were undergoing therapy with decitabine (N=1) and azacitidine (N=1), respectively. One renal transplant recipient (8%) required initiation of dialysis on therapy, but this patient only received ESA. No heart or liver transplant recipient experienced failure of the transplanted organ. The 4-week and 8-week mortality of patients who received potentially disease-modifying therapy were 0% and 0%, respectively. All 3 patients who underwent SCT experienced a poor outcome. One SCT-recipient died on day +65 post-transplant from pneumonia (in remission), one experienced primary hematopoietic graft failure and was transferred to a hospice, and another experienced secondary hematopoietic graft failure and died on day +138 post-transplant.

Large epidemiological studies have demonstrated an increase in incidence of both MDS and AML in SOT recipients (standardized incidence ratio: 4.6 for MDS and 2.7 for AML).[10] Long-term use of post-transplant immunosuppressive therapy may lead to decreased immune surveillance, which could facilitate the survival of a neoplastic clone leading to malignancy. In addition, specific drugs such as the antimetabolite azathioprine have been associated with increased risk compared to other immunosuppressive agents, possibly via selection of DNA mismatch repair (MMR) defective clones.[10,11]

Studies on myeloid malignancies among SOT recipients are rarely reported in the literature. In our study, outcomes were generally poor with low response rates to therapy and short survival, particularly in the patients with AML. Notably, almost all cases of post-SOT AML we identified harbored biallelic *TP53* mutations and complex cytogenetics, which are known to confer dismal outcomes. This finding should be interpreted with caution given the small number of patients with AML in our series. Our results are, however, consistent with other published series. Wu and colleagues retrospectively analyzed 12 patients with AML (median age: 64 years) and 5 with MDS (median age: 58 years) following SOT, with a high frequency of adverse cytogenetics. This series included a high proportion of liver (N=5) and lung (N=6) transplantation cases whereas our cohort was predominantly comprised of kidney transplantation cases. Outcomes were poor, with a median OS of only 3.5 months in AML and seven months in MDS.[12] Another report describing 14 patients with post-SOT AML (median age: 66 years) treated with intensive chemotherapy (N=7), low-intensity therapy (N=4), or supportive care (N=3) showed a 55% CR rate in those who received therapy. These patients were enriched with unfavorable cytogenetics. No patient in this cohort underwent SCT and the median OS was only six months.[13] In contrast to AML, patients with post-SOT MDS in our series included lower risk cases (25%) and had longer OS (26 months), consistent with other observations.[14] It is still not known whether the generally poor outcomes in post-SOT myeloid neoplasm reflect the unique biology of the post-transplant setting or simply represent a type of therapy-related myeloid neoplasm.

Patients who are SOT recipients have an increased comorbidity burden, complicating the administration of anti-leukemic therapy. In our series, lower-intensity therapy was feasible for most patients. Only one patient received intensive chemotherapy. No transplanted organ failure was attributable to therapy, although 2 patients with renal transplantations (17%) experienced acute kidney injury with HMA. One renal transplant recipient required dialysis following MDS diagnosis, but this patient only received ESA, which was unlikely to be related to renal failure. Infections represented the most common cause of death in our cohort. This risk may be exacerbated by ongoing immunosuppressive medications and warrant the aggressive use of prophylactic anti-infectives.

Allogeneic stem cell transplantation is a primary consolidative strategy in higher risk MDS and AML. Although only 3 patients underwent SCT in our series, precluding any definitive conclusions, outcomes were poor with 2 (67%) experiencing hematopoietic graft failure and another dying from infectious complications. A larger series reported post-SOT SCT was feasible, with low rates of transplanted organ and hematopoietic graft failures.[15]

Major limitations of our study include the small sample size, heterogeneity, single center data, and retrospective design. Furthermore, not all patients underwent NGS with the full 81-gene panel and complete data on immunosuppressive regimens were missing. However, overall, our data contribute to a growing body of literature showing that myeloid neoplasms arising in the context of prior SOT represent a high-risk entity with generally poor survival. Further optimization of treatment approaches to establish a balance between efficacy and toxicity in this vulnerable population is warranted.

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Contributions

AB and GGM designed the study. CTRV, AB and KS collected the data. SL provided molecular data. CTRV and AB analyzed the data, performed the statistical analysis, generated the figure, and wrote the manuscript. GGM provided resources and supervision. All authors critically reviewed and edited the manuscript.

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Data-sharing statement

The data used for this study is not publicly available in order to protect patient confidentiality. Reasonable requests for de-identified data should be directed to the corresponding author.

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