haematologica
Journal of the Ferrata Storti Foundation

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

by Charan Thej Reddy Vegivinti, Guillermo Montalban-Bravo, Kelly Chien, Danielle Hammond, Koji Sasaki, Alex Bataller, Ian Bouligny, Wei Ying Jen, Mahesh Swaminathan, Sanam Loghavi, Uday Popat, Guillermo Garcia-Manero and Alexandre Bazinet

Received: March 10, 2026.
Accepted: May 5, 2026.

Citation: Charan Thej Reddy Vegivinti, Guillermo Montalban-Bravo, Kelly Chien, Danielle Hammond, Koji Sasaki, Alex Bataller, Ian Bouligny, Wei Ying Jen, Mahesh Swaminathan, Sanam Loghavi, Uday Popat, Guillermo Garcia-Manero and Alexandre Bazinet. Outcomes of patients diagnosed with myelodysplastic syndrome or acute myeloid leukemia following solid organ transplantation.
Haematologica. 2026 May 14. doi: 10.3324/haematol.2026.300856 [Epub ahead of print]

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

Charan Thej Reddy Vegivinti[1], Guillermo Montalban-Bravo[2], Kelly Chien[2], Danielle Hammond[2], Koji Sasaki[2], Alex Bataller[2], Ian Bouligny[2], Wei Ying Jen[2], Mahesh Swaminathan[2], Sanam Loghavi[3], Uday Popat[4], Guillermo Garcia-Manero[2], Alexandre Bazinet[2]

Author affiliations:
1-Department of Cancer Medicine, University of Texas MD Anderson Cancer Center, Houston, TX, USA
2-Department of Leukemia, University of Texas MD Anderson Cancer Center, Houston, TX, USA
3-Department of Hematopathology, University of Texas MD Anderson Cancer Center, Houston, TX, USA
4-Department of Stem Cell Transplantation and Cellular Therapy, University of Texas MD Anderson Cancer Center, Houston, TX, USA

Corresponding author:
Dr. Alexandre Bazinet
Department of Leukemia, The University of Texas MD Anderson Cancer Center
1515 Holcombe Blvd. Unit 428, Houston, TX 77030-4009
Email: abazinet@mdanderson.org
Tel: (713) 792-7430 Fax: (713) 794-4297

Running title: Outcomes of patients with MDS or AML following SOT

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.

Funding: This work was supported in part by the University of Texas MD Anderson Cancer Center Support Grant CA016672.

Author 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 figures, and wrote the manuscript. GGM provided resources and supervision. All authors critically reviewed and edited the manuscript.

Conflict of Interest Disclosure: GMB: Research Funding: IFM Therapeutics, Takeda. **KS:** Honoraria: Otsuka, Amgen, Chugai Pharma. Consulting or Advisory Role: Novartis, Pfizer, Takeda. Research Funding: Novartis, Enliven Therapeutics. Travel, Accommodations, Expenses: Otsuka, Amgen. **SL:** Research Funding: Amgen, Astellas. Consulting fees: AbbVie, AlphaSight, Arima, Blueprint Medicine, BMS, Caris, Daiichi-Sankyo, Gerson Lehrman Group, Guide Point, Immunogen, Kura Oncology, QualWorld, Recordati, Servier, Stemline Syndax, Tempus AI. **GGM:** Honoraria: Curis, Bristol Myers Squibb/Celgene, Taiho Oncology, Ascentage Pharma, Servier, Keros Therapeutics. Consulting or Advisory Role: Bristol Myers Squibb, Taiho Oncology, Curis, Ascentage Pharma, Servier, Keros Therapeutics.

Research Funding: Novartis, AbbVie, Bristol Myers Squibb, Genentech, Curis, Rigel, Taiho Oncology, Stemline Therapeutics, Zentalis, Chordia Therapeutics, Jazz Pharmaceuticals. **The remaining authors declare no competing interests.**

To the Editor:

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 (PTLDs) 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. 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 was collected when available and 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 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) in MDS and 58 years (33-66) 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%) patients, and heart in 1 (6%) patient. 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 CKD of unknown cause (n=1). Reasons for liver transplantation 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 (1-43.7) in MDS and 8.3 years (1.1-31.4) 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), 1 (13%) was monoallelic mutated and 7 (88%) were biallelic mutated. Notably, all patients with AML and available testing 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 (ESAs) in 2 (17%), luspatercept in 1 (8%), lenalidomide in 1 (8%), and observation alone in 1 (8%). Of these, only 4 patients were response evaluable per IWG 2023. The overall response rate in MDS was 50% (2/4 patients), including 1 CR and 1 CR_L, which occurred in HMA-treated patients. In AML, 4 (67%) patients received HMA-based therapy, 1 (17%) received cladribine, low-dose cytarabine, and venetoclax, and 1 (17%) received intensive chemotherapy ("7 + 3"). The overall response rate in AML was 33% (2/6 patients), comprising 1 CR with "7+3" and 1 CRp with decitabine + vosaroxin. Two (17%) patients in the MDS group and 1 (17%) patient in the AML group underwent allogeneic stem cell transplantation (SCT).

The survival outcomes were generally poor. With a median 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 (0.7-3.7). Two (17%) renal transplant recipients 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 (8%) renal transplant recipient 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 three patients who underwent SCT experienced a poor outcome. One SCT-recipient died on day +65 from pneumonia (in remission), one experienced primary hematopoietic graft failure and transitioned to hospice, and another experienced secondary hematopoietic graft failure and died on day +138.

Large epidemiological studies have demonstrated an increase in incidence of both MDS and AML in SOT recipients (standardized incidence ratio of 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 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 7 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 6 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 currently unknown whether the generally poor outcomes in post-SOT myeloid neoplasm reflect 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 1 patient received intensive chemotherapy. No transplanted organ failure was attributable to therapy, although 2 patients (17%) with renal transplantations 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.

SCT is a primary consolidative strategy in higher risk MDS and AML. Although only 3 patients underwent SCT in our series, precluding 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 small sample size, heterogeneity, single center data, and retrospective design. Furthermore, not all patients underwent next-generation sequencing with the full 81-gene panel and complete data on immunosuppressive regimens were missing. Overall, our data contributes 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 balancing efficacy versus toxicity in this vulnerable population is warranted.

REFERENCES

1. Schladt DP, Israni AK. OPTN/SRTR 2021 annual data report: introduction. Am J Transplant. 2023;23(2 Suppl 1):S12-S20.

2. Engels EA, Pfeiffer RM, Fraumeni JF Jr, et al. Spectrum of cancer risk among US solid organ transplant recipients. JAMA. 2011;306(17):1891-901.

3. Wang Z, Deng L, Hou W, et al. Cancer mortality among solid organ transplant recipients: A systematic review and meta-analysis. Prev Med. 2024;189:108161.

4. Dillibabu T, Laprise C, Nicolau B, Madathil S. Risk of cancer among adult solid organ transplant recipients in Quebec, Canada: 1997-2016. BMC Cancer. 2025;25(1):1004.

5. Dharnidharka VR. Comprehensive review of post-organ transplant hematologic cancers. Am J Transplant. 2018;18(3):537-549.

6. Dohner H, Wei AH, Appelbaum FR, et al. Diagnosis and management of AML in adults: 2022 recommendations from an international expert panel on behalf of the ELN. Blood. 2022;140(12):1345-1377.

7. Greenberg PL, Tuechler H, Schanz J, et al. Revised international prognostic scoring system for myelodysplastic syndromes. Blood. 2012;120(12):2454-2465.

8. Bernard E, Tuechler H, Greenberg PL, et al. Molecular international prognostic scoring system for myelodysplastic syndromes. NEJM Evid. 2022;1(7):EVIDoa2200008.

9. Zeidan AM, Platzbecker U, Bewersdorf JP, et al. Consensus proposal for revised International Working Group 2023 response criteria for higher-risk myelodysplastic syndromes. Blood. 2023;141(17):2047-2061.

10. Morton LM, Gibson TM, Clarke CA, et al. Risk of myeloid neoplasms after solid organ transplantation. Leukemia. 2014;28(12):2317-2323.

11. Offman J, Opelz G, Doehler B, et al. Defective DNA mismatch repair in acute myeloid leukemia/myelodysplastic syndrome after organ transplantation. Blood. 2004;104(3):822-828.

12. Wu B, Ingersoll K, Jug R, et al. Myeloid neoplasms following solid organ transplantation: clinicopathologic studies of 23 cases. Am J Clin Pathol. 2017;149(1):55-66.

13. Lontos K, Agha M, Raptis A, et al. Outcomes of patients diagnosed with acute myeloid leukemia after solid organ transplantation. Clin Transplant. 2017;31(9):e13052.

14. Menes M, Vakiani E, Keller CE, et al. The spectrum of myelodysplastic syndromes post-solid organ transplantation: a single institutional experience. Leuk Res. 2007;31(1):59-65.

15. Doney KC, Mielcarek M, Stewart FM, Appelbaum FR. Hematopoietic cell transplantation after solid organ transplantation. Biol Blood Marrow Transplant. 2015;21(12):2123-2128.

TABLES

Table 1: Baseline patient characteristics

	Myelodysplastic Syndrome n=12	**Acute Myeloid Leukemia n=6**
Age (years)	67 [50-77]	58 [33-66]
Male	8 (67)	3 (50)
Time from transplant to MN (years)	8.6 [1-43.7]	8.3 [1.1-31.4]
Organ transplant type		
Kidney	9 (75)	5 (83)
Liver	2 (17)	1 (17)
Heart	1 (8)	0 (0)
Bone marrow blasts (%)	2 [1- 17]	26 [20-40]
Hemoglobin (g/dL)	8.8 [7.1-11.8]	9.15 [7.4-12.5]
Platelets ($x10^9$/L)	99 [30-453]	59 [34-137]
Absolute neutrophil count ($x10^9$/L)	1.86 [0.81-6.00]	0.93 [0.00-2.80]
IPSS-R risk		
Low	2 (17)	-
Intermediate	5 (42)	-
High	3 (25)	-
Very High	2 (17)	-
IPSS-M risk		
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		
Intermediate	-	1 (17)
Adverse	-	5 (83)
Cytogenetic risk (IPSS-R/ELN 2022)		
Good/Favorable	6 (50)	0 (0)
Intermediate/Intermediate	1 (8)	1 (17)
Poor/Adverse	2 (17)	5 (83)
Very Poor	3 (25)	-
Complex cytogenetics	4 (33)	5 (83)
***TP53* status**		
Wild type	9 (75)	0/5 (0)
Monoallelic mutated	1 (8)	0/5 (0)
Biallelic mutated	2 (17)	5/5 (100)

Data shown as n (%) or median [range].

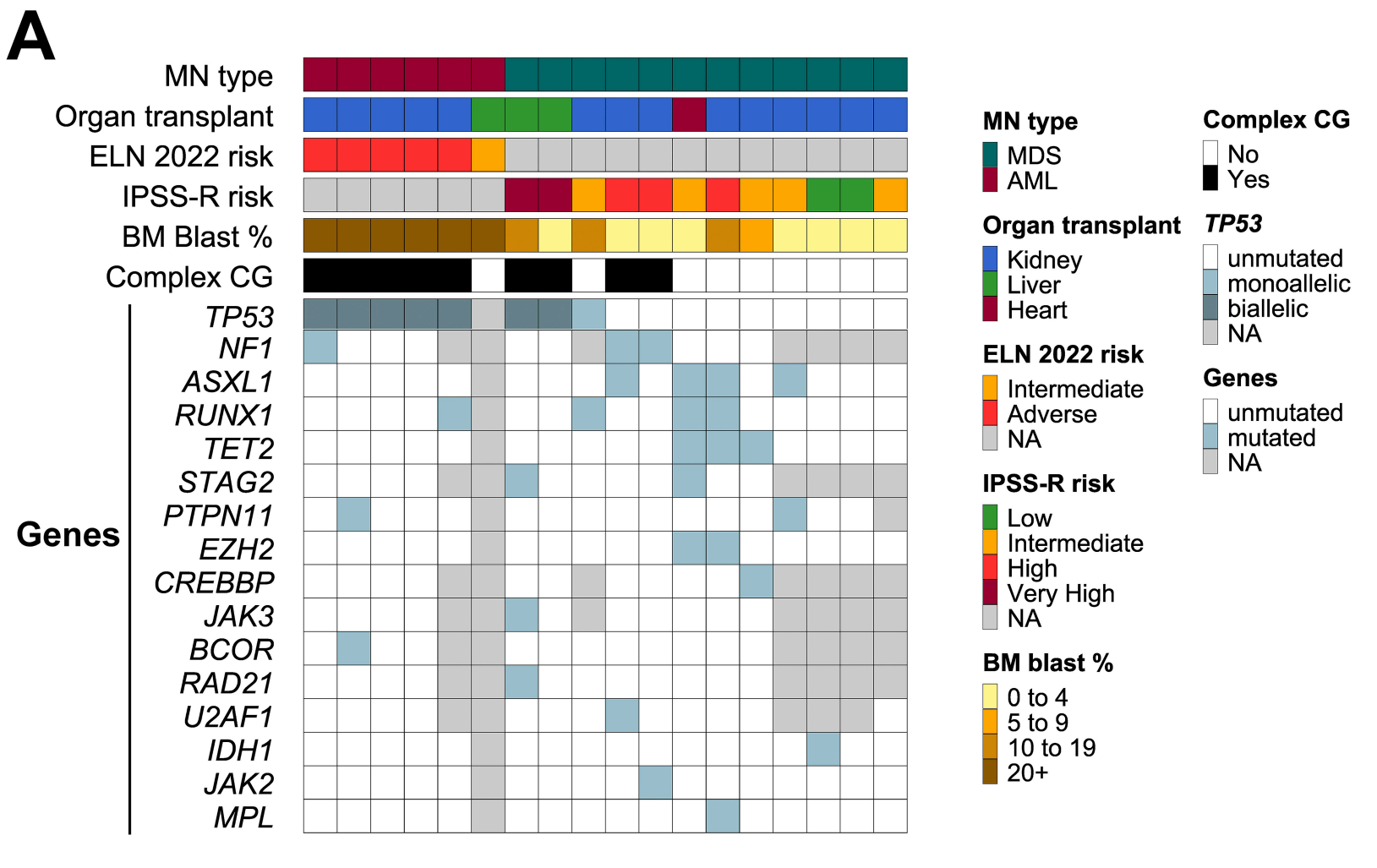
Abbreviations: ELN, European LeukemiaNet; IPSS-M, molecular International Prognostic Scoring System; IPSS-R, revised International Prognostic Scoring System; MN, myeloid neoplasm.

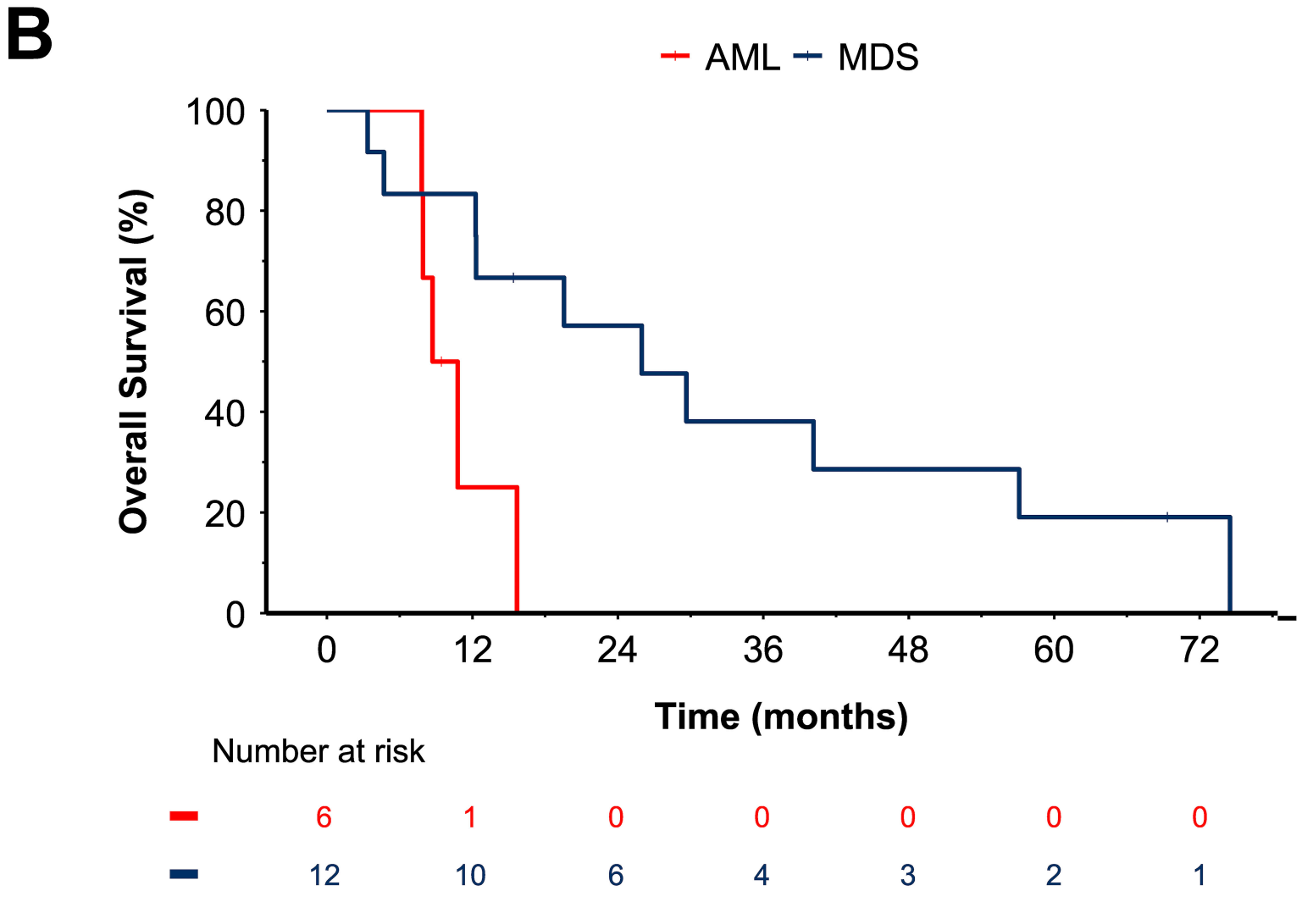
Table 2: Treatment and outcome by patient

ID	Age/Sex	Organ transplant	Diagnosis	IPSS-R/ELN 2022 risk	Cytogenetics	Mutations	Therapy	Response	SCT	Outcome
1	70M	Kidney	MDS	Low	Normal	negative	Darbepoetin alfa	-	no	Death from unknown cause.
2	52M	Kidney	MDS	Low	del(20q)	*IDH1*	Decitabine	-	no	Death from unknown cause.
3	72M	Kidney	MDS	Intermediate	del(7q)	*ASXL1*, *PTPN11*	none	-	no	Death from unknown cause.
4	50F	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	77F	Liver	MDS	Very High	Complex	*TP53* (bi)	Decitabine	NR	no	Death with refractory disease.
6	58F	Kidney	MDS	High	Complex	*JAK2*, *NF1*	Azacitidine	-	no	Death with refractory disease.
7	52F	Kidney	MDS	Intermediate	del(5q)	negative	Lenalidomide	-	no	Alive at 5.8 years.
8	62M	Kidney	MDS	Intermediate	Normal	*RUNX1*, *TP53* (mono)	Decitabine + venetoclax	CR	yes	Death from secondary graft failure on SCT D+138.
9	69M	Kidney	MDS	Intermediate	Normal	*CREBBP*, *TET2*	Darbepoetin alfa	-	no	Death from prostate cancer.
10	75M	Heart	MDS	Intermediate	add(21q)	*ASXL1*, *EZH2*, *RUNX1*, *STAG2*, *TET2*	Azacitidine	-	no	Death with refractory disease.
11	65M	Kidney	MDS	High	Normal	*ASXL1*, *EZH2*, *MPL*, *RUNX1*, *TET2*	Decitabine/cedazuridine	CR_L	no	Alive at 1.3 years.
12	75M	Kidney	MDS	High	Complex	*ASXL1*, *NF1*, *U2AF1*	Luspatercept	-	no	Death with refractory disease.
13	66F	Kidney	AML	Adverse	Complex	*TP53* (bi), *RUNX1*	Decitabine + vosaroxin	CRp	yes	Death in remission from pneumonia on SCT D+65.
14	61M	Liver	AML	Intermediate	Normal	N/A	7 + 3	CR	no	Death following relapse.
15	33M	Kidney	AML	Adverse	Complex	*TP53* (bi)	Decitabine	NR	no	Death with refractory disease.
16	52F	Kidney	AML	Adverse	Complex	*NF1*, *TP53* (bi)	Azacitidine + venetoclax	NR	no	Death with refractory disease.
17	54F	Kidney	AML	Adverse	Complex	*TP53* (bi)	Azacitidine + anti-CD47	NR	no	Lost to follow-up.
18	62M	Kidney	AML	Adverse	Complex	*BCOR*, *PTPN11*, *TP53* (bi)	Cladribine + low-dose cytarabine + Venetoclax	NR	no	Death with refractory disease.

Abbreviations: AML, acute myeloid leukemia; CR, complete remission; CR_L, complete remission with limited count recovery; CRp, complete remission with incomplete platelet recovery; ELN, European LeukemiaNet; IPSS-R, revised International Prognostic Scoring System; MDS, myelodysplastic syndrome; NR, no response; SCT, stem cell transplantation.

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 (VAF) >50%, two *TP53* mutations, or a *TP53* mutation with concurrent deletion of chromosome 17p. **(B)** Kaplan-Meier estimates of overall survival in patients with MDS and AML after solid organ transplantation.


A
MN type
Organ transplant
ELN 2022 risk
IPSS-R risk
BM Blast %
Complex CG
Genes
TP53
NF1
ASXL1
RUNX1
TET2
STAG2
PTPN11
EZH2
CREBBP
JAK3
BCOR
RAD21
U2AF1
IDH1
JAK2
MPL
MN type
MDS
AML
Organ transplant
Kidney
Liver
Heart
ELN 2022 risk
Intermediate
Adverse
NA
IPSS-R risk
Low
Intermediate
High
Very High
NA
BM blast %
0 to 4
5 to 9
10 to 19
20+
Complex CG
No
Yes
TP53
unmutated
monoallelic
biallelic
NA
Genes
unmutated
mutated
NA


B
AML
MDS
Overall Survival (%)
100
80
60
40
20
0
0
12
24
36
48
60
72
Time (months)
Number at risk
6 1 0 0 0 0 0
12 10 6 4 3 2 1