Integrative clinical and molecular characterization of an acute promyelocytic leukemia-like subgroup with high early death risk in newly diagnosed acute myeloid leukemia: a multicenter study

Acute promyelocytic leukemia (APL)-like leukemia is a subset of non-APL acute myeloid leukemia (AML) which has gained increasing attention, displaying a CD34 and HLA-DR double-negative immunophenotype that resembles that of APL. While previous studies of APL-like AML (APLL) focused on *NPM1* mutations,[1-3] further molecular characterization of APLL has not been extensively elucidated. Moreover, clinical outcomes of APLL, particularly early outcomes, have not been clearly presented. Therefore, on the basis of the largest APLL cohort to date, we present the features of a unique APLL subgroup of AML with distinct molecular profiles and elevated risk of early death. In this multicenter study, we screened consecutive patients diagnosed with AML between January 2017 and November 2022. A cohort of 871 newly diagnosed AML patients with complete clinical data were included in this study. APLL was defined as AML with an immunophenotype of CD34 and HLA-DR double negativity on the basis of a 20% threshold in flow cytometry analysis, along with the absence of retinoic acid receptor $\alpha/\beta/\gamma$ (*RARA/RARB/RARG*) rearrangements. Early death was defined as death from any cause within 60 days after initial diagnosis. This study was approved by the Ethics Committee of the First Affiliated Hospital of Soochow University and was conducted in accordance with the principles of the Declaration of Helsinki.

Of the 871 newly diagnosed AML patients, 241 (27.7%) were defined as having APLL according to immunophenotypic features, 123 (14.1%) as having APL by detection of a *PML*::*RARA* fusion and 507 (58.2%) as 'Other AML'. The clinicopathological characteristics of the study cohort are summarized in Table 1. Of the 241 APLL patients, 131 (54.4%) were male and their median age was 50 years (interquartile range, 38-58 years). One-hundred and eight (44.8%) APLL patients had overt disseminated intravascular coagulopathy (DIC) at diagnosis, a proportion significantly higher than the 15.8% in 'Other AML' (P<0.001). Similarly, the prevalence of major bleeding/thrombotic events before induction within the APLL group was significantly higher than in the 'Other AML' group (9.5% *vs*. 0.6%, P<0.001).

APLL patients had a significantly higher blast burden (71.3% *vs*. 59.0%, P<0.001) than 'Other AML' patients and distinct immunophenotypic features beyond the double negativity. For genomic alterations, *NPM1* mutation predominated in APLL and was significantly more common than in 'Other AML' (78.8% *vs*. 24.4%, P<0.001) (Figure 1A). Besides, cases of *NPM1*-mutated APLL presented significantly higher *NPM1* mutation variant allele frequencies than *NPM1*-mutated 'Other AML' (40.1% *vs*. 37.7%, P=0.016) (*Online Supplementary Figure S1A*). Regarding mutations of various signaling pathways, APLL exhibited dominant prevalence in functional pathways including transcription factors, activated signaling and epigenetic regulators (*Online Supplementary Figure S1B*). Within APLL, co-mutation analysis (*Online Supplementary Figure S1C*) demonstrated overlapping mutations of *NPM1* with *IDH1/2* (P<0.001), *DNMT3A* (P<0.001), *FLT3*-ITD (P=0.002) and *TET2* (P=0.012). *NPM1* mutations were mutually exclusive with *NRAS/KRAS* mutations (P=0.044). Apart from the central role of *NPM1* mutations, there was a series of additional molecular subtypes within APLL (*Online Supplementary Figure S1D*), including AML with *KMT2A* rearrangement (4.6%), core binding factor AML (4.1%), AML with *CEBPA* mutation (3.7%), etc.

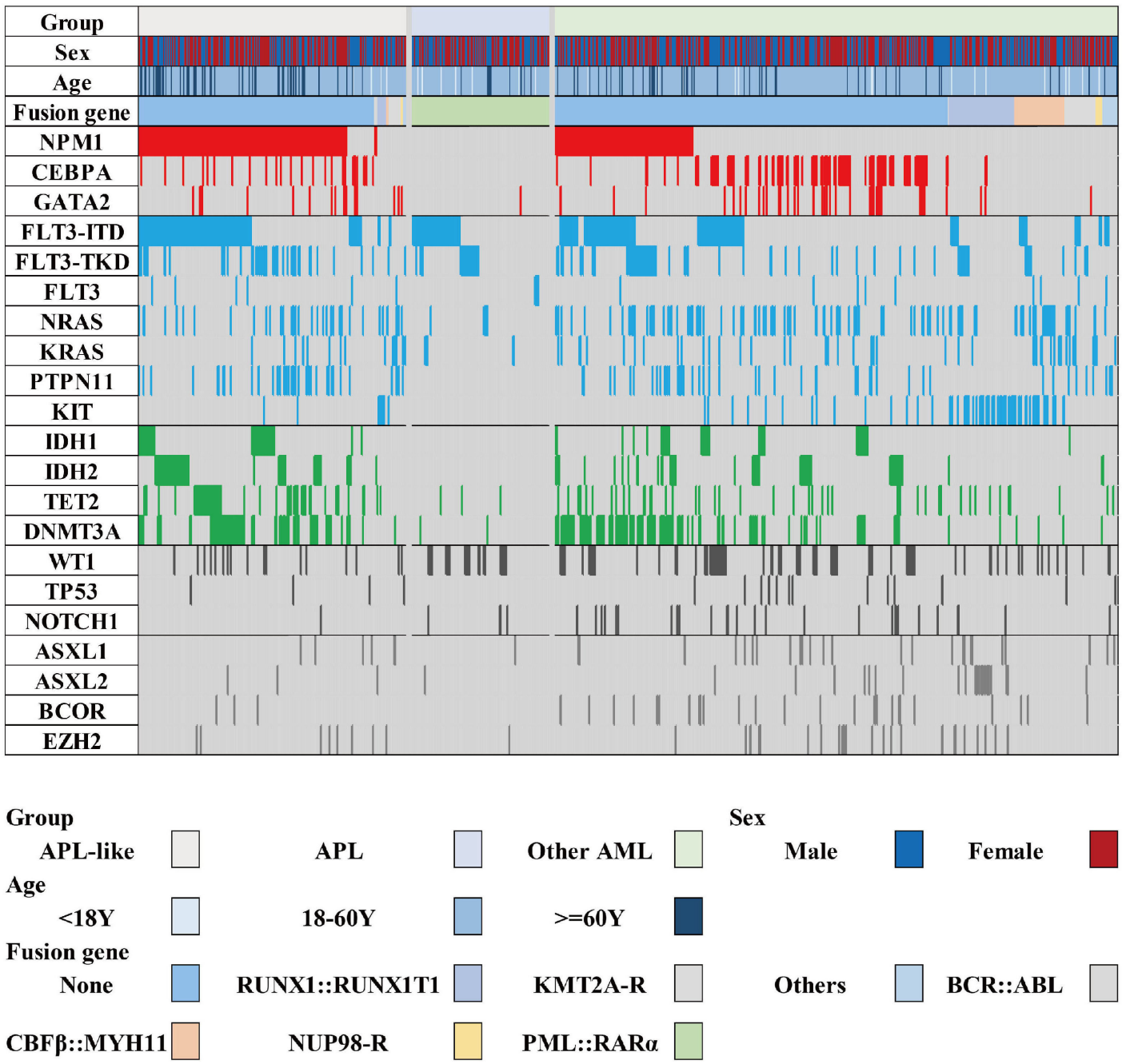
RNA-sequencing analysis was performed to explore transcriptomic characteristics of APLL. Gene Ontology analysis of differentially expressed genes indicated upregulation of myeloid cell differentiation pathways comparing 'Other AML' patients and healthy donors (*Online Supplementary Figure S1E, F*). Besides, Gene set enrichment analysis revealed that the APL gene set was upregulated in APLL (*Online Supplementary Figure S1G*). Next, unsupervised hierarchical clustering of the gene expression profile was performed. It was found that 34 of 52 (65.4%) APLL samples were clustered together, suggesting a potential distinct entity (Figure 1B). Based on the distribution of APLL samples within the cluster, we interpreted it as two parts. Thirty-one of 34 (91.2%) APL-like samples were clustered as a consecutive part of Ga. For the rest of the cluster (Gb), whose data were primarily from the BeatAML database, we found that differentially expressed genes of Gb against other BeatAML data were similar to those of Ga compared to other AML (*Online Supplementary Figure S1H, I*). This indicated that Gb exhibited similar gene expression profiles to Ga. The analysis of 38 patients with available information from Gb demonstrated that these patients also presented APL-like immunophenotypic features (*Online Supplementary Figure S1J*). We assumed that patients from Gb met the criteria of APLL and the whole cluster represented an APLL cluster, further indicating that APLL

Table 1. Clinicopathological characteristics of acute promyelocytic leukemia (APL)-like acute myeloid leukemia (AML), APL and 'Other AML'.

Parameter	APL-like AML N=241	APL N=123	Other AML N=507	*P* (APL-like AML *vs.* APL)	*P* (APL-like AML *vs.* Other AML)	*P* (APL *vs.* Other AML)
Age, years, median (IQR)	50.0 (38.0-58.0)	35.0 (28.0-48.0)	42.0 (32.0-53.0)	<0.001	< 0.001	0.002
Male sex, N (%)	131 (54.4)	62 (50.4)	241 (47.5)	0.475	0.081	0.567
ECOG score, N (%)				0.814	<0.001	<0.001
0-2	206 (85.5)	104 (84.6)	477 (94.1)	-	-	-
3-4	35 (14.5)	19 (15.4)	30 (5.9)	-	-	-
Complete blood count, median (IQR)						
WBC, x10^9/L	48.61 (15.51-103.23)	4.17 (1.29-20.81)	20.75 (5.13-64.19)	<0.001	<0.001	<0.001
Hemoglobin, g/L	80.0 (70.0-97.0)	90.0 (73.0-113)	80.0 (65.0-98.0)	0.001	0.415	<0.001
Platelets, x10^9/L	49.0 (27.5-94.5)	25.0 (14.0-47.0)	39.0 (20.0-76.0)	<0.001	0.008	<0.001
Biochemistry, median (IQR)						
LDH, U/L	489.1 (284.7-857.3)	335.0 (210.0-575.0)	427.8 (257.5-666.0)	<0.001	0.005	0.008
Albumin, g/L	36.00 (32.35-40.00)	41.90 (37.08-44.55)	35.20 (32.05-38.55)	<0.001	0.148	<0.001
Coagulation, N (%)						
Overt DIC[a]	108 (44.8)	110 (89.4)	80 (15.8)	<0.001	<0.001	<0.001
MBE/MTE before induction[b]	23 (9.5)	16 (13.0)	3 (0.6)	0.312	<0.001	<0.001
Morphology, median (IQR)						
BMB or BMAP, %	71.3 (49.0-85.0)	84.0 (75.5-90.3)	59.0 (40.5-77.5)	<0.001	<0.001	<0.001
Immunophenotype, N (%)						
CD13 positive	187 (77.6)	114 (92.7)	463 (91.3)	<0.001	<0.001	0.626
CD33 positive	237 (98.3)	121 (98.4)	487 (96.1)	0.981	0.097	0.209
CD117 positive	180 (74.7)	91 (74.0)	451 (89.0)	0.884	<0.001	<0.001
CD11b positive	57 (23.7)	19 (15.4)	116 (22.9)	0.069	0.815	0.072
CD4 positive	73 (30.3)	26 (21.1)	88 (17.4)	0.063	<0.001	0.328
CD7 positive	31 (12.9)	2 (1.6)	164 (32.3)	<0.001	<0.001	<0.001
CD56 positive	74 (30.7)	11 (8.9)	89 (17.6)	<0.001	<0.001	0.019
Fusion genes, N (%)						
KMT2A-rearrangement	11 (4.6)	0 (0)	34 (6.7)	0.019	0.250	0.001
CBF-AML	10 (4.1)	0 (0)	104 (20.5)	0.019	<0.001	<0.001
Other fusion	7 (2.9)	123 (100)	20 (3.9)	<0.001	0.476	<0.001
Somatic mutations, N (%)						
NPM1	190 (78.8)	0 (0)	124 (24.4)	<0.001	<0.001	<0.001
DNMT3A	75 (31.1)	2 (1.6)	116 (22.9)	<0.001	0.016	<0.001
IDH1/2	87 (36.1)	0 (0)	84 (16.6)	<0.001	<0.001	<0.001
FLT3-ITD	117 (48.5)	43 (35.0)	129 (25.4)	0.014	<0.001	0.034
CEBPA	25 (10.4)	0 (0)	106 (20.9)	<0.001	<0.001	<0.001
TET2	55 (22.8)	3 (2.4)	46 (9.1)	<0.001	<0.001	0.014
NRAS/KRAS	47 (19.5)	10 (8.1)	135 (26.6)	0.005	0.034	<0.001
PTPN11	35 (14.5)	0 (0)	46 (9.1)	<0.001	0.025	<0.001
WT1	17 (7.1)	23 (18.7)	84 (16.6)	<0.001	<0.001	0.572
ELN-risk 2022, N (%)				-	<0.001	-
Favorable	104 (43.9)	-	237 (48.2)	-	-	-
Intermediate	110 (46.4)	-	151 (30.7)	-	-	-
Adverse	23 (9.7)	-	104 (21.1)	-	-	-
Induction regimen, N (%)				-	0.009	-
"3+7" regimen	91 (39.6)	-	258 (51.3)	-	-	-
Low-intensity chemotherapy	74 (32.2)	-	120 (23.9)	-	-	-
Venetoclax + HMA	65 (28.3)	-	125 (24.9)	-	-	-
FLT3 inhibitor in induction, N (%)	22 (9.6)	-	12 (2.4)	-	<0.001	-

[a]Overt disseminated intravascular coagulation was retrospectively evaluated according to the 2018 revision of International Society on Thrombosis and Haemostasis (ISTH) guidelines. [b]Major bleeding events and major thrombotic events were defined by > grade 2 events of the revised World Health Organization bleeding scale and Common Terminology Criteria for Adverse Events version 5.0: respectively. APL: acute promyelocytic leukemia; AML: acute myeloid leukemia; IQR: interquartile range; ECOG: Eastern Cooperative Oncology Group; WBC: white blood cells; LDH: lactate dehydrogenase; DIC: disseminated intravascular coagulation; MBE: major bleeding event; MTE: major thrombotic event; BMB: bone marrow blasts; BMAP: bone marrow abnormal promyelocytes; CBF: core binding factor; ELN: European LeukemiaNet; ; HMA: hypomethylating agent.

A



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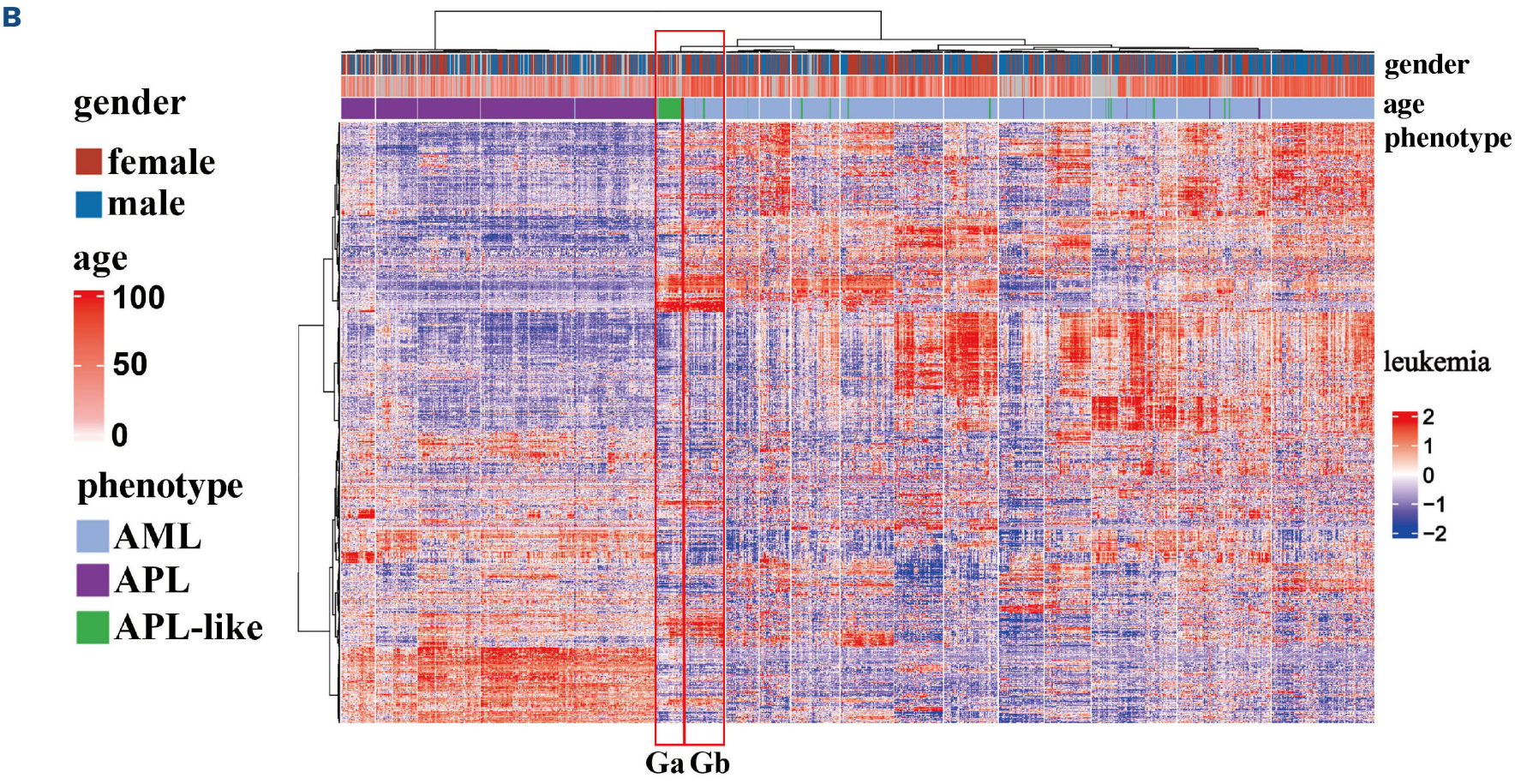


Figure 1. Genomic and transcriptomic landscape of acute promyelocytic leukemia-like acute myeloid leukemia. (A) Mutational landscape of acute promyelocytic leukemia (APL)-like acute myeloid leukemia (AML), APL and 'Other AML'. The upper panel shows the classification of the study, sex, age and fusion gene for each patient. The mutational landscape of common genes is displayed as the principal part of the figure. (B) Unsupervised hierarchical clustering of the gene expression pattern identifies a cluster in which 34 of 52 (65.4%) APL-like AML samples were involved. The cluster is interpreted as Ga and Gb. Thirty-one of 34 (91.2%) APL-like AML were interpreted as Ga. Gb is mainly composed of cases from the BeatAML database. Top: Gender, age and phenotype of each sample. Columns indicate cases, and rows indicate genes.

might be a distinct entity from a transcriptomic aspect.

Regarding early outcomes of APLL, at a median time of 17 days (range, 2-56) from initial diagnosis, early death had occurred in 31 of 237 (13.1%) APLL patients, which was a significantly higher rate than in 'Other AML' (20/505, 3.9%; *P*<0.001) and APL (7/123, 5.7%; *P*=0.031) (Figure 2A). Among 31 APLL patients who died early, 22 (71.0%) died of major bleeding/thrombotic events, which was in line with the proportion in APL patients (5/7, 71.4%). In contrast, the causes of early death among patients with 'Other AML' were primarily attributed to infections (15/20, 75.0%) (Figure 2B). This distribution was significantly different from that in APLL (*P*=0.001).

Multivariate analysis identified five predictive factors for early death in APLL (*Online Supplementary Table S1*): male sex (hazard ratio [HR]= 0.432, 95% confidence interval [95% CI]: 0.197-0.950, *P*= 0.037), age ≥60 years (HR=3.031, 95% CI: 1.446-6.352, *P*=0.003), albumin <35 g/L (HR=3.999, 95% CI: 1.846-8.661, *P*<0.001), overt DIC at diagnosis (HR=3.736, 95% CI: 1.557-8.966, *P*=0.003) and major bleeding/thrombotic events before induction (HR=9.995, 95% CI: 4.626-21.594, *P*<0.001). All these factors were independent of those for 'Other AML' except for age ≥60 years.

Utilizing regression coefficients from multivariate analysis, we developed a nomogram to accurately predict risks of early death in APLL, as is shown in Figure 2C. The assigned points for each variable were as follows: 38.0 for female sex, 49.2 for age ≥60 years, 62.2 for albumin <35 g/L, 57.8 for overt DIC at diagnosis and 100.0 for major bleeding/thrombotic events before induction. An external cohort of 65 APLL patients from Suzhou Hongci Hospital was collected as our validation cohort and performance analysis presented acceptable efficacy in both the development and validation cohorts (*Online Supplementary Figure S2*). Next, 157.8 was determined as the optimal cutoff point for discriminating low- and high-risk groups of APLL patients. Based on this stratification standard, the high-risk group exhibited an inferior early death rate in both the development cohort (55.0% *vs*. 4.6%, *P*<0.001) (Figure 2D) and the validation cohort (53.8% *vs*. 1.9%) (*P*<0.001) (Figure 2E).

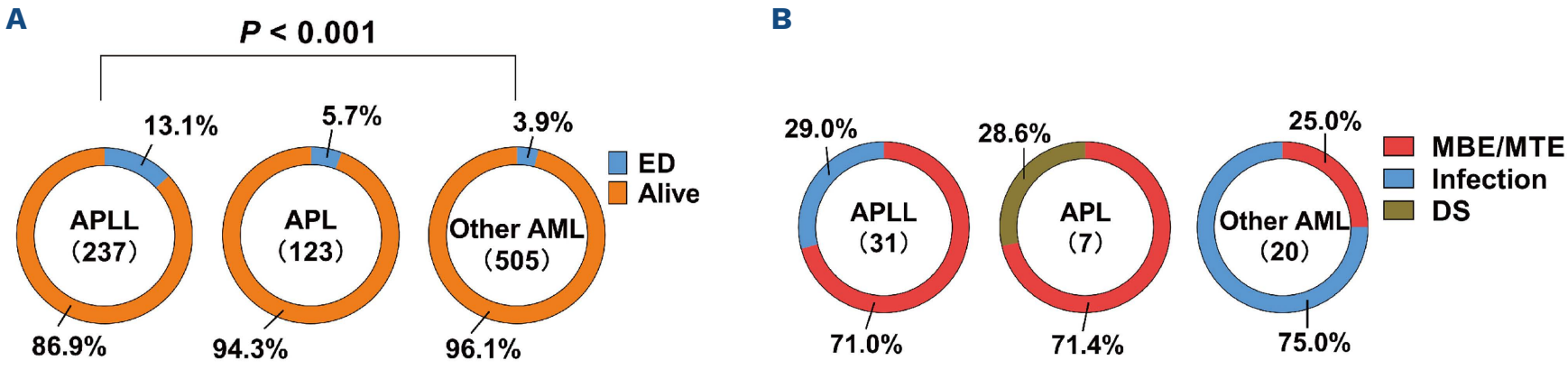
Univariate analysis was then conducted to uncover predictors of early death among high-risk APLL patients (Figure 2F). Surprisingly, we found that treatment with a venetoclax plus hypomethylating agent regimen was a risk factor for early death among these patients (HR=3.022, 95% CI: 1.263-7.243, *P*=0.013), while low-intensity chemotherapy played a protective role (HR= 0.300, 95% CI: 0.101-0.888, *P*=0.030).

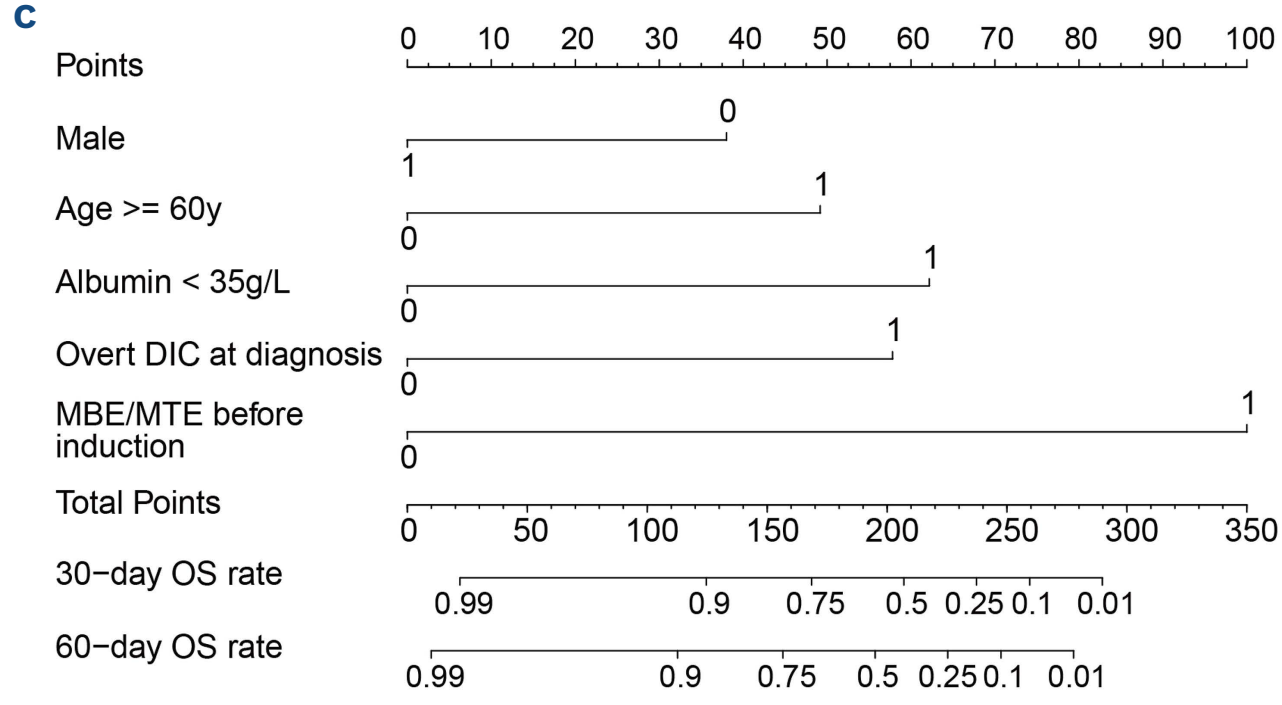
In this large-cohort, multicenter study, we focused on investigation of molecular profiles and early outcomes of APLL. Transcriptomic analysis of APLL has barely been reported. Here, on the basis of the largest set of APLL RNA-sequencing samples to date, we identified upregulation of APL-related gene sets and myeloid cell differentiation pathways in APLL samples, indicating a similar molecular biology between APLL and APL. However, 65.4% of APLL samples were clustered together and were distinct from classic APL and 'Other AML' clusters, suggesting a potential unique subtype. Supplementary information from the BeatAML database, particularly immunophenotypic features, further supported that the cluster was possibly a specific cluster of APLL. These findings suggest that while APLL may exhibit some similarities with APL from the standpoint of molecular pathogenesis, it is likely a distinct entity and the differentiation mechanism may be distinguished from classic APL.

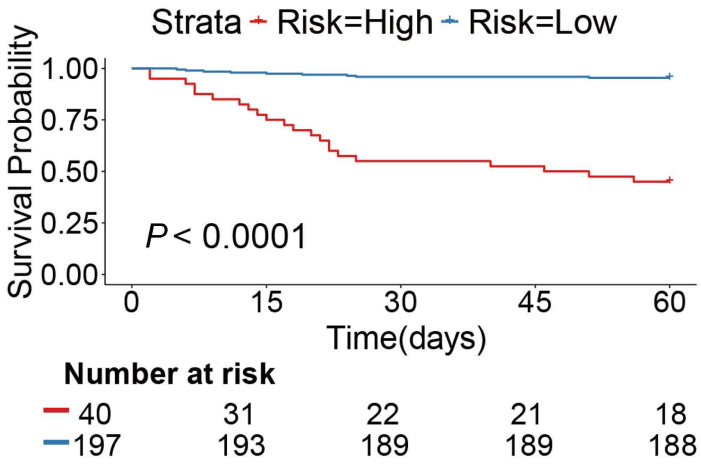
In our APLL cohort, the early death rate reached 13.1%, significantly higher than that of APL and 'Other AML', indicating that APLL is a unique subgroup with an elevated risk of early death. It is worth noting that 71.0% of early deaths in APLL patients were attributed to major bleeding/thrombotic events, a rate that not only is comparable to that of our APL group (71.4%), but also basically aligns with some of recent findings in APL.[4,5] We also elucidated the predictors of early death among APLL patients. Overt DIC at diagnosis was identified as a risk factor for early death in APLL. A recent investigation by Paterno *et al*.[6] identified DIC as one of the predictors of early mortality within non-APL AML. In our study, we confirmed it was a risk factor that was independent of those for 'Other AML'. Similarly, major bleeding/thrombotic events before induction contributed to the risk of early death in APLL. In contrast, infection at diagnosis was an independent risk factor in 'Other AML', demonstrating distinct patterns of early death in the two subgroups. It can be concluded that coagulation dysfunction specifically plays a central role in APLL patients' inferior early outcomes.

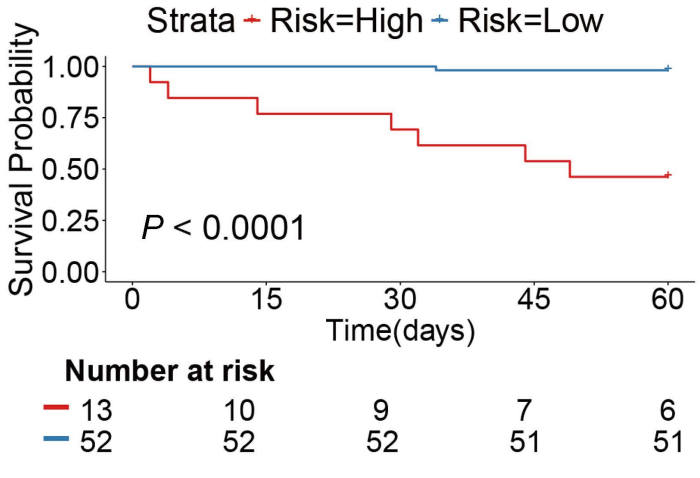
A surprising finding of our analysis is that choice of induction regimen might have an impact on early deaths among high-risk APLL patients, as venetoclax plus hypomethylating agent regimens increased early death risks within these patients. While venetoclax plus a hypomethylating agent has been a standard induction regimen for elderly or unfit AML patients,[7-9] it has been noted that the regimen also triggers tumor lysis syndrome, especially with the standard "ramp-up" dosage among AML patients with *IDH1/2* or *NPM1* mutations.[10-12] This might be associated with our finding, as potential tumor lysis syndrome could further impair poor fitness status and deteriorate coagulation dysfunction in high-risk APLL patients. A recent study revealed that reduced venetoclax exposure alleviated 8-week mortality rate in an AML cohort while having no impact on long-term survival.[13] Studies are also emerging on modification of venetoclax dosage to reduce adverse events.[14,15] Therefore, further prospective study may be important to determine appropriate induction regimen options and dosages among high-risk APLL patients.

In conclusion, we show that APLL is a distinct subgroup









F

Subgroup	N (%)	HR (95%CI)	P
Age (≥60y)	20 (37.7)	1.339 (0.639~2.807)	0.440
Sex (Male)	15 (28.3)	0.870 (0.371~2.041)	0.750
Overt DIC	50 (94.3)	0.755 (0.179~3.182)	0.702
Mutation			
NPM1	43 (81.1)	0.862 (0.351~2.117)	0.745
IDH1/2	18 (34.0)	0.372 (0.151~0.918)	0.032
TET2	21 (39.6)	1.291 (0.620~2.690)	0.434
Induction			
"3+7"	10 (18.9)	0.851 (0.288~2.517)	0.771
LIC	16 (30.2)	0.300 (0.101~0.888)	0.030
VEN-HMA	20 (37.7)	3.022 (1.263~7.234)	0.013

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Figure 2. Details and predictive nomogram of early death within acute promyelocytic leukemia-like acute myeloid leukemia. (A) Pie charts showing the 60-day survival and early death (ED) rate of acute promyelocytic leukemia-like acute myeloid leukemia (APLL), acute promyelocytic leukemia (APL) and other forms of acute myeloid leukemia ('Other AML'). (B) Pie charts showing the distribution of ED causes within APLL, APL and 'Other AML' patients. (C) The ED predictive nomogram for APLL patients. (D) Kaplan-Meier curves for 60-day survival of the high- and low-risk APLL patients categorized by the score of the nomogram in the development cohort. (E) Kaplan-Meier curves for 60-day survival of the high- and low-risk APLL patients categorized by the score of the nomogram in the external validation cohort. (F) Forest plot for univariate analysis results of predictors of ED in high-risk APLL patients. MBE: major bleeding event; MTE: major thrombotic event; DS: differentiation syndrome; DIC: disseminated intravascular coagulation; OS: overall survival; HR: hazard ratio; 95%CI: 95% confidence interval; LIC: low-intensity chemotherapy; VEN: venetoclax; HMA: hypomethylating agent.

of AML, provide the first elucidation of its unique transcriptomic profile, and offer a risk-stratification model for identifying patients at high risk of early death. These findings facilitate understanding of this subset of patients and advance guidelines for their management. Nevertheless, due to the limitation of retrospective nature of the study, biological research of APLL pathogenesis and optimal management of this subtype of leukemia await exploration in the future.

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Disclosures

No conflicts of interest to disclose.

Contributions

SC, MS, WD and LWen contributed to the research design. MS and WD collected data, performed the statistical analysis and prepared the manuscript. LinW, ZW, LijW, YunW and XD helped with the collection of data. MZ, DL and HS reviewed morphology, flow cytometry and molecular data. XM, YZ, YX, YingW, SX, JF, HQ, XT, YH and DW collected samples and treated the patients. JH supported the RNA-sequencing data analysis. HD, LWen and SC contributed to the revision of the manuscript and supervision of the study. All authors approved the manuscript and had final responsibility for the decision to submit it for publication.

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Data-sharing statement

The data reported in the study are available from the corresponding author on reasonable request.

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References

1. Mason EF, Kuo FC, Hasserjian RP, Seegmiller AC, Pozdnyakova O. A distinct immunophenotype identifies a subset of NPM1-mutated AML with TET2 or IDH1/2 mutations and improved outcome. Am J Hematol. 2018;93(4):504-510.
2. Mason EF, Hasserjian RP, Aggarwal N, Seegmiller AC, Pozdnyakova O. Blast phenotype and comutations in acute myeloid leukemia with mutated NPM1 influence disease biology and outcome. Blood Adv. 2019;3(21):3322-3332.
3. Chen EC, Shimony S, Luskin MR, Stone RM. Biology and management of acute myeloid leukemia with mutated NPM1. Am J Hematol. 2025;100(4):652-665.
4. Voso MT, Guarnera L, Lehmann S, et al. Acute promyelocytic leukemia: long-term outcomes from the HARMONY project. Blood. 2025;145(2):234-243.
5. Zhao Y, Yu X, Yan L, et al. Study on clinical features of early death in older patients with acute promyelocytic leukemia. Ann Hematol. 2025;104(1):183-192.
6. Paterno G, Palmieri R, Tesei C, et al. The ISTH DIC-score predicts early mortality in patients with non-promyelocytic acute myeloid leukemia. Thromb Res. 2024;236:30-36.
7. Döhner 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.
8. Bhansali RS, Pratz KW, Lai C. Recent advances in targeted therapies in acute myeloid leukemia. J Hematol Oncol. 2023;16(1):29.
9. Döhner H, DiNardo CD, Appelbaum FR, et al. Genetic risk classification for adults with AML receiving less-intensive therapies: the 2024 ELN recommendations. Blood. 2024;144(21):2169-2173.
10. Wei AH, Loo S, Daver N. How I treat patients with AML using azacitidine and venetoclax. Blood. 2025;145(12):1237-1250.
11. Rowe M, Babushok D, Carroll M, et al. Tumor lysis syndrome in acute myeloid leukemia patients treated with a venetoclax based regimen. Eur J Haematol. 2025;114(4):626-635.
12. Diao S, Nichols ED, DiNardo C, et al. Incidence of tumor lysis syndrome in patients with acute myeloid leukemia undergoing low-intensity induction with venetoclax. Am J Hematol. 2021;96(3):E65-E68.
13. Willekens C, Bazinet A, Chraibi S, et al. Reduced venetoclax exposure to 7 days vs standard exposure with hypomethylating agents in newly diagnosed AML patients. Blood Cancer J. 2025;15(1):68.
14. Kantarjian HM, DiNardo CD, Kadia TM, et al. Acute myeloid leukemia management and research in 2025. CA Cancer J Clin. 2025;75(1):46-67.
15. Sastow D, Levavi H, Wagner N, Pratz K, Tremblay D. Ven the dose matters: venetoclax dosing in the frontline treatment of AML. Blood Rev. 2024;68:101238.