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JFZ conceptualized and designed the study. **JFZ** and **DTP** managed patient care, interpreted data, drafted, and revised the manuscript. **AI** and **JH** designed and performed the statistical analyses and analyzed the data. **KK, GJR, UB, JG-L, TL, BP, EC, MH, DR, AM, WC, LL, IG** contributed to patient accrual, data collection. All authors critically revised the manuscript and approved the final submitted version.

Data Sharing:
Data generated during this study are available from the corresponding author **JFZ** upon reasonable request

To the Editor:

Myelodysplasia-related acute myeloid leukemia (AML-MR) is a high-risk AML subtype defined by the World Health Organization (WHO 2022) and International Consensus Classification (ICC 2022) by a history of prior myeloid malignancy (AML-AHD), MDS-related cytogenetics (AML-MRc), and/or MDS-related mutations (AML-MRm).[1–3] Outcomes with conventional chemotherapy are historically poor in this subgroup.[4,5] While liposomal formulation of cytarabine and daunorubicin (CPX-351) demonstrated superior complete remission (CR) rates and overall survival (OS) compared to 7+3 in older patients with secondary AML (sAML) and AML-MRc, clinical trial and real-world outcomes in molecularly defined AML-MR remain suboptimal.[6–9]

Pomalidomide (POM) is a next-generation immunomodulatory drug (IMiD) that binds cereblon, inducing selective ubiquitination and degradation of Ikaros and Aiolos, thereby enhancing T-cell and natural killer-cell effector functions.[10] In a phase 1 trial, POM 4 mg for 21 days administered at early lymphocyte recovery (ELR; median day 21) following timed-sequential induction was well tolerated and demonstrated promising CR rates in sAML (71%) and unfavorable-risk cytogenetics (86%).[11] On this basis, we conducted a multicenter, open-label, randomized phase 2 trial evaluating CPX-351 with or without POM in newly diagnosed AML-MR (ClinicalTrials.gov: NCT04802161; National Cancer Institute [NCI] protocol: NCI-10434). The study protocol was approved by the NCI Central Institutional Review Board (IRB) and the IRB at each participating institution. The study was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent.

Prior to randomization, a 3+3 safety run-in (n=9) evaluated POM starting on day 21 across two dose levels (DL-1: 4 mg daily x 10 days, n=3; DL1: 4 mg daily x 14 days, n=6; DL2: 4 mg x 21 days, n=0). Of note, POM was never escalated to DL2 given concerns for prolonged marrow suppression. POM 4 mg daily for 14 days starting on day 21 (DL1) was selected as the recommended phase 2 dose after full safety assessment.[12]

In the randomized phase, patients were assigned 1:1 prior to induction to CPX-351 plus POM (CPX+POM) or CPX-351 alone (CPX), stratified by age (<60 vs. ≥60 years) and AML subtype. Enrollment occurred August 2022-May 2024 across nine U.S. institutions. Randomization occurred prior to induction, but because POM was initiated on day 21, the primary efficacy analysis utilized a modified intention-to-treat (mITT) population, excluding patients who did not meet eligibility to receive POM or received non-protocol anti-leukemia therapy prior to day 21.

Thirty-nine patients were randomized (**Figure 1**). Five patients (CPX+POM, n=4; CPX, n=1) were excluded from the mITT population: three patients assigned to CPX+POM experienced clinical instability or early death before day 21 (septic shock, n=1; intubation/hypoxic respiratory failure, n=1; GI bleed, n=1), and two patients (CPX+POM, n=1; CPX, n=1) received alternative anti-leukemia therapy before day 21. Thus, 34 patients were included in the mITT efficacy population (CPX+POM, n=16; CPX, n=18). One patient randomized to the CPX+POM arm received 3 days of POM before coming off study to pursue hospice; this patient was non-evaluable but retained in the mITT

analysis. Although enrollment was planned for up to 78 patients, an interim analysis (IA) was performed after 16 evaluable patients per arm, but accrual could continue while IA was being conducted.

Baseline demographics and clinical characteristics are summarized in **Table 1**. While general clinical demographics were broadly balanced, key genetic and post-remission treatment imbalances were present. The CPX arm carried a higher proportion of *TP53* mutations (33.3% vs. 12.5%) whereas *IDH2* mutations were more common in the CPX+POM arm (25.0% vs. 0%). Notably, *TP53*-mutant patients qualified for trial entry based on either AML-MRc, therapy-related AML, or antecedent disorder (AML-AHD).

The primary endpoint of CRc rate (CR+CRi+CRh) per ELN 2022 guidelines was numerically lower in the CPX+POM arm (56.3% [9/16]; 95% CI: 29.9–80.2%) compared with CPX alone (72.2% [13/18]; 95% CI: 46.5–90.3%; Fisher's *P=0.475*), meeting the pre-specified boundary for early termination due to futility (**Table 2**). Response rates across all AML-MR subsets (AML-MRm, AML-MRc, AML-AHD, t-AML) consistently favored the CPX arm (**Supplemental Figure 1**). Measurable residual disease (MRD) negativity among responders by central multiparameter flow cytometry (sensitivity 0.02%) was similar between arms (66.7% vs. 76.9%, *P=0.647*).

Safety and adverse events occurring after day 21 are summarized in the **Supplemental Figure 2**. Grade ≥3 febrile neutropenia (68.8% vs. 33.3%), sepsis/bacteremia (31.3% vs. 0%), dyspnea (18.8% vs. 5.6%), and hypoxia (37.5% vs. 11.1%) were markedly higher

with CPX+POM. CPX+POM also induced significantly higher rates of any-grade hyperbilirubinemia (37.5% vs 0%), elevated AST/ALT (37.5% vs 11.1%), and pleural effusions (31.2% vs 16.7%). Additionally, CPX+POM significantly prolonged hematologic recovery (median time to neutrophil recovery [≥0.5 x 10^9 /L] was 48 vs. 38 days; median time to platelet recovery [≥50 x 10^9 /L] was 48 vs. 38 days for CPX+POM versus CPX, respectively).

Two patients experienced clinically significant toxicities directly attributable to POM during or after day 21 (Grade 3 AST/ALT elevation: n=1; Grade 2 AST/ALT elevation with hyperbilirubinemia and subsequently developed fatal septic shock on day 38: n=1). An additional case of fatal septic shock occurred in the CPX+POM arm, but it occurred prior to POM administration (day 14) and was not attributed to POM.

Early 60-day mortality in the mITT population was 12.5% (2/16; progressive AML n=1, sepsis n=1) in the CPX+POM arm versus 0% (0/18) in the control arm. To report safety without day 21 exclusion bias, true Intent-to-Treat (ITT; N=39) 60-day mortality in the CPX+POM arm was 25.0% (5/20) versus 0% (0/19) with CPX alone. This includes early death in three of the four patients that were ineligible to receive POM and were due to septic shock (day 14), parainfluenza/respiratory failure (day 30), GI bleeding/fungal infection/AML (day 35).

All time-to-event analyses are exploratory due to small sample sizes and early trial termination (**Supplemental Figure 3**). With a median follow-up of 16.3 months

(CPX+POM) and 23.3 months (CPX), median event-free survival (EFS) was 6.00 vs. 8.56 months (log-rank *P=0.079*), and median OS was 18.64 months vs. not reached (*P=0.335*). Allogeneic hematopoietic stem cell transplantation (alloHSCT) also favored the CPX arm with 31.3% (5/16) vs. 66.7% (12/18) receiving alloHSCT in CPX+POM vs. CPX alone, heavily confounding survival endpoints.

These findings reveal no benefit of adding POM to CPX-351 induction in newly diagnosed AML-MR, and demonstrated a signal of increased toxicity, prolonged myelosuppression, and higher early mortality. The higher-than-expected response in the CPX-351 control arm (72% CRc) and low induction mortality compare favorably to historical benchmarks from the pivotal CPX-351 trial (47.7% CRc; 60-day mortality 13.7%).[6,7] This may reflect the inclusion of younger patients (47% <60 years) compared to the pivotal study and enrichment for molecularly defined AML-MRm, a subgroup recently shown in post-hoc analyses of pivotal trials and real-world registries to derive selective benefit from CPX-351[9,13]

Several factors may explain why POM failed to improve outcomes, including an attenuated 14-day dosing schedule, distinct immune recovery kinetics following CPX-351, or a lack of intrinsic antileukemic activity. Specifically, fixed-day administration on day 21 may have been suboptimal in timing, duration, or both. While biomarker-driven administration (e.g., targeting early lymphocyte recovery [ELR]) poses operational challenges during intensive induction, tailor-fitting immunomodulation to individual immune reconstitution kinetics may prove essential to the approach. Ongoing

translational correlative analyses will clarify the biological impact of POM on the post-CPX-351 immune environment.

Ultimately, these findings underscore the inherent challenges of integrating post-induction immunotherapy with delayed-recovery regimens and establish a benchmark for future randomized trial designs in AML-MR. Future combination with CPX-351 should prioritize non-myelosuppressive combinations or restrict sequential immune-directed therapies to post-recovery maintenance settings after complete hematologic recovery has been established. Modulating the immune response following lower-intensity, venetoclax-based regimens warrants parallel investigation.

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Tables

TABLE 1. Baseline demographic and clinical characteristics

	CPX+POM (n=16)	CPX (n=18)	All (N=34)
Age - yrs., median [range]	61 [49-74]	64 [34–74]	63 [34-74]
≥ 60 yr. - no. (%)	7 (43.8)	11 (61.1)	18 (52.9)
≥ 70 yr. - no. (%)	3 (18.8)	4 (22.2)	7 (20.6)
Female sex - no (%)	7 (43.8)	9 (50.0)	16 (47.1)
AML type - no. (%)			
De novo	9 (56.3)	10 (55.6)	19 (55.9)
Secondary (sAML)†	7 (43.8)	8 (44.4)	15 (44.1)
Therapy-related (tAML)	3 (18.8)	3 (16.7)	6 (17.6)
AML-MR§ Subtype - no. (%)			
AML-AHD	4 (25.0)	5 (27.8)	9 (26.5)
AML-MRc	9 (56.3)	13 (72.2)	22 (64.7)
AML-MRm	10 (62.5)	9 (50.0)	19 (55.9)
Disease Characteristics - median [range]			
Bone marrow blast count - %	53 [20–90]	48 [30–90]	50 [20–90]
WBC count* - K/μL	3 [0.3–54]	3 [0.7–82.3]	3 [0.3–82.3]
PLT count - K/μL	63 [8–288]	52 [7–317]	60 [7–317]
ELN 2022 risk category - no. (%)			
Favorable	0 (0.0)	1 (5.6)	1 (2.9)
Intermediate	1 (6.3)	0 (0.0)	1 (2.9)
Adverse	15 (93.8)	17 (94.4)	32 (94.1)
Complex Karyotype - no. (%)	6 (37.5)	5 (27.8)	11 (32.4)
MDS-related mutations - no. (%)			
RUNX1	4 (25.0)	6 (33.3)	10 (29.4)
ASXL1	5 (31.3)	4 (22.2)	9 (26.5)
SRSF2	3 (18.8)	3 (16.7)	6 (17.6)
EZH2	2 (12.5)	2 (11.1)	4 (11.8)
BCOR	2 (12.5)	1 (5.6)	3 (8.8)
STAG2	0 (0.0)	2 (11.1)	2 (5.9)
SF3B1	1 (6.3)	0 (0.0)	1 (2.9)
ZRSR2	1 (6.3)	0 (0.0)	1 (2.9)
U2AF1	0 (0.0)	0 (0.0)	0 (0.0)
Mutations - no. (%)			
TP53	2 (12.5)	6 (33.3)	8 (23.5)
IDH1	2 (12.5)	1 (5.6)	3 (8.9)
IDH2	4 (25%)	0 (0.0)	4 (11.8)
FLT3-ITD	0 (0.0)	2 (11.1)	2 (5.9)

TABLE 1. Baseline demographic and clinical characteristics.

Abbreviations: AML, acute myeloid leukemia; AML-MR, myelodysplasia-related AML; MRc, cytogenetically defined AML-MR; MRm, molecularly defined AML-MR; AHD, antecedent hematologic disorder; MDS, myelodysplastic syndrome; CPX, liposomal cytarabine and daunorubicin; WBC, white blood cell; PLT, platelet; ELN, European LeukemiaNet

† Secondary AML includes patients with AHD and/or tAML

§ AML-MR as defined by cytogenetic and molecular features using both World Health Organization (WHO) 2022 and International Consensus Criteria (ICC) 2022

* Baseline WBC count was defined as highest WBC value prior to start of induction

TABLE 2. Clinical outcomes		
	CPX+POM (n=16)	**CPX** (n=18)
Response - no. (%)		
CR	9 (56.3)	13 (72.2)
CRi	0	0
CRh	0	0
MLFS	0	1 (5.6)
NR	4 (25.0)	3 (16.7)
NE	3 (18.8)	1 (5.6)
CRc by Subtype - no. (%)		
AML-AHD	1/4 (25)	5/5 (100)
AML-MRc	5/9 (56)	9/13 (69)
AML-MRm	6/10 (60)	7/9 (78)
Time to recovery - median days [range]		
Platelets	48 [34-60]	38 [26-79]
Neutrophils	48 [38-54]	38 [26-58]
60-day mortality - no. (%)		
mITT	2/16 (12.5)	0/18 (0)
ITT	5/20 (25.0)	0/19 (0)
AlloHSCT - no. (%)	5 (31)	12 (67)

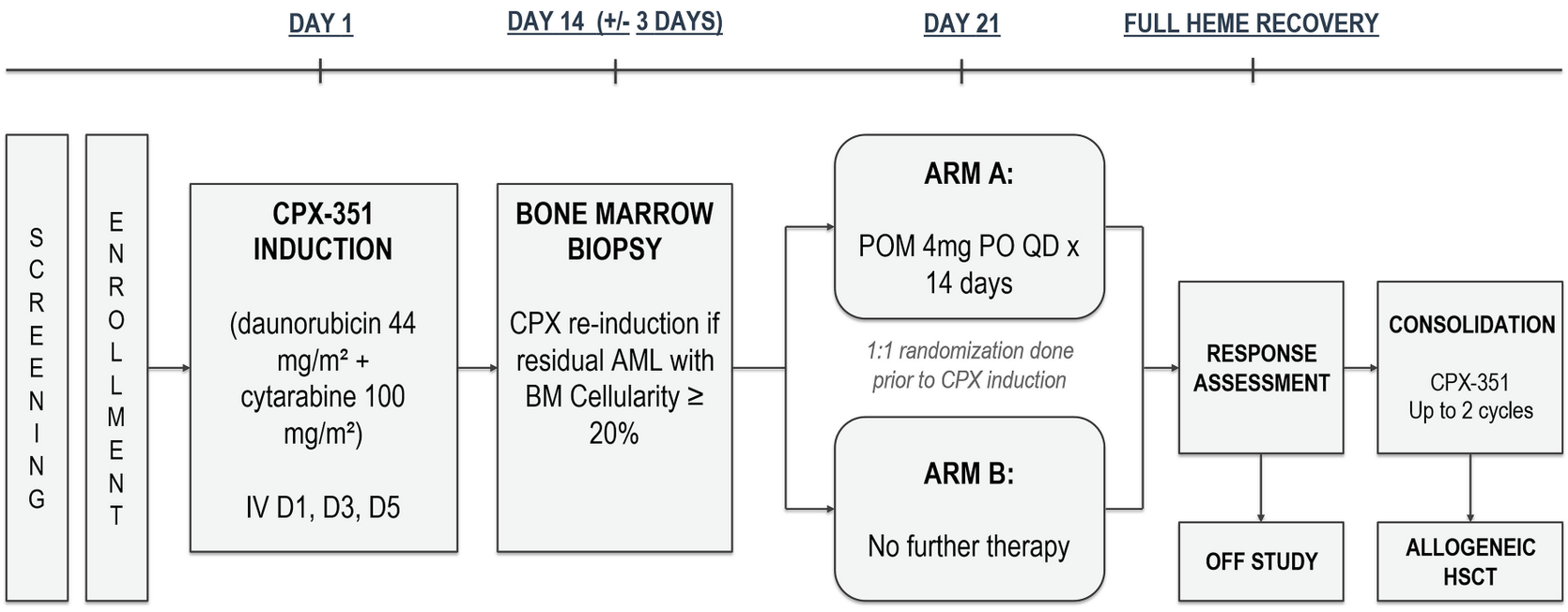
TABLE 2. Clinical outcomes.

Abbreviations: AlloHSCT, allogeneic hematopoietic stem cell transplantation; AML, acute myeloid leukemia; AML-MR, AML, myelodysplasia-related; MRc, cytogenetically defined AML-MR; MRm, molecularly defined AML-MR; AHD, antecedent hematologic disorder; CPX, liposomal cytarabine and daunorubicin; POM, pomalidomide.

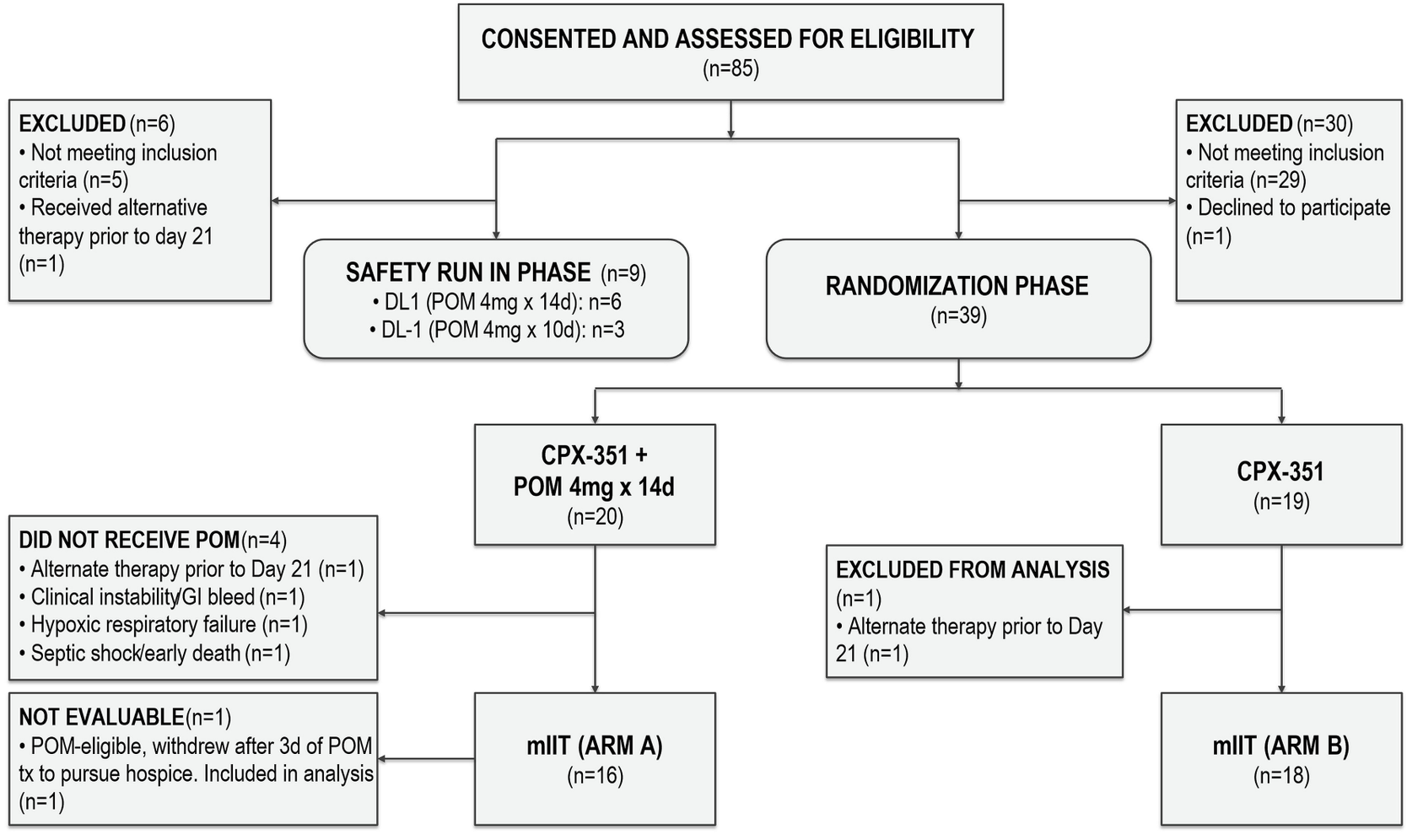
† Secondary AML includes patients with AHD and/or tAML

§ AML-MR as defined by cytogenetic and molecular features using both World Health Organization (WHO) 2022 and International Consensus Criteria (ICC) 2022

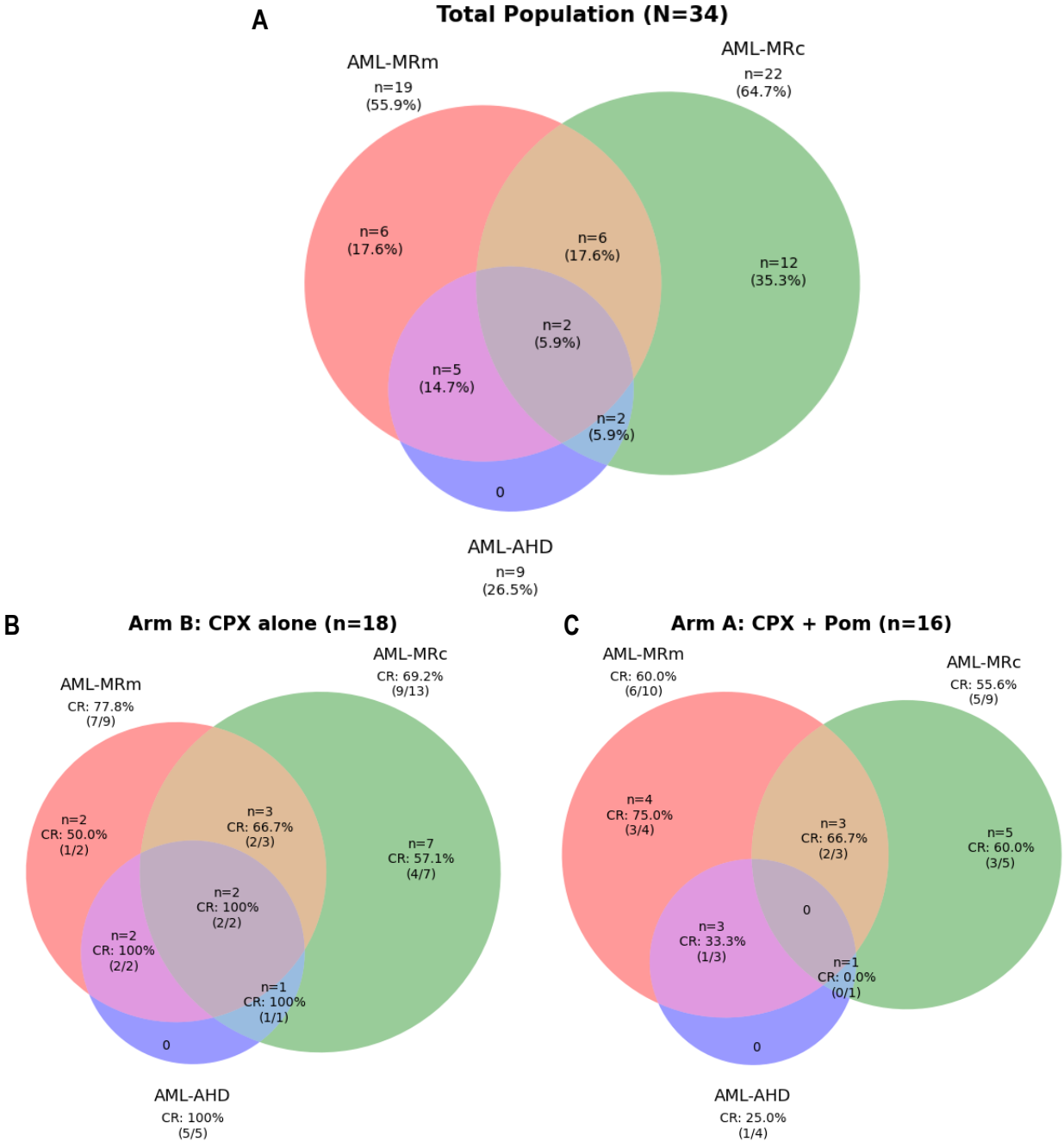
A. Study Schema for NCI-10434.



B. CONSORT Diagram for NCI-10434.

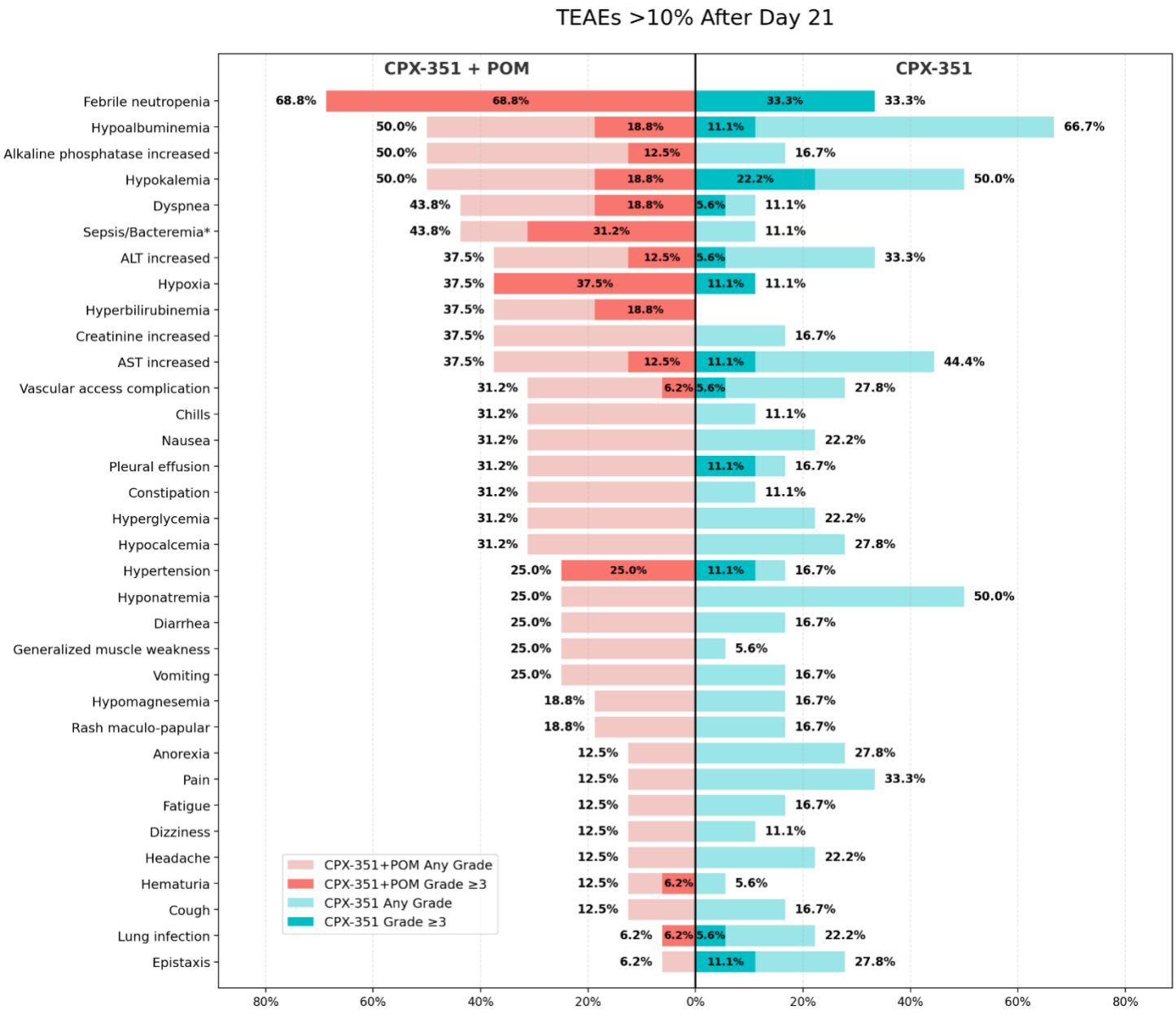


Supplementary Data

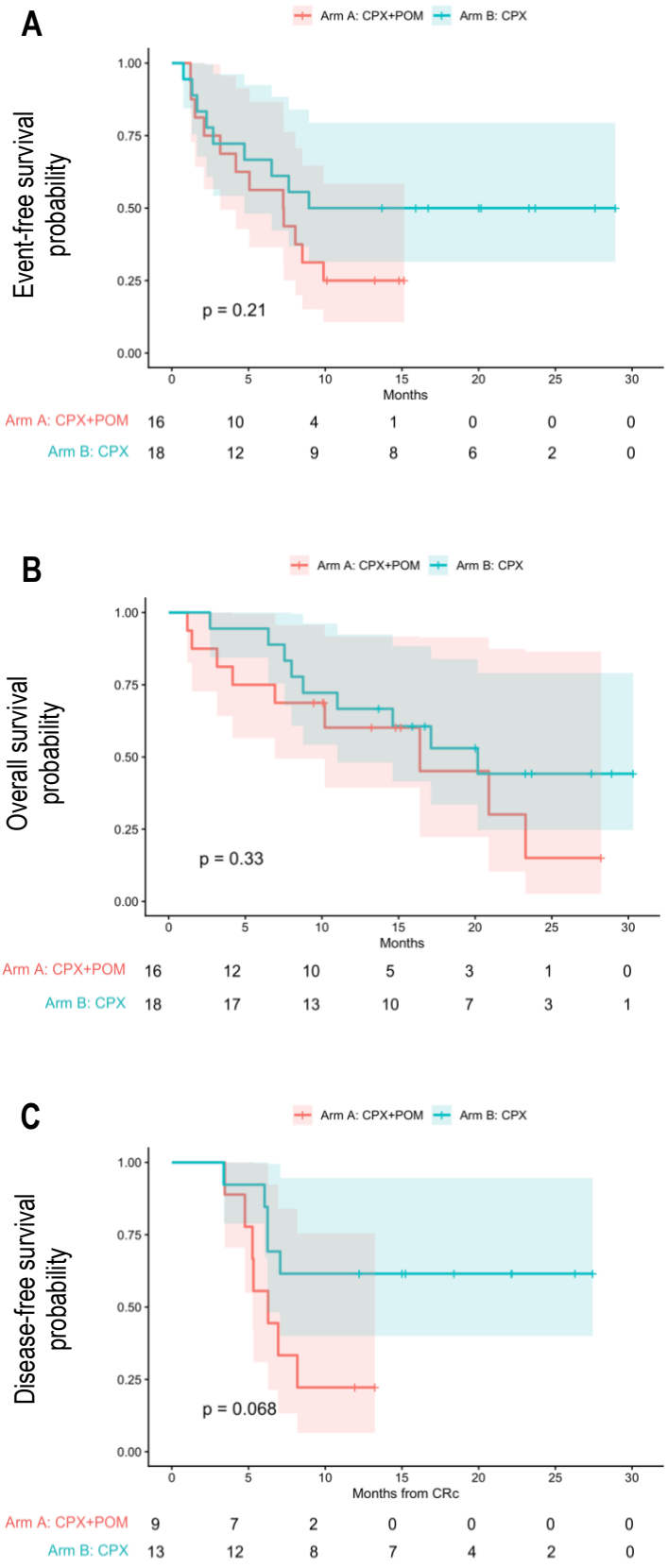


Supplemental Figure 1. Diagnostic subset overlap in acute myeloid leukemia with myelodysplasia-related changes. Area-proportional Venn diagrams depict patient distributions meeting criteria for myelodysplasia-related mutations (AML-MRm), cytogenetic abnormalities (AML-MRc), and antecedent hematologic disorder (AML-AHD) across **(A)** the total study population (n, % per subset), **(B)** CPX alone, and **(C)** CPX+POM, with treatment-specific complete response (CR) rates provided per subset for panels B and C.

Abbreviations: AHD, antecedent hematologic disorder; AML, acute myeloid leukemia; CPX, liposomal cytarabine and daunorubicin; CR, complete response; MRc, myelodysplasia-related cytogenetic abnormalities; MRm, myelodysplasia-related mutations; POM, pomalidomide; tAML, therapy-related acute myeloid leukemia.



Supplemental Figure 2. Non-hematologic Treatment Emergent Adverse Events (TEAE) Occurring in >10% of patients after Day 21.
Abbreviations: CPX, liposomal cytarabine and daunorubicin; POM, pomalidomide.
*Sepsis/Bacteremia represents a composite term combining Common Terminology Criteria for Adverse Events (CTCAE) preferred terms for bacteremia, septic shock, and sepsis to reflect total systemic infectious events.



Supplemental Figure 3. Survival outcomes in the modified intention-to-treat (mITT) population. **(A)** Event-free survival (EFS) in CPX+POM (median 6.00 months, 95% CI: 2.10–12.45) vs. CPX alone (median 8.56 months, 95% CI: 4.73–NR). **(B)** Overall survival (OS) in CPX+POM (median 18.64 months, 95% CI: 6.93–NR) vs. CPX alone (median NR, 95% CI: 8.77–NR). **(C)** Disease-free survival (DFS) among patients in composite complete remission (median 6.93 months, 95% CI: 5.32–NR in CPX+POM vs. NR, 95% CI: 6.24–NR in CPX alone). **(D)** Causes and study day of death by treatment arm (day calculated from Cycle 1 Day 1 induction [D+1]). Data cut-off: May 6, 2026.

Abbreviations: alloHSCT, allogeneic. hematopoietic stem cell transplantation; AML, acute myeloid leukemia; CI, confidence interval; CPX, liposomal cytarabine and daunorubicin; CRc, composite complete remission; NR, not reached; POM, pomalidomide.

D

Study day of death	Cause of death
CPX+POM (N=9)	
D+38	AML, Sepsis
D+47	AML
D+97	AML
D+128	AML
D+212	AML
D+311	Febrile neutropenia/Sepsis
D+500	AML
D+637	AML
D+710	AML
CPX alone (N=8)	
D+83	AML
D+199	AML
D+231	Failure to thrive post alloHSCT
D+245	AML, invasive fungal sinusitis
D+250	AML, Sepsis
D+268	AML
D+446	AML
D+522	AML