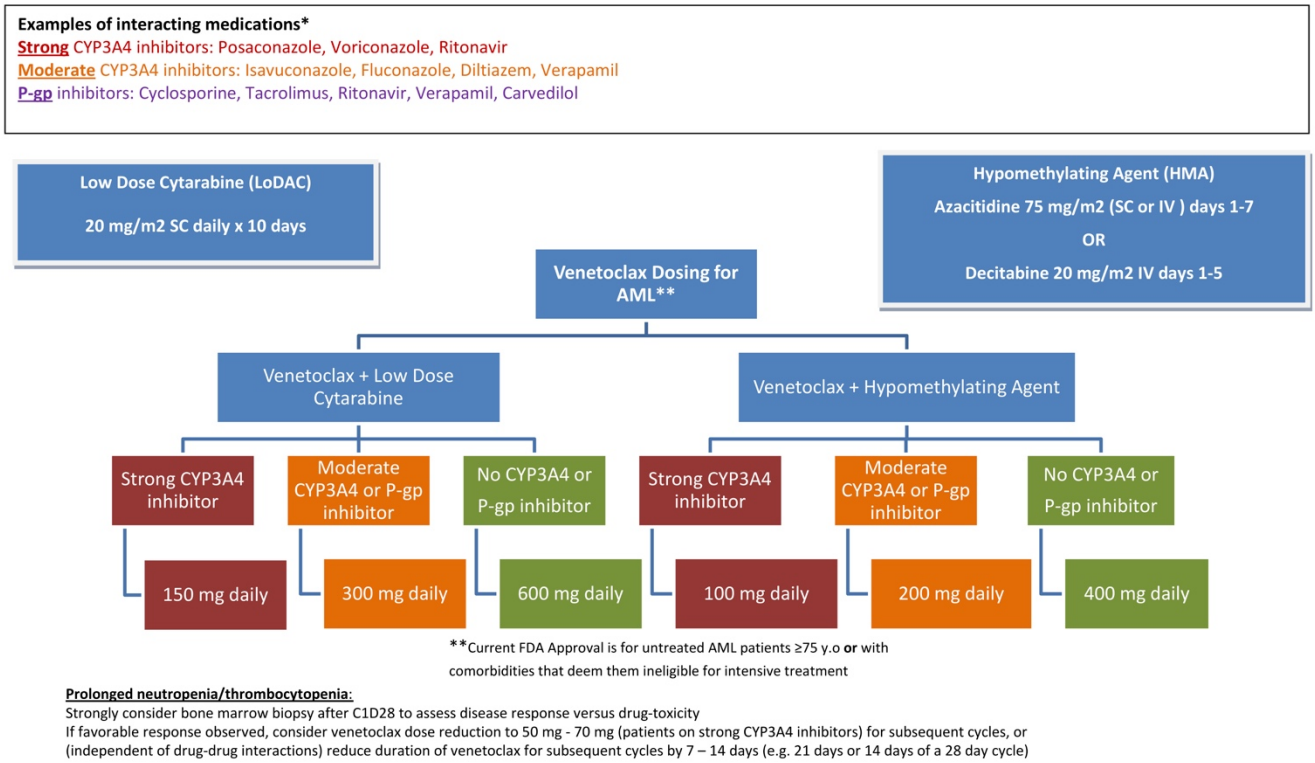
Supplement

Supplemental methods:

Venetoclax treatment protocol: Patients were treated following a standardized protocol, which was developed in collaboration between the Leukemia Service and the Pharmacy Service at MSKCC (see below). This protocol follows the practice guidelines as published by Brian Jonas and Daniel Pollyea (Jonas et al Leukemia 2019; doi: 10.1038/s41375-019-0612-8). Patients treated with venetoclax in combination with azacitidine (aza/ven) or decitabine (dec/ven) received 400 mg of venetoclax daily whereas patients treated with venetoclax in combination with LDAC (LDAC/ven) received 600 mg of venetoclax daily. If the absolute neutrophil count (ANC) of patients fell below 0.5 at any time during treatment, patients were started on antifungal prophylaxis with a strong CYP34 inhibitor such as voriconazole or posaconazole and the venetoclax dose was reduced to 100 mg daily for aza/ven or dec/ven and to 150 mg for LDAC/ven.



Though adjustment of venetoclax to 50 mg when used with LDAC and a strong CYP3A4 inhibitor is described in the VIALE-C trial (Wei et al Blood 2020; doi: 10.1182/blood.2020004856), current manufacturer recommendations are an adjustment to 100 mg daily with concomitant strong CYP3A4 inhibitors. A 75% dose reduction from the venetoclax target (e.g. 150 mg for 600 mg target and 100 mg for a 400 mg target) is kinetically reasonable with strong CYP3A4 inhibitors (venetoclax prescribing information; https://www.rxabbvie.com/pdf/venclexta.pdf). Our rationale is supported by pharmacokinetic data from Agarwal and colleagues, their findings noted that posaconazole (a strong CYP3A4 inhibitor) can be used safely after reducing the venetoclax dose by at least 75% (Agarwal et al Clin Ther. 2017; doi: 10.1016/j.clinthera.2017.01.003). Furthermore, the initial safety/efficacy report of 145 patients in the landmark phase 1b trial studied venetoclax doses as high as 1200 mg (DiNardo et al Blood 2019; doi: 10.1182/blood-2018-08-868752). The 1200 mg cohort experienced a *trend* toward higher frequency of hematologic and gastrointestinal adverse events, while the 400 mg and 800 mg were noted to be similarly tolerated. Our adjustment strategy would lend to exposure under 800 mg, additionally the authors do not believe that venetoclax exposure of 800 mg would cause excess toxicity.

Analysis of cytogenetic and molecular data at time of diagnosis and time of relapse: Cytogenetic data were taken from clinical reports produced by the MSK Clinical Cytogenetics Laboratory and included data from both karyotype and fluorescent in-situ hybridization (FISH). Molecular data were obtained from reports of clinical next-generation sequencing (NGS) performed using RainDance Technologies, ThunderStorm and ThunderBolts Cancer Panel, as well as Memorial Sloan Kettering Integrated Mutation Profiling of Actionable Cancer Targets (MSK-IMPACT). The test characteristics of MSK-IMPACT have previously been described in *Nature Medicine* (Zehir et al, Nature 2017; doi: 10.1038/nm.4333). In the *Nature Medicine* paper on the assay, it was shown to have much greater sensitivity when compared to two commercially-available amplicon assays. The IMPACT assay sequences to a mean read depth of greater than 700x and in our analysis of clinical data, molecular features present at a VAF of 0.05 or greater were considered to be present in a given sample.For patients sequenced using a platform that did not contain all of the genes of interest, the missing genes were considered non-informative instead of non-mutated. NGS and cytogenetic evaluations were performed on bone marrow aspirates (BMA) or peripheral blood; if both were available, data from BMA were used preferentially. A variant allele frequency of 0.5% was used as a cutoff for consideration of variant calls, which were annotated using MSKCC's clinical pipeline. The data was reviewed by both the clinical bioinformatics and pathology teams prior to the mutational calls being confirmed. For the relapse analysis, only patients who had had IMPACT testing performed both prior to treatment initiation and at the time of relapse were included.

Immunophenotypic analysis by flow cytometry: In brief, up to 1.5 million cells from freshly drawn bone marrow aspirate were stained with 3 to 6 ten-color panels, washed, and acquired on a Canto-10 cytometer (BD Biosciences, San Jose, CA). The results were analyzed using custom Woodlist software (generous gift of B. L. Wood, University of Washington) and interpreted by AC and MR.
Immunophenotypic characteristics of responding patients (Resp) were compared with non-responding patients (NonResp). Additionally, immunophenotypic characteristics at time of treatment start (Dx) were compared with immunophenotypic characteristics at time of relapse (RL).
Immunophenotypic characteristics included monocytes, immature monocytes, blasts and CD38 dim blasts. Monocytes were identified by moderate side scatter, variable expression of CD14, CD45 and HLA-DR, and relatively bright expression of CD64. Immature monocytes were identified as a monocyte population with dim CD11b, dim CD14, and bright HLA-DR. Blasts were identified by CD34 or CD117 expression. CD38 dim blasts were identified as a blast population expressing dim CD38.

Statistical analysis: Categorical patient characteristics were summarized by frequency. Univariate logistic regression models were used to estimate the odds ratios (ORs) with 95% confidence intervals (CIs), for associations between overall response and patient and disease characteristics. Univariate Cox proportional hazards models were used to estimate Hazard ratios (HRs) with 95% CIs, for association between mortality and patient and disease characteristics. P-values for the significance of these associations were calculated by a Wald-test. Associations between molecular predictors and overall response were tested by Fisher's exact test and odds ratios with 95% CIs were estimated by fitting logistic regression. Associations between mortality and molecular predictors were assessed by log-rank test and HRs with 95% CIs were estimated using univariate Cox proportional hazards models. Survival plots and estimates of median survivals with 95% CIs were generated for the main findings using the Kaplan-Meier method, and statistical significance was assessed by log-rank test. Lastly, for each group of patients, the associations with survival of clinical and molecular predictors that had a significant association with ORR or survival in our univariate analysis were jointly evaluated by using multivariable Cox proportional hazards model. Statistical significance was defined using a two-sided significance level at 0.05. All statistical analyses were using R version 3.6.1.

Supplemental Tables

Supplemental Table 1: Frequency of mutations

Mutation	N (%)
DNMT3A	27 (33%)
SRSF2	16 (22%)
RUNX1	16 (20%)
IDH2	15 (18%)
NPM1	13 (16%)
TET2	13 (16%)
STAG2	10 (14%)
TP53	11 (13%)
IDH1	9 (11%)
SF3B1	9 (11%)
CEBPA	8 (10%)
NRAS	10 (12%)
FLT3	9 (11%)
GATA2	7 (9%)
ASXL1	6 (7%)
BCOR	5 (7%)
PTPN11	6 (8%)
BCORL1	4 (5%)
CSF3R	4 (5%)
EZH2	4 (5%)
JAK2	4 (5%)
KRAS	4 (5%)
ETV6	3 (4%)
PHF6	3 (4%)
RAD21	3 (4%)
SETBP1	3 (4%)
U2AF1	3 (4%)
WT1	3 (4%)

Supplemental Table 2: Univariable analysis of clinical predictors for response and survival for RR-AML patients treated with venetoclax combination therapy

Variable	Response (CR/CRi/MLFS)		Death	
	OR (95%CI)	**P-value**	**HR (95%CI)**	**P-value**
Age (ref: < 65 years)				
> 75 years	1.56 (0.45,5.35)	0.20	0.81 (0.4,1.66)	0.57
65-75 years	1.96 (0.69,5.57)	0.48	0.84 (0.46,1.54)	0.57
AML type (ref: de novo)				
Secondary	0.53 (0.21,1.35)	0.18	1.18 (0.69,2.01)	0.55
Prior HMA therapy (ref: no)				
Yes	0.59 (0.24,1.49)	0.26	1.54 (0.88,2.7)	0.13
Cycles of prior HMA therapy (ref: naïve)				
1-4 cycles	0.62 (0.2,1.95)	0.41	1.86 (0.97,3.57)	0.06
>4 cycles	0.64 (0.21,1.91)	0.42	1.29 (0.66,2.51)	0.45
Prior alloSCT (ref: no)	0.76 (0.22,2.64)	0.66	**2.0 (1.06,3.96)**	**0.03**
Number of prior lines of salvage therapy excluding induction/re-induction/consolidation therapy (ref: <3)	**0 (0,Inf)**	**0.04**[1]	**3.12 (1.45,6.7)**	**0.004**
≥ 3				
Venetoclax combination (ref: LDAC/ven)				
Aza/ven	**5.43 (1.55,19.00)**	**0.008**	**0.36 (0.19,0.7)**	**0.003**
Dec/ven	1.92 (0.44,8.31)	0.38	0.59 (0.3,1.17)	0.13
Best response with combination venetoclax therapy (time dependent)				
CR/CRi/MLFS achieved (ref: not achieved)	---		**0.1 (0.04,0.3)**	**<0.001**
CR/CRi MRD- achieved (ref: not achieved)	---		**0.09 (0.01,0.69)**	**0.02**
alloSCT received (ref: not received)	---		**0.17 (0.04,0.74)**	**0.02**

P< 0.05 marked bold

[1] P-value from score test.

Supplemental Table 3: Univariable analysis of molecular predictors for response and survival for RR-AML patients treated with venetoclax combination therapy

Variable	Overall response (CR/CRi/MLFS)		Complete remission (CR/CRi)		Death	
	OR (95%CI)	P value	OR (95%CI)	P value	HR (95%CI)	P value
DNMT3A	2.34 (0.89,6.19)	0.13	2.65 (0.94;7.48)	0.1	0.74 (0.4,1.36)	0.33
SRSF2	1.88 (0.6,5.85)	0.37	2.3 (0.7;7.6)	0.2	0.62 (0.29,1.34)	0.22
RUNX1	0.43 (0.11,1.67)	0.25	0.67 (0.17;2.62)	0.75	1.19 (0.62,2.28)	0.6
IDH2	1.57 (0.49,4.99)	0.54	2.52 (0.77;8.29)	0.18	0.79 (0.37,1.69)	0.54
NPM1	**4.53 (1.31,15.66)**	**0.02**	3.37 (0.98;11.62)	0.07	0.68 (0.29,1.59)	0.37
TET2	1.43 (0.42,4.88)	0.75	0.92 (0.23;3.73)	0.92	0.97 (0.47,1.99)	0.93
STAG2	2.37 (0.61,9.14)	0.28	2.38 (0.59;9.63)	0.24	**0.32 (0.1,1.02)**	**0.04**
TP53	0.18 (0.02,1.52)	0.16	0 (0;Inf)	0.06	**2.24 (1.07,4.69)**	**0.03**
IDH1	**5.3 (1.21,23.24)**	**0.03**	**4.83 (1.15;20.24)**	**0.04**	0.31 (0.07,1.26)	0.08
SF3B1*	0 (0,Inf)	0.05	0 (0;Inf)	0.1	**2.5 (1.1,5.65)**	**0.02**
CEBPA	2.36 (0.54,10.31)	0.26	2.01 (0.44;9.29)	0.40	0.52 (0.19,1.44)	0.2
NRAS+KRAS	0.6 (0.15,2.39)	0.54	0.52 (0.1;2.55)	0.50	**2.1 (1.04,4.22)**	**0.03**
FLT3	1.85 (0.45,7.57)	0.45	1.65 (0.37;7.3)	0.68	1.01 (0.4,2.55)	0.99
GATA2	1.64 (0.34,8)	0.68	1.27 (0.23;7.22)	1	0.68 (0.24,1.91)	0.47
ASXL1	0.41 (0.05,3.68)	0.66	0.6 (0.07;5.46)	1	1.35 (0.53,3.41)	0.53
BCOR	1.42 (0.22,9.14)	0.66	0.76 (0.08;7.32)	1	1.33 (0.52,3.4)	0.55
PTPN11*	0 (0,Inf)	0.17	0 (0;Inf)	0.33	1.55 (0.61,3.97)	0.35
BCORL1*	0 (0,Inf)	0.3	0 (0,Inf)	0.56	1.36 (0.42,4.42)	0.61
CSF3R*	0 (0,Inf)	0.3	0 (0,Inf)	0.56	2.38 (0.73,7.82)	0.14
EZH2*	0 (0,Inf)	0.3	0 (0,Inf)	0.56	**4.13 (1.43,11.96)**	**0.004**
JAK2*	0 (0,Inf)	0.3	0 (0,Inf)	0.56	1.74 (0.54,5.64)	0.35
ETV6	1.08 (0.09,12.48)	1	1.08 (0.09,12.48)	1	0.31 (0.04,2.22)	0.21
PHF6	1.08 (0.09,12.48)	1	1.08 (0.09,12.48)	1	0.52 (0.07,3.81)	0.52
U2AF1	1.04 (0.09,12.11)	1	1.04 (0.09,12.11)	1	0.51 (0.07,3.74)	0.5
Adverse cytogenetics	**0.32 (0.11,0.9)**	**0.03**	**0.2 (0.05;0.76)**	**0.02**	**1.95 (1.13,3.35)**	**0.01**
Complex/ monosomal karyotype	0.31 (0.1,1.03)	0.05	**0.1 (0.01;0.76)**	**0.009**	**1.98 (1.13,3.5)**	**0.02**
del17	0.72 (0.07,7.24)	0.78	0 (0;Inf)	0.57	1.47 (0.46,4.76)	0.51
TP53+del17	0.16 (0.02,1.34)	0.09	**0 (0;Inf)**	**0.03**	**2.42 (1.19,4.95)**	**0.01**

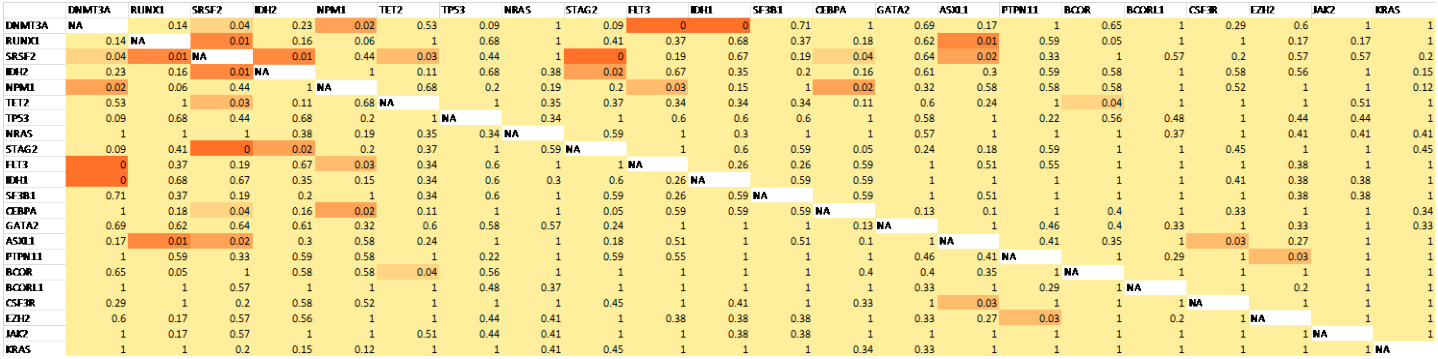
P< 0.05 marked bold

* mutations are present only in responding or non-responding patients

** mutations are present only in surviving or non-surviving patients

Supplemental Table 4: Co-occurrence of gene mutations

Each pair of gene mutations was tested for the presence of dependency using Fisher's exact test. Pair of genes with p-value < 0.05 (indicated in orange/red) are determined to have significant dependency in occurrence (i.e. co-occurrence or mutual-exclusivity). Pair of genes with p-value >= 0.05 (indicated in yellow) are determined to have no significant dependency in occurrence.



	DNMT3A	RUNX1	SRSF2	IDH2	NPM1	TET2	TP53	NRAS	STAG2	FLT3	IDH1	SF3B1	CEBPA	GATA2	ASXL1	PTPN11	BCOR	BCORL1	CSF3R	EZH2	JAK2	KRAS
DNMT3A	NA	0.14	0.04	0.23	0.02	0.53	0.09	1	0.09	0	0	0.71	1	0.69	0.17	1	0.65	1	0.29	0.6	1	1
RUNX1	0.14	NA	0.01	0.16	0.06	1	0.68	1	0.41	0.37	0.68	0.37	0.18	0.62	0.01	0.59	0.05	1	1	0.17	0.17	1
SRSF2	0.04	0.01	NA	0.01	0.44	0.03	0.44	1	0	0.19	0.67	0.19	0.04	0.64	0.02	0.33	1	0.57	0.2	0.57	0.57	0.2
IDH2	0.23	0.16	0.01	NA	1	0.11	0.68	0.38	0.02	0.67	0.35	0.2	0.16	0.61	0.3	0.59	0.58	1	0.58	0.56	1	0.15
NPM1	0.02	0.06	0.44	1	NA	0.68	0.2	0.19	0.2	0.03	0.15	1	0.02	0.32	0.58	0.58	0.58	1	0.52	1	1	0.12
TET2	0.53	1	0.03	0.11	0.68	NA	1	0.35	0.37	0.34	0.34	0.34	0.11	0.6	0.24	1	0.04	1	1	1	0.51	1
TP53	0.09	0.68	0.44	0.68	0.2	1	NA	0.34	1	0.6	0.6	0.6	1	0.58	1	0.22	0.56	0.48	1	0.44	0.44	1
NRAS	1	1	1	0.38	0.19	0.35	0.34	NA	0.59	1	0.3	1	1	0.57	1	1	1	0.37	1	0.41	0.41	0.41
STAG2	0.09	0.41	0	0.02	0.2	0.37	1	0.59	NA	1	0.6	0.59	0.05	0.24	0.18	0.59	1	1	0.45	1	1	0.45
FLT3	0	0.37	0.19	0.67	0.03	0.34	0.6	1	1	NA	0.26	0.26	0.59	1	0.51	0.55	1	1	1	0.38	1	1
IDH1	0	0.68	0.67	0.35	0.15	0.34	0.6	0.3	0.6	0.26	NA	0.59	0.59	1	1	1	1	1	0.41	0.38	0.38	1
SF3B1	0.71	0.37	0.19	0.2	1	0.34	0.6	1	0.59	0.26	0.59	NA	0.59	1	0.51	1	1	1	1	0.38	0.38	1
CEBPA	1	0.18	0.04	0.16	0.02	0.11	1	1	0.05	0.59	0.59	0.59	NA	0.13	0.1	1	0.4	1	0.33	1	1	0.34
GATA2	0.69	0.62	0.64	0.61	0.32	0.6	0.58	0.57	0.24	1	1	1	0.13	NA	1	0.46	0.4	0.33	1	0.33	1	0.33
ASXL1	0.17	0.01	0.02	0.3	0.58	0.24	1	1	0.18	0.51	1	0.51	0.1	1	NA	0.41	0.35	1	0.03	0.27	1	1
PTPN11	1	0.59	0.33	0.59	0.58	1	0.22	1	0.59	0.55	1	1	1	0.46	0.41	NA	1	0.29	1	0.03	1	1
BCOR	0.65	0.05	1	0.58	0.58	0.04	0.56	1	1	1	1	1	0.4	0.4	0.35	1	NA	1	1	1	1	1
BCORL1	1	1	0.57	1	1	1	0.48	0.37	1	1	1	1	1	0.33	1	0.29	1	NA	1	0.2	1	1
CSF3R	0.29	1	0.2	0.58	0.52	1	1	1	0.45	1	0.41	1	0.33	1	0.03	1	1	1	NA	1	1	1
EZH2	0.6	0.17	0.57	0.56	1	1	0.44	0.41	1	0.38	0.38	0.38	1	0.33	0.27	0.03	1	0.2	1	NA	1	1
JAK2	1	0.17	0.57	1	1	0.51	0.44	0.41	1	1	0.38	0.38	1	1	1	1	1	1	1	1	NA	1
KRAS	1	1	0.2	0.15	0.12	1	1	0.41	0.45	1	1	1	0.34	0.33	1	1	1	1	1	1	1	NA

Supplemental Table 5: Multivariable model of mutations with favorable impact on response

Mutation	OR	95% CI	P-value
IDH1	3.94	0.82-18.85	0.08618344
NPM1	3.95	1.01-15.39	0.04809235
STAG2	2.76	0.61-12.53	0.18834592
IDH2	1.22	0.32-4.67	0.77178274

Supplemental Table 6: Clinical outcomes based on 2017 ELN molecular risk status

Response based on 2017 ELN risk	favorable	intermediate	adverse	P value
Response				
- ORR = CR/CRi/MLFS	8/12 (67%)	8/21 (38%)	11/53 (21%)	0.006
- CR/CRi	6/12 (50%)	7/21 (33%)	8/53 (15%)	0.02
- MRD- CR/CRi	5/12 (42%)	3/21 (14%)	3/53 (6%)	0.005
- Partial remission	1/12 (8%)	2/21 (10%)	4/53 (8%)	1
- Persistent disease	3/12 (25%)	11/21 (52%)	38/53 (71%)	0.008
Overall survival in months (Median, 95% CI)	15.02 (8.09,NR)	8.25 (3.45,NR)	5.62 (3.88,7.17)	0.1*

* comparing median OS of favorable/intermediate ELN risk with adverse ELN risk:
15.02 months (95% CI: 6.87-NR) vs. 5.62 (3.88,7.17), p=0.034

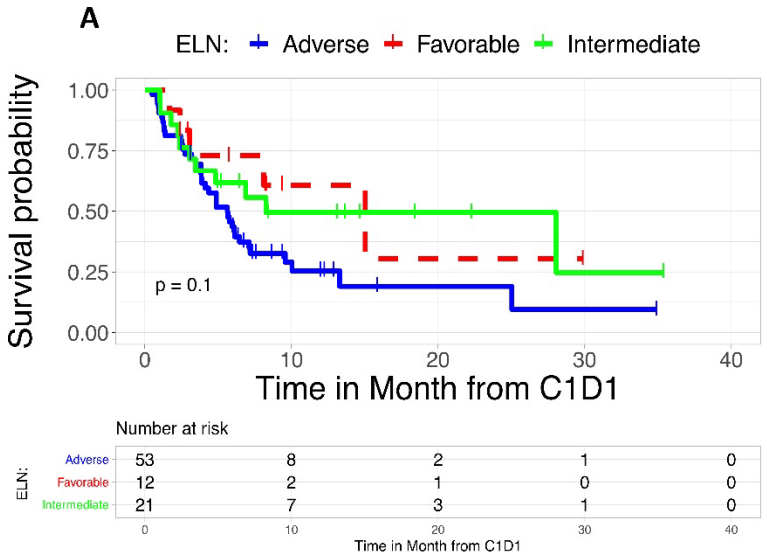
Supplemental Table 7: Multivariable analysis of clinical and genetic predictors of OS including for *DNMT3A* mutation status, prior HMA treatment, age and all-SCT after venetoclax therapy

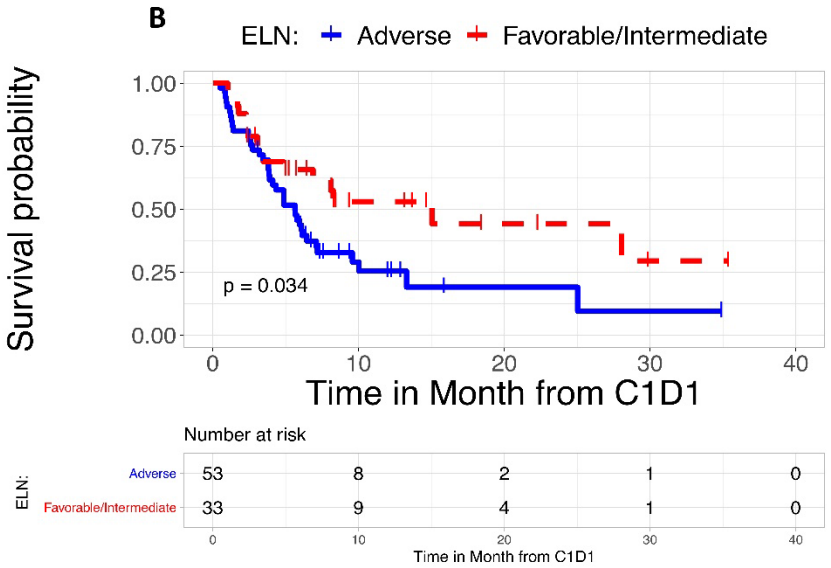
Characteristics	HR	95% CI	P-value
DNMT3A	1.13	0.4-3.16	0.82174885
Prior HMA	1.42	0.59-3.44	0.43033688
Age	1.21	0.72-2.03	0.47171481
allo-SCT after venetoclax therapy	0.25	0.05-1.14	0.07312844
allo-SCT prior to venetoclax therapy	1.2	0.44-3.27	0.71529671
Number of prior lines of therapy >=3	3.29	0.64-16.82	0.15215497
Aza/ ven (vs. LDAC/ven)	0.37	0.15-0.89	0.02552188
Dec/ven (vs. LDAC/ven)	0.39	0.14-1.1	0.07451515
Adverse cytogenetics	1.85	0.74-4.65	0.19117232
NRAS / KRAS	4.67	1.52-14.38	0.00718829
TP53	2.54	0.8-8	0.11269257
SF3B1	3.14	0.89-11.06	0.07519218
EZH2	2.69	0.81-8.98	0.10704014
STAG2	0.42	0.08-2.12	0.29422045
IDH1/2 / NPM1	0.69	0.27-1.75	0.43358707

Supplemental Table 8: Cytogenetic Changes at Relapse

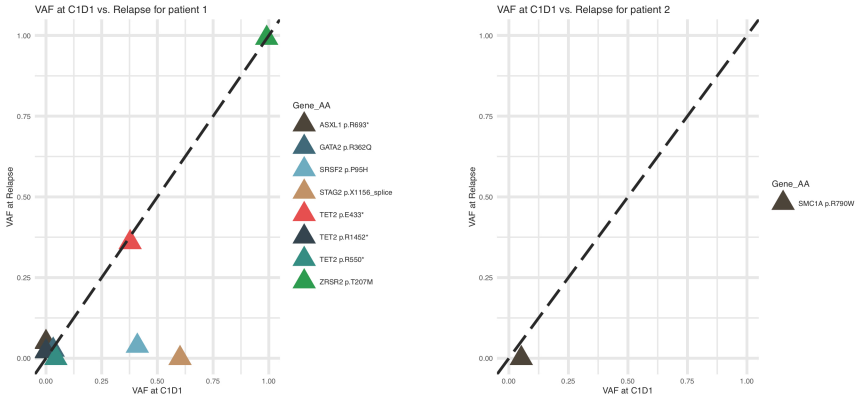
ID	Molecular Data Timepoint	Date of Marrow for Molecular Data	Karyotype	Change in Cytogenetics
RR-1	Diagnosis	2/7/17	46,XY[20]	
RR-1	Relapse	10/2/17	no CG submitted on 10/2/2017; 8/6/2018: +21[19/20]; 3/29/17: normal CG	Unknown
RR-2	Diagnosis	2/27/17	CG not performed. DX 9/6/2016, inv(3q)[30] FISH Pos 46%, normal MLL	
RR-2	Relapse	1/23/20	no samples submitted on 1/23/2020; however, 12/17/2019: 46,XY,inv(3)(q21q26.2)[14]/46,XY[6]; also 12/5/2019 inv(3q) by FISH	suggestive of persistent inv(3)
RR-5	Diagnosis	10/26/17	46,XY[20] DX 7/6/2016, normal K/F	
RR-5	Relapse	11/8/18	46,XY,add(11)(p15)[9]/46,XY[12]	add(11p15)
RR-9	Diagnosis	1/11/17	46,XY[20]	
RR-9	Relapse	1/15/18	not performed	Unknown
RR-10	Diagnosis	11/12/18	46,XX[20]	
RR-10	Relapse	12/31/19	46,XX,+1,der(1;12)(q10;10)[3]//46,XY[4]	+1,der(1;12)(q10 ;10)
RR-11	Diagnosis	11/29/18	no CG done; DX OH 9/10/2018, t(1;6)(q31;q15)[20]	
RR-11	Relapse	7/29/19	46,XY,t(1;6)(q32;q21)[7]/46,XY[13]	No change
RR-15	Diagnosis	1/2/19	46,XY,del(20)(q12q13.3)[9]/46,XY[11]	
RR-15	Relapse	11/1/19	46,XY,i(17)(q10),del(20)(q11.2q13.3)[20]	i(17)(q10)
RR-19	Diagnosis	4/4/19	46,XX,del(7)(q22q32)[8]/46,XX,t(4;12)(q12;p11.2)[4]/46,XX[8]	
RR-19	Relapse	1/16/20	46,XX,t(4;12)(q12;p11.2)[15]/46,idem,add(8)(q24)[2]//46,XY[3]	+8, normalization of del(7)
RR-26	Diagnosis	1/11/19	46,XX,t(3;17)(p25;q12)[8]/46,XX[9]//46,XY[3]	
RR-26	Relapse	7/8/19	46,XX,t(3;17)(p25;q12),add(4)(q35)[13]/46,XX[7]	add(4q35)
RR-45	Diagnosis	7/26/19	failed cytogentics. DX OH, 2/17/2016, 46,XX,+1,der(1;21)(q10;q10)[15]/46,XX[5]	
RR-45	Relapse	1/13/20	51,XX,+der(1;21)(q10;q10),+4,+6,add(6)(q22),del(6)(q23q27),+9,+22[7]// 46,XY[13]	suggestive of increasing complexity

Supplemental Figure 1: Overall Survival based on 2017 ELN molecualr risk
A: KM survival blots stratified by ELN 2017 molecular risk favorable vs. intermediate vs. adverse
B: KM survival blots stratified by ELN 2017 molecular risk favorable/intermediate vs. adverse

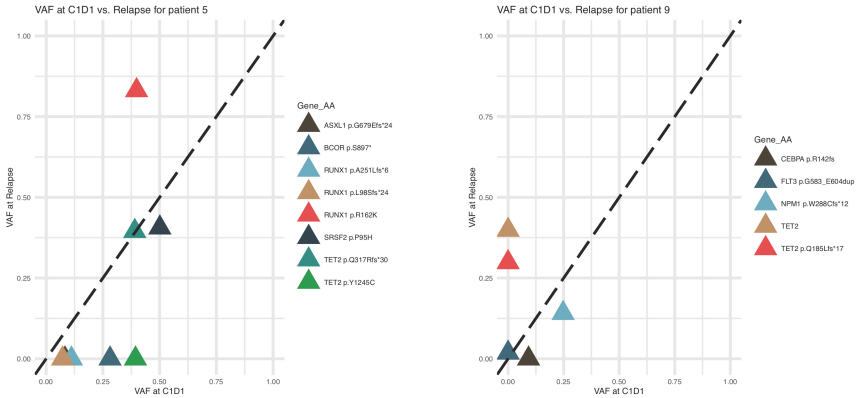




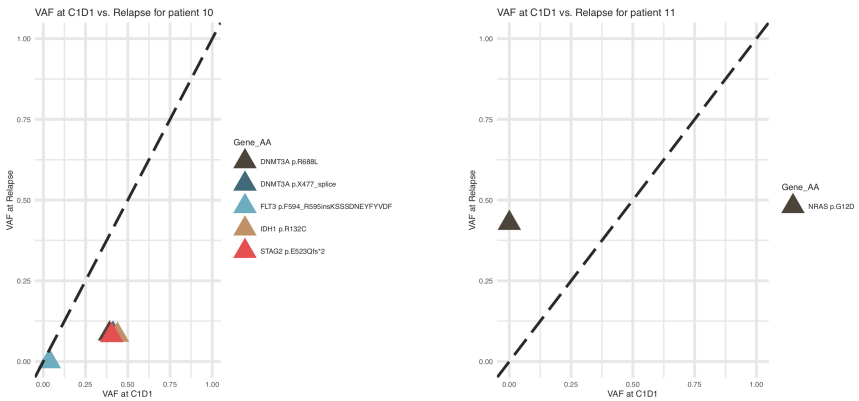
Supplemental Figure 2: Molecular changes with variant allele frequency (VAF) at time of relapse
These panels show the alterations in clonal architecture from the time of therapy initiation to relapse on a per-patient basis. The dashed line represents y = x and signifies the point at which any mutation would have an equal VAF in both the initial (cycle 1, day 1 = C1D1) and relapsed sample. Mutations towards the x-axis had a higher VAF prior to starting therapy, and mutations towards the y-axis had a higher VAF at the time of relapse. Mutations that fall along each axis were seen exclusively at that timepoint.


VAF at C1D1 vs. Relapse for patient 1
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
ASXL1 p.R693*
GATA2 p.R362Q
SRSF2 p.P95H
STAG2 p.X1156_splice
TET2 p.E433*
TET2 p.R1452*
TET2 p.R550*
ZRSR2 p.T207M

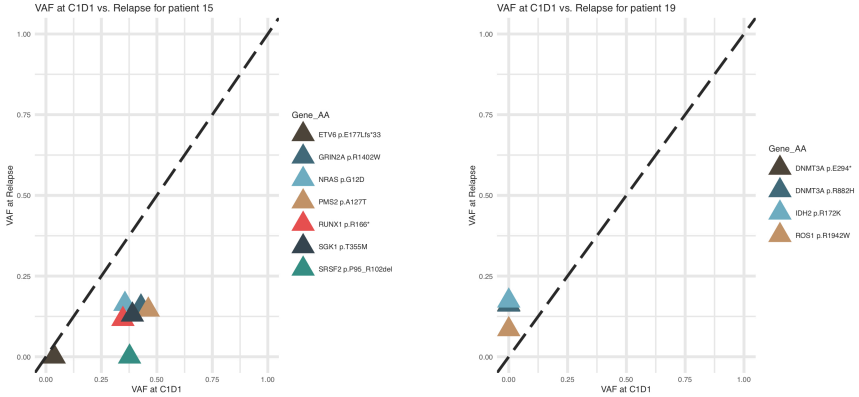
VAF at C1D1 vs. Relapse for patient 2
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
SMC1A p.R790W


VAF at C1D1 vs. Relapse for patient 5
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
ASXL1 p.G679Efs*24
BCOR p.S897*
RUNX1 p.A251Lfs*6
RUNX1 p.L985fs*24
RUNX1 p.R162K
SRSF2 p.P95H
TET2 p.Q317Rfs*30
TET2 p.Y1245C

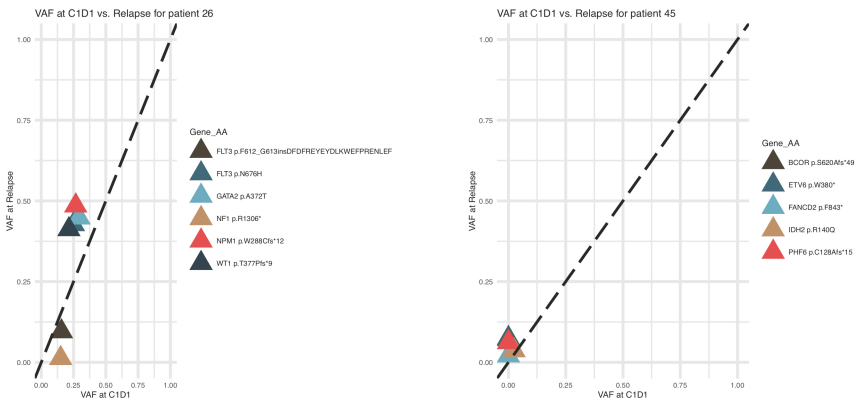
VAF at C1D1 vs. Relapse for patient 9
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
CEBPA p.R142fs
FLT3 p.G583_E604dup
NPM1 p.W288Cfs*12
TET2
TET2 p.Q185Lfs*17


VAF at C1D1 vs. Relapse for patient 10
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
DNMT3A p.R688L
DNMT3A p.X477_splice
FLT3 p.F594_R595insKSSSDNEYFYVDF
IDH1 p.R132C
STAG2 p.E523Qfs*2

VAF at C1D1 vs. Relapse for patient 11
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
NRAS p.G12D


VAF at C1D1 vs. Relapse for patient 15
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
ETV6 p.E177Lfs*33
GRIN2A p.R1402W
NRAS p.G12D
PMS2 p.A127T
RUNX1 p.R166*
SGK1 p.T355M
SRSF2 p.P95_R102del

VAF at C1D1 vs. Relapse for patient 19
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
DNMT3A p.E294*
DNMT3A p.R882H
IDH2 p.R172K
ROS1 p.R1942W


VAF at C1D1 vs. Relapse for patient 26
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
FLT3 p.F612_G613insDFDFREYEYDLKWEFPRENLEF
FLT3 p.N676H
GATA2 p.A372T
NF1 p.R1306*
NPM1 p.W288Cfs*12
WT1 p.T377Pfs*9

VAF at C1D1 vs. Relapse for patient 45
VAF at Relapse
VAF at C1D1
0.00
0.25
0.50
0.75
1.00
Gene_AA
BCOR p.S620Afs*49
ETV6 p.W380*
FANCD2 p.F843*
IDH2 p.R140Q
PHF6 p.C128Afs*15