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Quantitative CD19 expression in pediatric *NUP98*-rearranged myeloid neoplasms: enrichment at relapse or progression and in the *NUP98::NSD1* subtype

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To the Editor:

NUP98 rearrangements define biologically distinct pediatric myeloid neoplasms with fusion-partner-associated differentiation programs.[1, 2] CD19 expression in acute myeloid leukemia (AML) is best recognized in *RUNX1::RUNX1T1* AML, but CD19 expression alone is not lineage-defining and may change over time. It can complicate B/myeloid lineage assignment, contribute to leukemia-associated immunophenotypes used for measurable residual disease (MRD) assessment, and provide a potential therapeutic target.[3, 4] Our prior clinicopathologic study of 62 children and young adults with *NUP98*-rearranged myeloid neoplasms described fusion-partner-associated patterns and qualitative CD19 changes across sequential specimens.[1] Here, we extend those observations through a focused quantitative analysis of CD19-positive blasts and their CD19 expression intensity in relation to disease status, fusion partner, immunophenotype, cooperating mutations, and treatment context. CD19 expression on ≥10% of blasts were enriched at relapse/progression and in *NUP98::NSD1* cases, while remained below this threshold in all *NUP98::KDM5A* cases; CD19 expression was also associated with CD34/CD7/CD13 expression and *NRAS/KRAS* and *CEBPA* mutations. The median fluorescence intensity (MFI) of CD19 expression strongly correlated with CD19-positive blast fraction.

For cross-sectional analysis, the first specimen from each patient evaluated at St. Jude Children's Research Hospital was included. CD19-positive blast fraction was evaluable in 61 of 62 patients: 37 newly diagnosed, 21 relapse/progression, and 3 therapy-related specimens. Subsequent specimens were used only for within-patient longitudinal assessment. All CD19 MFI measurements were acquired on the BD FACSCanto II flow cytometer and analyzed in FCS Express. APC- or PE-Cy5-conjugated CD19 antibodies were interpreted against an isotype control. The ≥10% threshold was used as study-specific operational grouping; MFI was analyzed by within-cohort quartile (Q1-3 vs Q4; cutoff, 1,847). Fisher exact, Mann-Whitney U, and Spearman correlation tests were used as appropriate. The institutional review boards of St. Jude approved the study and waived informed consent.

Among 61 evaluable patients, CD19-positive blast fractions ranged from 0% to 89%, and 15 (25%) had CD19 on ≥ 10% of blasts. These included 4 of 37 newly diagnosed specimens (11%), 11 of 21 relapse/progression specimens (52%), and 0 of 3 therapy-related specimens (Figure 1A; Table 1). The odds were higher in relapse/progression than in other specimens (OR, 9.9; 95% CI, 2.6-37.9; P=0.0005). Analyzed over the course, median CD19-positive blast fraction was 13% at relapse/progression versus 0% at de novo diagnosis (*P*=0.0007). CD19 expression

was also enriched in *NUP98::NSD1* (13/ 34 [38%] vs 2/27 [7%]; OR, 7.7; 95% CI, 1.6-38.2; P=0.007) when compared to all other *NUP98* fusions combined, with a higher continuous distribution in *NUP98::NSD1* (*P*=0.004; Figure 1B); all 12 *NUP98::KDM5A* specimens remained below 10%.

Among 35 specimens with MFI data, median CD19 MFI was 275 (IQR, 134-1,530), with medians of 145 at de novo diagnosis and 762 at relapse/ progression. MFI correlated strongly with CD19-positive blast fraction (Spearman ρ=0.82; *P*<0.0001; Figure 1C). All nine Q4 specimens were *NUP98::NSD1* versus 10/26 Q1-3 specimens (*P*=0.001), and seven were relapse/progression specimens (Table 1). These concordant findings support blast fraction as the primary quantitative measure, with MFI providing complementary information on antigen intensity.

Specimens with at least 10% CD19-positive blasts showed a distinct immunophenotypic profile: CD34 was expressed in all 15 versus 28 of 46 specimens below the threshold (61%; *P*=0.003); CD7 was present in 73% versus 30% (*P*=0.006), and CD13 in 100% versus 74% (*P*=0.028). In the MFI analysis, all nine Q4 specimens expressed CD34 versus 15 of 26 Q1-3 specimens (58%; *P*=0.033). Median absolute monocyte count was approximately tenfold lower in the ≥10% group (101.5/mm³ vs 1,008/mm³; *P*=0.004). Other evaluated flow-cytometric markers did not differ significantly between groups (Online Supplementary Table S2). Other demographic and hematologic variables were similar between groups; a diploid karyotype was more frequent in MFI Q4 than Q1-3 (67% vs 21%; P=0.035) (Table 1).

At the molecular level, *NRAS*/*KRAS* mutations occurred in 7/14 evaluable specimens (50%) with ≥10% CD19-positive blasts versus 7/43 (16%) with <10% CD19-positive blasts (*P*=0.027), and *CEBPA* mutations in 4/14 (29%) versus 1/43 (2%; *P*=0.011) (Figure 1D; Table 1). *WT1* alterations occurred in 64% versus 34% (*P*=0.064), *FLT3*-ITD in 36% versus 40% (P>0.999), and *PTPN11* mutations in 14% versus 5% (*P*=0.250).

For other B-cell markers, 7/11 (64%) CD19-positive relapse/progression specimens expressed CD22 and/or cytoplasmic CD79a, generally with dim, partial, or heterogeneous expression. In sequential specimens, the expression patterns of B markers ranged from isolated CD19 to broader B-lineage-associated expression, including partial/moderate CD22 with moderate cyCD79a+ in case 28 and bright cyCD79a in case 29. CD19 was acquired in cases 3, 28, 29, and 30 and lost in cases 10, 11, and 28. In case 31, CD19-positive blasts decreased from 30% to 20% and then 2% across sequential relapses (Online Supplementary Figure S1).

Under current WHO-HAEM5/ICC criteria for B/myeloid mixed-phenotype acute leukemia (MPAL), weak CD19 expression must be accompanied by strong expression of at least two of the following markers: CD10, CD22, and CD79a. With strong CD19, WHO-HAEM5 requires at least one additional strongly expressed B-lineage marker, whereas the published ICC table specifies at least one additional marker without an explicit intensity qualifier; myeloid lineage must be established independently by myeloperoxidase or at least two monocytic markers.[5-7] On retrospective review of all 15 CD19-positive cases in our cohort, none fulfilled current WHO-HAEM5 B-lineage criteria, and therefore none met WHO-HAEM5 criteria for MPAL. The four CD19-positive diagnostic specimens had dim CD19 without qualifying additional B-lineage markers. Among relapse/progression specimens, case 32 showed CD19 expression comparable to normal B cells, with dim CD22 and cytoplasmic CD79a and absent CD10 (Online Supplementary Table S1). This pattern met the B-lineage marker combination specified in the published ICC criteria but lacked the additional strongly expressed B-lineage marker required by WHO-HAEM5. The remaining cases showed isolated CD19 or insufficient additional B-lineage marker expression for assignment under either classification. The previously reported *NUP98::NSD1* B/myeloid MPAL illustrates this diagnostic boundary but was not part of this cohort.[8]

CD19 was present in only 2/24 cases (8%) in our separate series of myeloid neoplasms with *UBTF* tandem duplications, despite shared HOX-high biology.[9] Together with the absence of ≥10% CD19 expression in *NUP98::KDM5A*, this supports preferential association of CD19 with the *NUP98::NSD1* molecular context rather than HOX dysregulation broadly.[1, 2]

Treatment and outcome data were interpreted descriptively because the cohort spanned 1994–2025, during which induction regimens, transplant approaches, supportive care, and targeted therapies changed substantially; a reliable survival comparison by CD19 group was therefore not feasible. Stem-cell transplantation rates were similar in the ≥10% and <10% groups (10/14 [71%] vs 24/36 [67%]; $P>0.999$), as were documented complete-remission rates (4/14 [29%] vs 17/38 [45%]; $P=0.353$).

CD19 expression on AML blasts may potentially serve as a therapeutic target. In a prospective phase II study of 10 patients with relapsed CD19-positive t(8;21) AML, all achieved complete

remission with CD19 CAR-T therapy, and six achieved molecular MRD-negative remission. Six relapsed, including five with CD19-negative disease; the median leukemia-free survival was 3.8 months.[10] In the five-patient with BlinAML trial, three achieved complete molecular remission and one a molecular response with blinatumomab treatment.[11] These studies establish feasibility of targeting CD19 in AML, while antigen loss can provide a route to relapse. Targeting CD19 in *NUP98*-rearranged AML is not explored, but CD19-positive *NUP98::NSD1* is a rational setting for prospective trial given its poor response to current treatment protocols. Of note, as demonstrated in our study, CD19 expression should be reassessed at relapse/progression and before therapy rather than inferred solely from the initial diagnostic specimen.

Menin inhibition provides a relevant setting in which treatment-associated immunophenotypic changes, potentially including changes in CD19 expressions, may occur. Preclinical studies identified the menin-*KMT2A* interaction as a molecular dependency in *NUP98*-rearranged AML.[12] Complete remission with icovamenib (BMF-219) monotherapy has been reported in a patient with relapsed *NUP98::NSD1* AML.[13] In a revumenib-treated cohort, 16/31 evaluable patients (52%) showed treatment-associated immunophenotypic changes; three of 48 patients had *NUP98* rearrangements, but CD19-specific changes were not reported.[14] The enrichment of *NRAS/KRAS* mutations in specimens with at least 10% CD19-positive blasts (50% vs 16%; P=0.027) also raises the question of whether *RAS*/*MAPK* signaling influences CD19 expression. Because CD19 changed across sequential specimens in our cohort, assessment before and during menin inhibition would be informative. The clinically and molecularly defined subgroups remain small, precluding multivariable assessment of whether relapse/progression status and *NUP98::NSD1* are independently associated with CD19 expression. Concordant blast-fraction and MFI findings, together with within-patient gain and loss of CD19, identify a dynamic quantitative phenotype enriched at relapse/progression and associated with *NUP98::NSD1*. The concurrent expression of other B markers in a subset of cases may raise consideration of B/myeloid MPAL. For MRD assessment, CD19 may contribute to a patient-specific leukemia-associated immunophenotype but should be interpreted with the broader phenotype and molecular context, and reassessed when it may affect classification, MRD interpretation, or treatment selection. Reporting both CD19-positive blast fraction and intensity may be useful when CD19 is considered for MRD tracking or therapeutic targeting.

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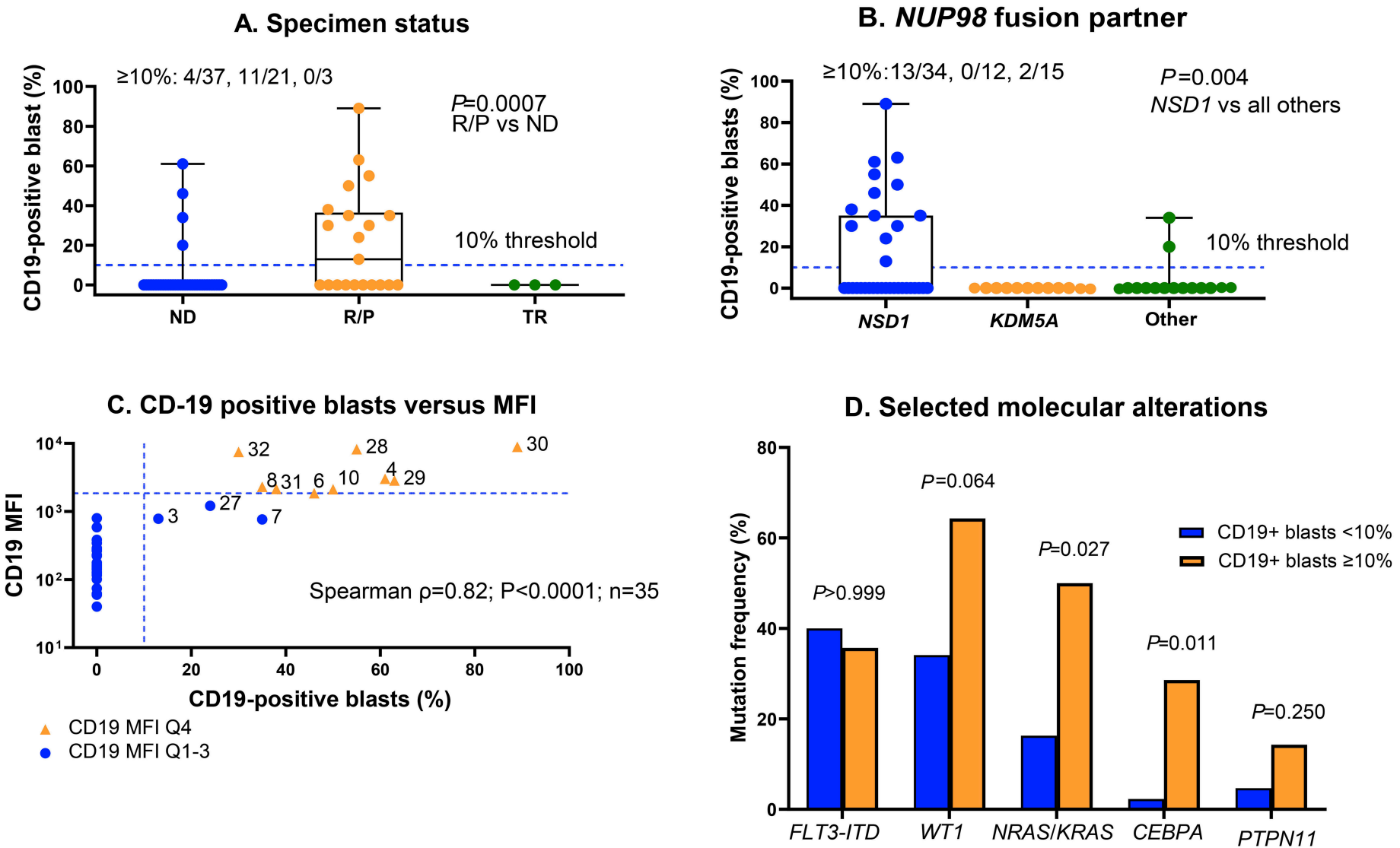
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Table 1. Clinicopathologic, treatment, immunophenotypic, and molecular correlates of quantitative CD19 expression in *NUP98*-rearranged myeloid neoplasms.

Characteristic	Entire cohort (n=62)	CD19+ blasts <10% (n=46)	CD19+ blasts ≥10% (n=15)	P value	CD19 MFI Q1-3 (n=26)	CD19 MFI Q4 (n=9)	P value
Age, years, median (range)	10.0 (0.6-20.2)	9.0 (0.6-20.0)	11.0 (2.0-15.0)	0.568	6.0 (0.6-20.0)	11.0 (3.0-15.0)	0.505
Sex (female/male)	23/39	18/28	4/11	0.539	9/17	2/7	0.685
R/P specimen, n/N (%)	21/62 (34)	10/46 (22)	11/15 (73)	**0.0005**	12/26 (46)	7/9 (78)	0.135
SCT, n/N (%)	34/50 (68)	24/36 (67)	10/14 (71)	>0.999	13/20 (65)	7/9 (78)	0.675
Complete remission	21/52 (40)	17/38 (45)	4/14 (29)	0.353	8/21 (38)	3/9 (33)	>0.999
WBC, ×10⁹/L, median (range)	15.9 (0.6-460.0)	15.9 (0.6-460.0)	18.0 (0.6-437.3)	0.509	12.3 (0.6-460.0)	18.0 (0.7-437.3)	0.618
HGB, g/dL, median (range)	8.7 (4.4-13.0)	8.6 (4.8-13.0)	8.7 (4.4-11.5)	0.834	7.8 (4.8-10.1)	8.7 (4.4-11.5)	0.569
Platelets, ×10⁹/L, median(range)	67 (4-398)	68 (4-216)	67 (19-398)	0.760	70.5 (4-216)	47 (19-398)	0.495
PB blasts, %, median (range)	32.5 (0-99)	39 (1-99)	29 (0-97)	0.490	32.5 (0-99)	29 (2-97)	0.703
AMC, /mm³, median (range)	360 (0-49,464)	1,008 (0-49,464)	101.5 (0-8,746)	**0.004**	439 (0-43,714)	81 (0-8,746)	0.122
BM blasts, %, median (range)	61 (12-99)	67 (15-95)	36 (12-99)	0.355	63.5 (21-94)	36 (12-80)	0.294
CNS involvement, n/N (%)	18/46 (39)	14/32 (44)	4/14 (29)	0.513	9/19 (47)	2/8 (25)	0.405
Diploid karyotype, n/N (%)	21/50 (42)	14/35 (40)	7/14 (50)	0.542	4/19 (21)	6/9 (67)	**0.035**
Balanced translocation, n/N (%)	18/50 (36)	12/35 (34)	6/14 (43)	0.744	9/19 (47)	3/9 (33)	0.687
Complex karyotype, n/N (%)	11/50 (22)	9/35 (26)	1/14 (7)	0.244	6/19 (32)	0/9 (0)	0.136
Trisomy 8, n/N (%)	5/56 (9)	4/40 (10)	1/15 (7)	>0.999	3/24 (13)	0/9 (0)	0.545
11p15 abnormality, n/N (%)	11/57 (19)	9/41 (22)	2/15 (13)	0.708	4/24 (17)	0/9 (0)	0.555
Trisomy 21, n/N (%)	7/56 (13)	7/40 (18)	0/15 (0)	0.171	5/24 (21)	0/9 (0)	0.290
5q abnormality, n/N (%)	9/55 (16)	7/39 (18)	2/15 (13)	>0.999	5/23 (22)	2/9 (22)	>0.999
CD34 expression, n/N (%)	44/62 (71)	28/46 (61)	15/15 (100)	**0.003**	15/26 (58)	9/9 (100)	**0.033**
CD7 expression, n/N (%)	25/61 (41)	14/46 (30)	11/15 (73)	**0.006**	9/26 (35)	6/9 (67)	0.129
CD13 expression, n/N (%)	49/61 (80)	34/46 (74)	15/15 (100)	**0.028**	17/26 (65)	9/9 (100)	0.074
NUP98::NSD1, n/N (%)	35/62 (56)	21/46 (46)	13/15 (87)	**0.007**	10/26 (38)	9/9 (100)	**0.001**
Other *NUP98* fusion, n/N (%)	27/62 (44)	25/46 (54)	2/15 (13)	—	16/26 (62)	0/9 (0)	—
FLT3-ITD, n/N (%)	23/60 (38)	18/45 (40)	5/14 (36)	>0.999	7/26 (27)	3/8 (38)	0.666
WT1 alteration, n/N (%)	24/59 (41)	15/44 (34)	9/14 (64)	0.064	9/25 (36)	5/8 (63)	0.238
NRAS/*KRAS* mutation, n/N (%)	14/58 (24)	7/43 (16)	7/14 (50)	**0.027**	6/24 (25)	5/8 (63)	0.088
CEBPA mutation, n/N (%)	5/57 (9)	1/43 (2)	4/14 (29)	**0.011**	0/24 (0)	3/8 (38)	**0.011**
PTPN11 mutation, n/N (%)	4/57 (7)	2/43 (5)	2/14 (14)	0.250	2/24 (8)	2/8 (25)	0.254

Data are n/N (%) or median (range); denominators vary according to test availability. One patient lacked a quantitative CD19-positive blast fraction. P values are from two-sided Fisher exact tests for categorical variables, and Mann-Whitney U tests for continuous variables. The 10% threshold was used for group comparisons. Q4 denotes the highest within-cohort CD19 MFI quartile (cutoff, 1,847). **Abbreviations:** AMC, absolute monocyte count; BM, bone marrow; CNS, central nervous system; HGB, hemoglobin; ITD, internal tandem duplication; MFI, median fluorescence intensity; PB, peripheral blood; R/P, relapse/progression; SCT, stem-cell transplantation; WBC, white blood cell count.

Figure 1. Quantitative CD19 expression by specimen status, *NUP98* fusion partner, antigen intensity, and selected molecular alterations. (A) CD19-positive blast fraction by specimen status. Points represent individual index specimens; boxes show median and interquartile range. The P value compares relapse/progression with de novo specimens by Mann-Whitney U test; the upper-left label gives the number meeting the ≥10% operational threshold/denominator. (B) CD19-positive blast fraction by fusion partner. The P value compares *NUP98::NSD1* with all other *NUP98* fusions by Mann-Whitney U test. (C) Relationship between CD19 MFI and CD19-positive blast fraction among 35 evaluable specimens; MFI is shown on a logarithmic scale. Dashed lines indicate the 10% blast-fraction threshold and cohort-derived Q4 MFI cutoff (1,847). Numbers identify cases with detectable CD19. Spearman ρ=0.82; *P*<0.0001. (D) Frequencies of selected molecular alterations *(FLT3-ITD, WT1,* NRAS/KRAS, *CEBPA,* and *PTPN11)* in specimens with <10% versus ≥10% CD19-positive blasts; gene-specific denominators are provided in Table 1 and P values are from two-sided Fisher exact tests. MFI, median fluorescence intensity; ND, de novo (new diagnosis); R/P, relapse/progression; TR, therapy related.


A. Specimen status
≥10%: 4/37, 11/21, 0/3
P=0.0007
R/P vs ND
10% threshold
CD19-positive blast (%)
ND
R/P
TR
B. NUP98 fusion partner
≥10%:13/34, 0/12, 2/15
P=0.004
NSD1 vs all others
10% threshold
CD19-positive blasts (%)
NSD1
KDM5A
Other
C. CD-19 positive blasts versus MFI
Spearman ρ=0.82; P<0.0001; n=35
CD19 MFI
CD19-positive blasts (%)
CD19 MFI Q4
CD19 MFI Q1-3
D. Selected molecular alterations
P>0.999
P=0.064
P=0.027
P=0.011
P=0.250
Mutation frequency (%)
CD19+ blasts <10%
CD19+ blasts ≥10%
FLT3-ITD
WT1
NRAS/KRAS
CEBPA
PTPN11

Online Supplementary Material

Quantitative CD19 expression in pediatric *NUP98*-rearranged myeloid neoplasms: enrichment at relapse or progression and in the *NUP98::NSD1* subtype

Online Supplementary Table S1. Longitudinal CD19 and accompanying B-lineage-associated antigen changes in eleven CD19-positive relapse/progression specimens.

Case	Initial or earliest evaluable immunophenotype	First CD19-positive relapse/progression immunophenotype	Later disease immunophenotype	CD19 change over time
3	Myeloid; CD19-, CD10-, CD22-, cyCD79a-	Monocytic; CD19+ (dim/partial), CD10-, CD22-, cyCD79a-	Monocytic; CD19+ (dim), CD10-, CD22-, cyCD79a-	Acquired and retained
8	Not available	Myeloid; CD19+ (dim/partial), CD22+ (moderate), cyCD79a+ (moderate), CD10-	Not available	CD19 positive at first evaluable relapse
10	Not available	Myeloid; CD19+ (dim/partial), CD7+, CD10-, CD22-	Myeloid; CD19-, CD7-	Complete loss
11	Not available	Myelomonocytic; CD19+ (dim/partial), CD7+, CD22-, cyCD79a-	Myelomonocytic; CD19-, CD7-	Complete loss
27	Not available	Myeloid; CD19+ (dim/partial), CD7+, CD10-, CD22-, cyCD79a+ (dim)	Myelomonocytic; CD19+, CD7-, CD10-, CD22-, cyCD79a+ (dim)	CD19 retained; CD7 lost
28	Monocytic; CD19-, CD10-, CD22-, cyCD79a not tested	Myelomonocytic; CD19+ (dim/partial), CD7+, CD10-, CD22+ (partial/moderate), cyCD79a+ (moderate)	Immature monocytic; CD19-, CD10-, CD22-, cyCD79a-, CD7+ (retained)	Acquired, then lost
29	Myeloid; CD19-, CD10-, CD22-, cyCD79a-	Myelomonocytic; CD19+ (dim/partial), CD10-, CD22-, cyCD79a+ (bright)	Not available	Acquired
30	Myelomonocytic; CD19-; other B-lineage markers not tested	Myeloid; CD19+ (dim/partial), CD10-, CD22-, cyCD79a-	Not available	Acquired
31	Not available	Myelomonocytic; CD19+ (dim/partial), CD7+, CD10-, CD22-, cyCD79a+ (dim)	Myeloid; CD19+ (20%), CD22+ (dim), CD7+, CD56+, CD10-, cyCD79a-. At later relapse, CD19 expression in 2% of blasts (below the 10% study threshold), CD10-, CD22-, cyCD79a-.	Declined below 10% at third relapse
32	Myelomonocytic; CD19+, CD10-, CD22+ (dim), cyCD79a+ (dim)	Myeloid; CD19+ (moderate/bright), CD10-, CD22+ (dim), cyCD79a+ (dim)	Not available	Retained with broader B-lineage-associated pattern
45	Myelomonocytic; CD19+ (dim), CD2+, CD7+, CD10-, CD22-, cyCD79a-	Poorly differentiated/myeloid; CD19+ (dim), CD22+ (dim), CD7+ (subset), CD10-, cyCD79a-	Not available	CD19 retained; CD22 acquired in a subset

This table summarizes CD19-focused longitudinal immunophenotypic findings from selected cases previously reported in Table 4 of reference 1. It excludes unrelated cases, karyotypes, broad immunophenotypic category shifts, and time-to-relapse data. Seven of 11 CD19-positive relapse/progression specimens expressed CD22 and/or cytoplasmic CD79a, generally with dim, partial, or heterogeneous expression. CD19 was acquired in cases 3, 28, 29, and 30 and completely lost in cases 10,

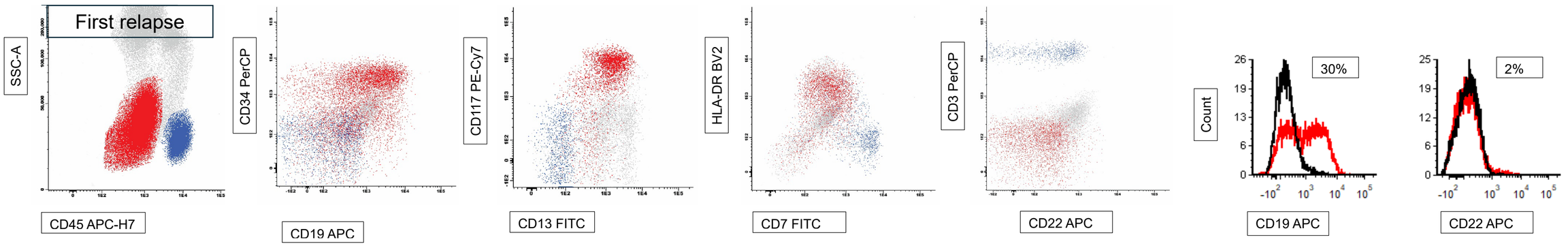
11, and 28; because case 28 both acquired and later lost CD19, these events involved six unique patients. Case 31 retained CD19 in sequential relapse specimens before decreasing below the 10% study threshold. Cases 10 and 11 document complete loss between sequential relapse specimens because an initial diagnostic immunophenotype was unavailable. cyCD79a, cytoplasmic CD79a.

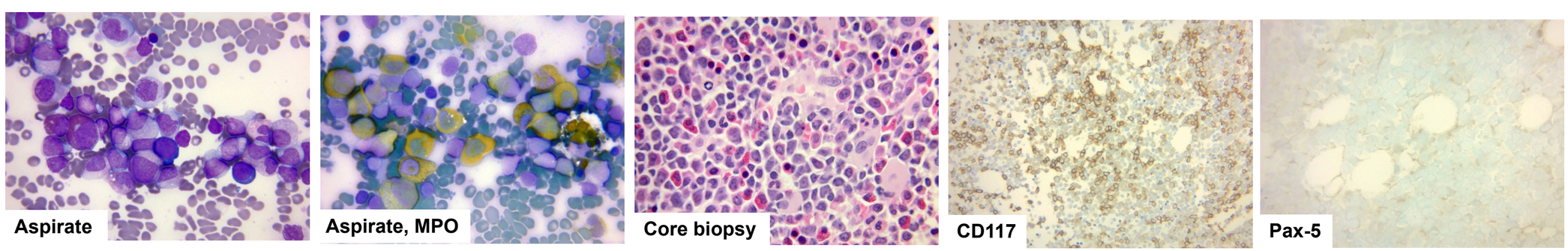
Online Supplementary Table S2. Flow cytometry immunophenotypic correlates of quantitative CD19 expression in NUP98-rearranged myeloid neoplasms.

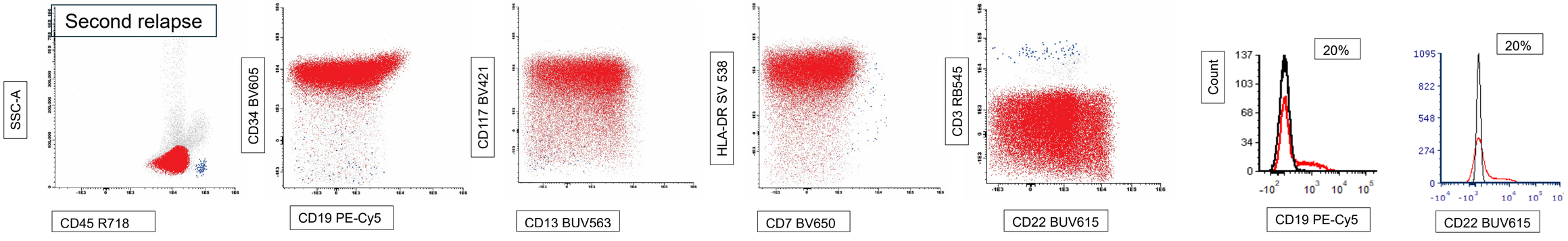
Marker	Entire cohort (n=62)	CD19+ blasts <10% (n=46)	CD19+ blasts ≥10% (n=15)	P value	CD19 MFI Q1-3 (n=26)	CD19 MFI Q4 (n=9)	P value
CD64	30/56 (54)	21/40 (53)	8/15 (53)	>0.999	9/22 (41)	3/9 (33)	>0.999
CD11b	45/58 (78)	32/44 (73)	13/14 (93)	0.155	18/25 (72)	8/9 (89)	0.403
CD14	18/56 (32)	13/40 (33)	4/15 (27)	0.754	7/23 (30)	2/9 (22)	>0.999
CD13	49/61 (80)	34/46 (74)	15/15 (100)	**0.028**	17/26 (65)	9/9 (100)	0.074
CD15	39/56 (70)	26/41 (63)	12/14 (86)	0.183	14/26 (54)	8/9 (89)	0.109
CD33	59/62 (95)	43/46 (93)	15/15 (100)	0.569	24/26 (92)	9/9 (100)	>0.999
CD34	44/62 (71)	28/46 (61)	15/15 (100)	**0.003**	15/26 (58)	9/9 (100)	**0.033**
CD117	53/61 (87)	37/45 (82)	15/15 (100)	0.182	24/26 (92)	9/9 (100)	>0.999
CD38	36/39 (92)	23/26 (88)	13/13 (100)	0.538	23/25 (92)	9/9 (100)	>0.999
CD7	25/61 (41)	14/46 (30)	11/15 (73)	**0.006**	9/26 (35)	6/9 (67)	0.129
CD56	8/58 (14)	7/43 (16)	1/15 (7)	0.666	5/26 (19)	1/9 (11)	>0.999
HLA-DR	53/61 (87)	37/45 (82)	15/15 (100)	0.182	20/26 (77)	9/9 (100)	0.304
CD123	31/38 (82)	19/25 (76)	12/13 (92)	0.385	19/24 (79)	9/9 (100)	0.290

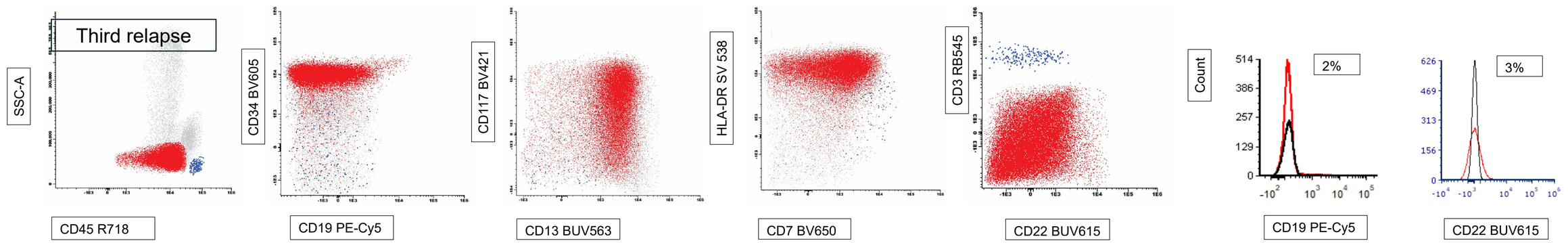
Data are n/N (%). Denominators vary by marker availability. P values are from two-sided Fisher exact tests. MFI, median fluorescence intensity.

Online Supplementary Figure S1. Longitudinal flow-cytometric, morphologic, and immunohistochemical findings in patient 31 with *NUP98*-rearranged myeloid neoplasm. Flow-cytometry plots show sequential disease specimens from first, second, and third relapse (top to bottom). The abnormal population is shown in red and the lymphocyte/internal-control population in blue. CD19-positive blasts decreased from 30% at first relapse to 20% at second relapse and 2% at third relapse; CD22-positive blasts were 2%, 20%, and 3%, respectively. Morphologic and immunohistochemical findings from the first-relapse specimen include bone marrow aspirate (x600), MPO cytochemistry (x600), core biopsy (x400), CD117 immunohistochemistry (x200), and Pax-5 immunohistochemistry (x200); blasts are Pax-5 negative.


First relapse
SSC-A
CD45 APC-H7
CD34 PerCP
CD19 APC
CD117 PE-Cy7
CD13 FITC
HLA-DR BV2
CD7 FITC
CD3 PerCP
CD22 APC
Count
30%
CD19 APC
2%
CD22 APC


Aspirate
Aspirate, MPO
Core biopsy
CD117
Pax-5


Second relapse
SSC-A
CD45 R718
CD34 BV605
CD19 PE-Cy5
CD117 BV421
CD13 BUV563
HLA-DR SV 538
CD7 BV650
CD3 RB545
CD22 BUV615
Count
20%
CD19 PE-Cy5
20%
CD22 BUV615


Third relapse
SSC-A
CD45 R718
CD34 BV605
CD19 PE-Cy5
CD117 BV421
CD13 BUV563
HLA-DR SV 538
CD7 BV650
CD3 RB545
CD22 BUV615
Count
2%
CD19 PE-Cy5
3%
CD22 BUV615