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Insights into clonal and complete blood count changes at disease evolution among patients with clonal cytopenia of undetermined significance

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To The Editor:

Clonal cytopenia of undetermined significance (CCUS) is defined as clonal hematopoiesis (CH) in the absence of a hematologic neoplasm, along with unexplained cytopenia.[1, 2] While CCUS is recognized as a precursor state with an elevated risk of progression to myelodysplastic syndrome (MDS) or acute myeloid leukemia (AML), the biological mechanisms and clinical factors influencing this transition remain incompletely understood.[3] A recent prospective study showed that specific mutations significantly affect blood counts and overall survival (OS), while the number of mutations had the highest predictive value for disease progression.[4] Emerging evidence suggests that several factors are associated with an increased risk of progression from CCUS to overt myeloid neoplasms, including the presence of high-risk mutations (e.g., in *TP53, RUNX1*, or spliceosome genes), a variant allele frequency (VAF) greater than 20%, and a higher number of co-occurring mutations.[5-7] Despite current insights, the clinical and molecular features of disease progression remain poorly characterized. This study aims to evaluate the clinical and clonal profiles of CCUS patients, focusing on the longitudinal evolution of these parameters over time.

After institutional review board and ethics committee approval, we retrospectively reviewed charts of patients with unexplained cytopenia (2015-2024), recording data at the time of CCUS diagnosis (based on WHO 5th edition diagnostic criteria[2]) and at time of follow-up bone marrow biopsy after diagnosis. The clonal hematopoiesis risk score (CHRS)[6], clonal cytopenia risk score (CCRS)[7], revised international prognostic scoring system (IPSS-R)[8], and the molecular international prognostic scoring system (IPSS-M)[9] were used for risk stratification (Only cases with complete data were included in score calculations). All 212 patients CCUS cases were diagnosed with CCUS based on BM biopsy and NGS. Follow-up complete blood count (CBC) and karyotype data were recorded for patients who had repeat bone marrow biopsies (BMB) (n= 109), while follow-up clonal parameters and risk scores were calculated for those who underwent repeat next-generation sequencing (NGS; n= 74). In case of multiple follow-up BMB/NGS, we recorded the most recent one for patients who did not progress to MN, and the one at progression for those who progressed. A reduction or expansion in clone size was defined as a change exceeding 5% ($CE^{5\%}$), or change exceeding 10% ($CE^{10\%}$) from the baseline value. Overall survival (OS) was

calculated from diagnosis to last follow-up. For statistical analysis, we used BlueSky Statistics V10.3.4. Continuous variables were compared using ANOVA test, while categorical variables were analyzed using the chi-square test or Fisher's exact test, as appropriate. The NGS panel consists of 43-47 genes, has accuracy >99%; reproducibility 100% (both intra- and inter-assay), and its sensitivity ranges from 5-10% variant allele fraction.

Two hundred and twelve CCUS patients (median age 72 years, 68% male) were included. Abnormal cytogenetic changes were present in 30% (65/212) of patients, with del (20q) being the most common (8%; n= 18), followed by loss of chromosome Y (7% of males; n= 10), and trisomy 8 (6%; n= 13). The median number of mutations was 2 (range, 1-5), with the most common mutation being *TET2* (37%, 74/212), followed by *SRSF2* (21.7%, 43/212), and *ASXL1* (21%, 42/212). Twenty-one percent (n = 45/212) had therapy-related CCUS, and 15% (n = 32/212) had a family history of hematological malignancy. Most patients had high risk CHRS (60%, 104/174), followed by intermediate (30%, 53/174), and low risk (10%, 17/174). On the other hand, 41% (81/196) had high risk CCRS, 29% (57/196) had intermediate risk, and 30% (59/196) had low risk (**Table 1**). After a median follow-up of 46 months, 63 patients died (median OS not reached), and 63 (30%) patients progressed to myeloid neoplasms (MN; 47 to MDS, 15 to CMML, and 1 to AML). 14 out of the 15 cases who progressed to CMML were sub-classified as Clonal Cytopenia and monocytosis of undetermined significance (CCMUS).

When comparing deceased and surviving patients, survivors had higher median hemoglobin levels compared to deceased patients (11 vs 9.8, p=0.02), however no significant difference was seen in WBC, platelet counts, bone marrow or peripheral blasts (p>0.05). 41% (26/63) of deceased patients had abnormal karyotype, compared to 26% (39/149) among the survivor group (p=0.03). Progression to myeloid neoplasm was seen more frequently among the deceased group (40%, 25/63) compared to the survivor group (25.5%, 38/149) (p=0.048). The median number (Q1-Q3) of mutations was higher in the deceased group (2; 1-3) compared to the survivor group (1; 1-2) (p=0.01). mutations were evenly distributed between the 2 groups (p>0.05) except ASXL1 that was seen more frequently in the deceased group (19/63 (33%) vs 23/149 (16%), p=0.01).

The median time between diagnosis and follow-up BMB was 18 months (Q1= 10; Q3= 29, months). Among the 109 patients with available follow-up labs, 74% (80/109) experienced a decrease in platelet count, 61% (66/109) a decrease in hemoglobin levels, and 62% (67/109) a decrease in white blood cell (WBC) counts. A decrease in the WBC count from the time of diagnosis to follow-up was more frequently associated with progression to MN (71%, 44/62 vs 50%, 23/46, p= 0.026); however, changes in hemoglobin and platelets did not differ between those who progressed and those who did not (p> 0.05). Although the frequency of abnormal karyotypes at diagnosis was similar between patients who eventually progressed (28%, 18/63) and those who did not (32%, 47/149) (p= 0.7), 4 (2%) patients with initially normal karyotypes acquired chromosomal abnormalities during follow-up (including +X, -7, +21, del20), and all progressed to MDS (**Table s1**)

The median time between diagnosis and follow-up NGS was 22 months (Q1= 13; Q3= 41, months). Among the 74 patients who underwent follow-up NGS, 31% (22/74) acquired new mutations. Clonal expansion of pre-existing mutations was seen in 54% (37/74) using $CE^{5\%}$ and 19% (13/74) per $CE^{10\%}$, and 16% (11/74) developed new variants of uncertain significance (VUS). We further went through each patient's genomic evolution data as represented in **Figure 1**. The frequency of acquisition of new pathogenic mutations was similar in patients with and without progression to MN (34%(13/40) vs 28%(9/34), p= 0.6); whereas, clonal expansion was significantly more frequently observed in patients who progressed compared to those who did not, using both $CE^{5\%}$ (73%(27/40) vs 31%(10/34), p <0.001) and $CE^{10\%}$ (27%(10/40) vs 9%(3/34), p= 0.03) (**Table s1**). At follow-up, *ASXL1* was the most commonly acquired mutation among patients who did not progress (4 cases), followed by *TET2* (2 cases), and *U2AF1*, *GATA2*, *DNMT3A*, *SF3B1*, *ZRSR2*, *TP53*, and *CBL* (1 case each). On the other hand, *SRSF2*, *SETBP1*, *CBL*, *JAK2*, *TET2*, and *RUNX1* were more frequently acquired among those who progressed to myeloid neoplasms (each appeared in 2 patients). When recalculating CCRS, CHRS, IPSS-M, and IPSS-R at follow-up for patients who had repeat NGS, there was no significant difference in score changes - in either direction - between those who progressed and those who did not (p> 0.05) (**Table s1). Figures 2A and 2B** display fishplots depicting clonal expansion, new mutations, and chromosomal abnormalities during progression from CCUS to MN.

Our retrospective cohort included 214 patients, with a subset undergoing follow-up BMB and NGS (111 with follow up BMB and 74 with follow up NGS). This subgroup likely represents a higher-risk population, as follow-up investigations are typically pursued when there is clinical suspicion of disease progression; this should be taken into consideration while interpreting the results of this study. The impact of cytogenetic aberrations on the outcomes of CCUS patients has been highly variable[4-6, 10]. In our study, we anticipate that the evolution of chromosomal aberrations over time (although not common), rather than their presence at diagnosis alone, may be more indicative of disease progression. None of the patients had MDS directed therapy during the follow up period, which could have potentially affected the evaluation of progression. A previous prospective study reported a negative impact on both platelet and hemoglobin levels in patients harboring mutations in chromatin modifier and transcription factor genes[4]. In our cohort, we did not observe a statistically significant difference, which may be due to the limited number of patients with available follow-up labs and the reliance on a single follow-up time point for comparison. A decrease in the WBC count might give an additional clue to physicians that a further workup is indicated if patients also have other signs of progression. Chien et al suggested that the loss of mutations over time predicts a worse OS and trend toward a shorter progression time[11]; we did not observe a significant association between the acquisition or loss of mutations and either OS or progression in our cohort. Limitations to our study include its retrospective design, which inherently introduces selection bias, particularly the tendency to monitor higher-risk cases more closely, and the relatively small number of patients with follow-up labs. Additionally, our analysis relied on only two time points (at diagnosis and follow-up), which may overlook dynamic fluctuations and other clinical factors that can influence laboratory parameters.

In conclusion, among CCUS patients who progressed to MN, we observed WBC decline, clonal expansion, and evolving chromosomal aberrations. This supports the value of longitudinal genomic and clinical monitoring through serial NGS, BMBs, and CBCs in the ongoing management of CCUS (These findings are not intended as definitive conclusions but rather as hypothesis-generating observations that warrant validation in larger, prospective studies).

References:

1. Arber DA, Orazi A, Hasserjian RP, et al. International Consensus Classification of Myeloid Neoplasms and Acute Leukemias: integrating morphologic, clinical, and genomic data. Blood. 2022;140(11):1200-1228.
2. Khoury JD, Solary E, Abla O, et al. The 5th edition of the World Health Organization Classification of Haematolymphoid Tumours: Myeloid and Histiocytic/Dendritic Neoplasms. Leukemia. 2022;36(7):1703-1719.
3. Jajosky AN, Sadri N, Meyerson HG, et al. Clonal cytopenia of undetermined significance (CCUS) with dysplasia is enriched for MDS-type molecular findings compared to CCUS without dysplasia. Eur J Haematol. 2021;106(4):500-507.
4. Cargo C, Bernard E, Beinortas T, et al. Predicting cytopenias, progression, and survival in patients with clonal cytopenia of undetermined significance: a prospective cohort study. Lancet Haematol. 2024;11(1):e51-e61.
5. Gu M, Kovilakam SC, Dunn WG, et al. Author Correction: Multiparameter prediction of myeloid neoplasia risk. Nat Genet. 2023;55(10):1777.
6. Weeks LD, Niroula A, Neuberg D, et al. Prediction of risk for myeloid malignancy in clonal hematopoiesis. NEJM Evid. 2023;2(5):10.1056/evidoa2200310.
7. Xie Z, Komrokji R, Al Ali N, et al. Risk prediction for clonal cytopenia: multicenter real-world evidence. Blood. 2024;144(19):2033-2044.
8. Greenberg PL, Tuechler H, Schanz J, et al. Revised international prognostic scoring system for myelodysplastic syndromes. Blood. 2012;120(12):2454-2465.
9. Bernard E, Tuechler H, Greenberg PL, et al. Molecular International Prognostic Scoring System for Myelodysplastic Syndromes. NEJM Evid. 2022;1(7):EVIDoa2200008.
10. Gao T, Ptashkin R, Bolton KL, et al. Interplay between chromosomal alterations and gene mutations shapes the evolutionary trajectory of clonal hematopoiesis. Nat Commun. 2021;12(1):338.
11. Chien KS, Braish JS, Li Z, et al. Clinicopathologic characteristics and outcomes of patients with clonal hematopoiesis who progress to myeloid neoplasms. Leukemia. 2025;39(5):1269-1272.

Table 1: General clinical and molecular characteristics for patients diagnosed with CCUS

	(N=212)
Age, median (range)	72 (19, 99)
Gender (male) , n (%)	146 (68.2%)
ANC 10^9/L, median (Q1, Q3)	1.7 (0.9, 3.3)
AMC 10^9/L, median (Q1, Q3)	0.4 (0.2, 0.6)
RDW, median (Q1, Q3)	15.4 (14.0, 17.0)
MCV fL, median (Q1, Q3)	99.6 (91.0, 106.4)
Hemoglobin g/dl, median (Q1, Q3)	10.8 (9.1, 12.3)
Platelets 10^9/L, median (Q1, Q3)	124.0 (78.0, 179.0)
WBC 10^9/L, median (Q1, Q3)	3.5 (2.4, 5.6)
Pancytopenia, n (%)	51 (24.2%)
Peripheral blasts, median (range)	0 (0, 2)
BM blasts, median (range)	1 (0, 5)
BMT, n (%)	19 (9.0%)
Abnormal karyotype, n (%)	65 (30.8%)
NGS, n (%)	198 (93.4%)
Number of co-mutations, median (range)	2 (1, 5)
Co-mutations	
TET2, n (%)	74 (37.4%)
SRSF2, n (%)	43 (21.7%)
ASXL1, n (%)	42 (21.2%)
ZRSR2, n (%)	36 (18.2%)
DNMT3A, n (%)	18 (9.1%)
U2AF1, n (%)	18 (9.1%)
IDH1, n (%)	13 (6.6%)
TP53, n (%)	10 (5.1%)
IDH2, n (%)	9 (4.5%)
RUNX1, n (%)	9 (4.5%)
Disease progression, n (%)	63 (30%)
MDS, n (%)	47 (74.6%)
CMML, n (%)	15 (24%)
AML, n (%)	1 (1.4%)
CHRS categories	
Low, n (%)	17 (9.8%)
Intermediate, n (%)	53 (30.5%)
High, n (%)	104 (59.8%)
CCRS categories	
Low, n (%)	59 (29.9%)
Intermediate, n (%)	57 (28.9%)
High, n (%)	81 (41.1%)

Abbreviations: ANC: Absolute neutrophilic count, AMC: Absolute monocytic count, RDW: Red cell distribution width, MCV: Mean corpuscular volume, WBC: White blood cells, BM: Bone marrow, BMT: Bone marrow transplantation, NGS: Next generation sequencing, MDS: Myelodysplastic syndrome, CMML: Chronic myelomonocytic leukemia, AML: Acute myeloid leukemia.

Figure legend: Figure 1) Oncoplot showing the genomic evolution of mutations between time of CCUS diagnosis and time of follow-up; Figure 2) Fishplots showing the clonal evolution and cytogenetic changes during progression to myeloid neoplasm; A) Fishplot representing the evolution from CCUS to MDS and then to AML, with the expansion of *SRSF2* and *TET2* mutations and acquiring *SETBP1*, *RUNX1*, and *ETNK1* mutations; B) Fishplot representing the evolution from CCUS to MDS, with the expansion of *SRSF2* mutation and acquiring cytogenetic aberration.

Figure 1

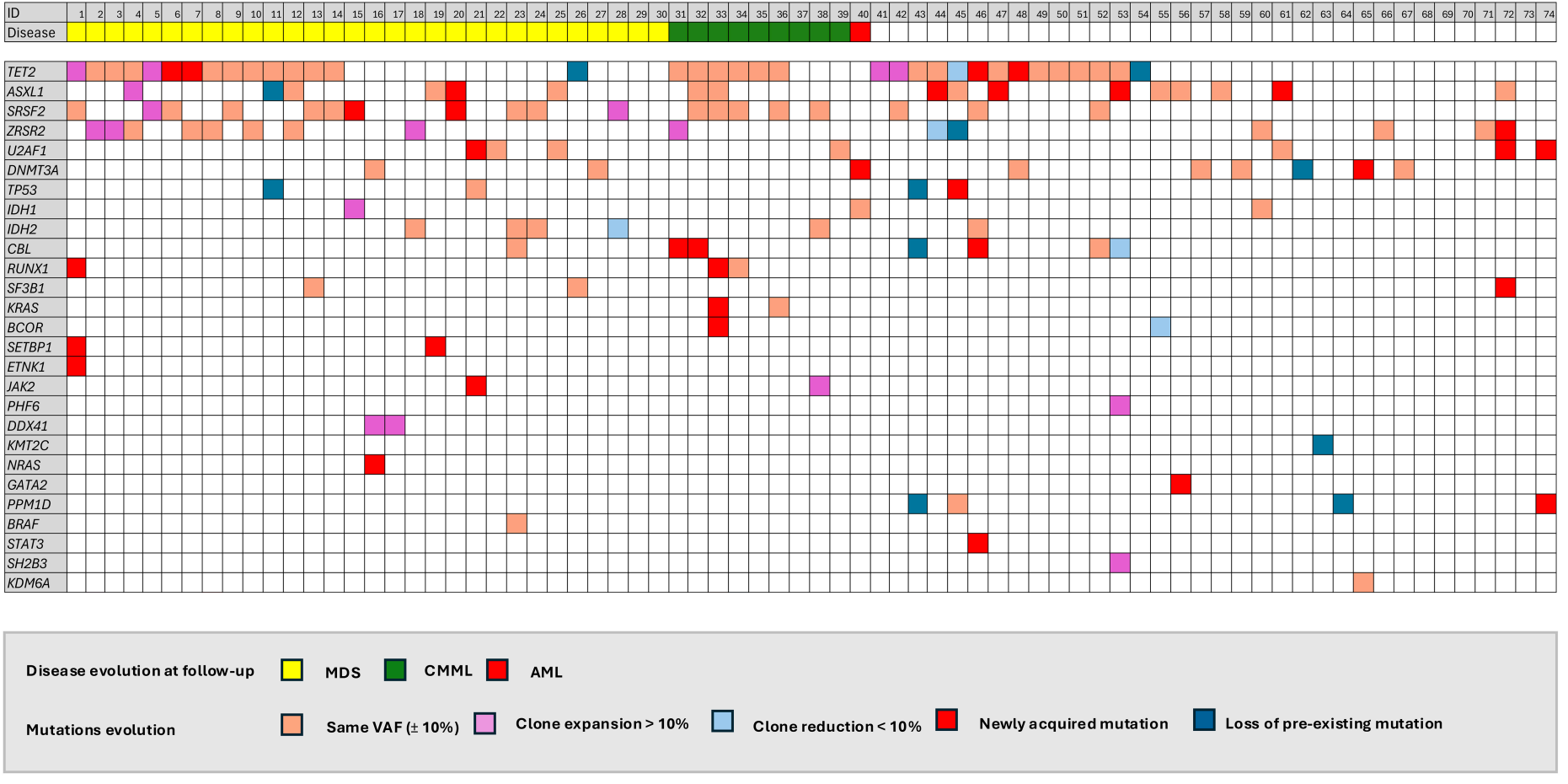
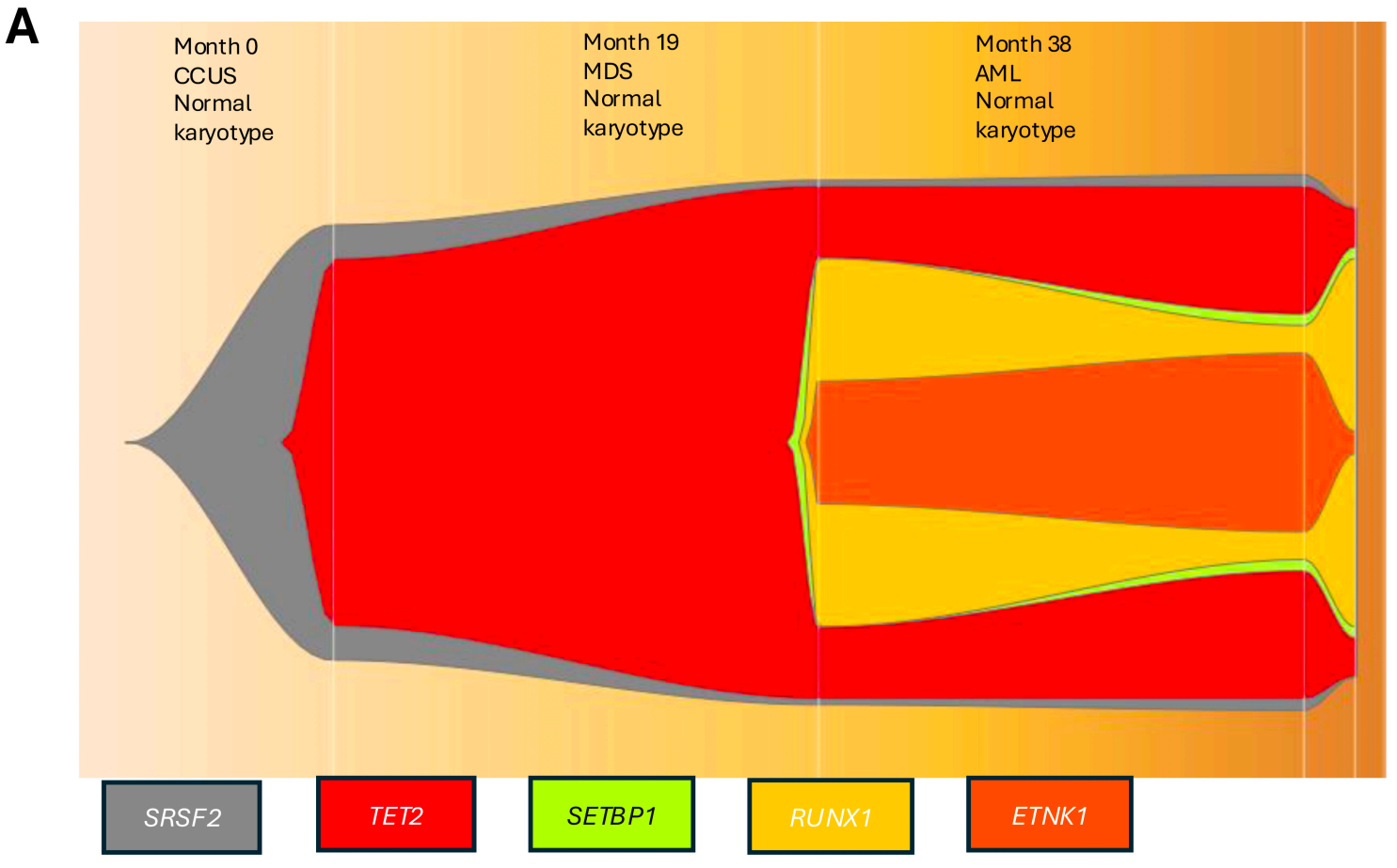

ID
Disease
TET2
ASXL1
SRSF2
ZRSR2
U2AF1
DNMT3A
TP53
IDH1
IDH2
CBL
RUNX1
SF3B1
KRAS
BCOR
SETBP1
ETNK1
JAK2
PHF6
DDX41
KMT2C
NRAS
GATA2
PPM1D
BRAF
STAT3
SH2B3
KDM6A
Disease evolution at follow-up
MDS
CMML
AML
Mutations evolution
Same VAF (± 10%)
Clone expansion > 10%
Clone reduction < 10%
Newly acquired mutation
Loss of pre-existing mutation

Figure 2


A
Month 0
CCUS
Normal
karyotype
Month 19
MDS
Normal
karyotype
Month 38
AML
Normal
karyotype
SRSF2
TET2
SETBP1
RUNX1
ETNK1

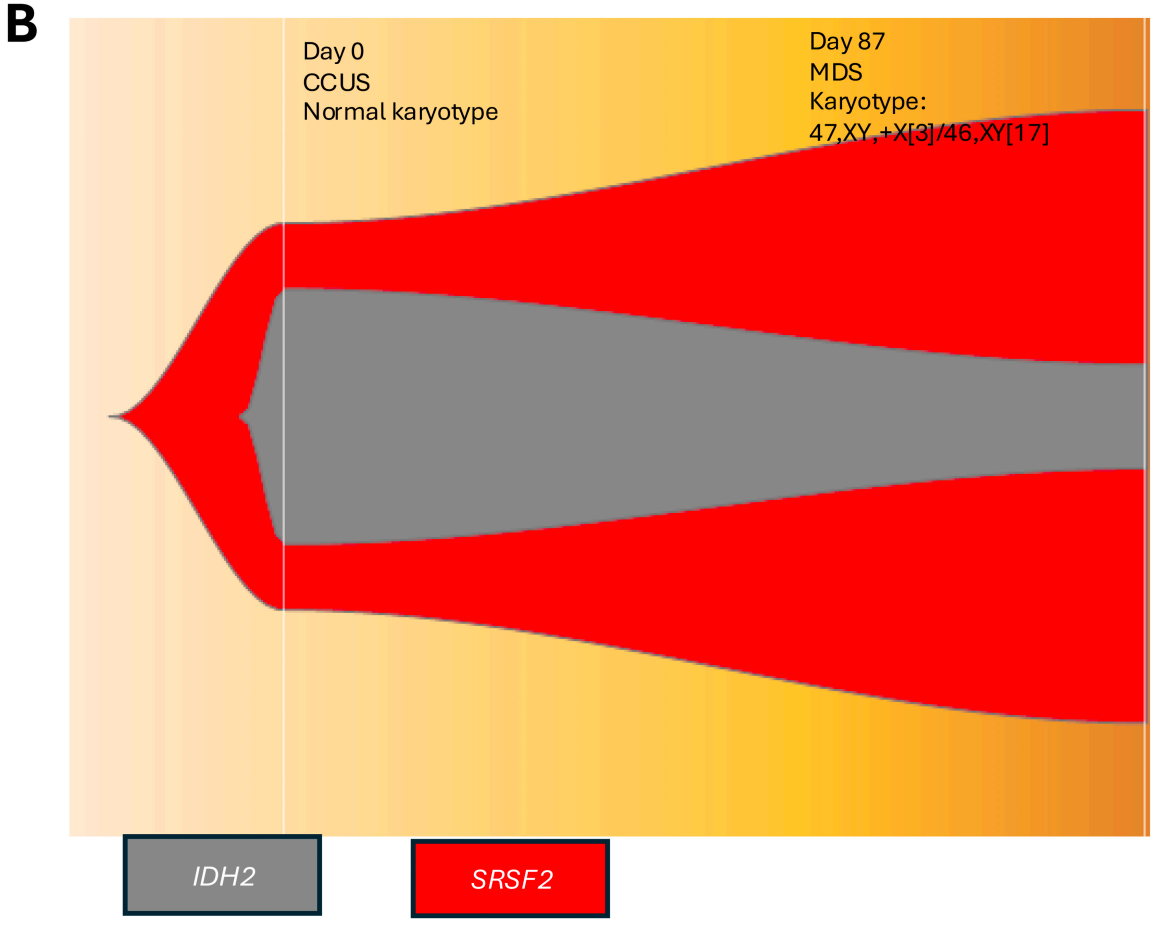

B
Day 0
CCUS
Normal karyotype
Day 87
MDS
Karyotype:
47,XY,+X[3]/46,XY[17]
IDH2
SRSF2

Table s1: Differences in CBC and karyotype between diagnosis and follow-up BMB (section 1); and NGS and scores between diagnosis and follow-up for patients with follow-up NGS (section 2)

Section 1				
	Overall (n= 109)	Did not progress (N=46)	progress (N=63)	p value
Delta cytogenetics				0.42
Normal to abnormal	4 (3.7%)	0 (0.0%)	4 (6.4%)	
Remained normal	74 (69.2%)	33 (71.7%)	41 (66.1)	
Abnormal to normal	4 (3.7%)	3 (6.5%)	1 (1.6%)	
Remained abnormal	25 (23.4%)	9 (19.6%)	16 (25.8%)	
Delta Hg				0.43
Decrease	66 (61.1%)	26 (56.5%)	40 (64.5%)	
Increase	42 (38.9%)	20 (43.5%)	22 (35.5%)	
Delta Plt				0.18
Decrease	80 (74.1%)	31 (67.4%)	49 (79%)	
Increase	28 (25.9%)	15 (32.6%)	13 (21%)	
Delta WBC				0.03*
Decrease	67 (62%)	23 (50.0%)	44 (71%)	
Increase	41 (38%)	23 (50.0%)	18 (29%)	

Section 2				
	Overall (N=74)	Did not progress(N=34)	Progress (N=40)	p value
Pathogenic mutations				0.622
Gain of new pathogenic mutations, n (%)	22 (31.4%)	9 (28.1%)	13 (34.2%)	
Gain and loss of pathogenic mutations, n (%)	1 (1.4%)	1 (3.1%)	0 (0.0%)	
Loss of pathogenic mutations, n (%)	7 (10.0%)	4 (12.5%)	3 (7.9%)	
Same, n (%)	40 (57.1%)	18 (56.2%)	22 (57.9%)	
VAF variation (5%)				0.002*
Expansion, n (%)	37 (53.6%)	10 (31.2%)	27 (73.0%)	
Reduction, n (%)	8 (11.6%)	4 (12.5%)	4 (10.8%)	
Same, n (%)	17 (24.6%)	14 (43.8%)	3 (8.1%)	
Expansion and reduction, n (%)	7 (10.1%)	4 (12.5%)	3 (8.1%)	
Expansion in clones (>5%), n (%)	37 (53.6%)	10 (31.2%)	27 (73.0%)	< 0.001*
VAF variation (10%)				0.154
Expansion, n (%)	13 (18.8%)	3 (9.4%)	10 (27.0%)	
Reduction, n (%)	5 (7.2%)	4 (12.5%)	1 (2.7%)	
Same, n (%)	49 (71.0%	24 (75.0%)	25 (67.6%)	
Expansion and reduction, n (%)	2 (2.9%)	1 (3.1%)	1 (2.7%)	
Expansion in clones (>10%), n (%)	13 (18.8%)	3 (9.4%)	10 (27.0%)	0.0344*

VUS				0.235
New, n (%)	11 (16.2%)	6 (18.8%)	5 (13.9%)	
Loss, n (%)	5 (7.4%)	4 (12.5%)	1 (2.8%)	
Same, n (%)	52 (76.5%)	22 (68.8%)	30 (83.3%)	
Delta CHRS class				0.926
Step up, n (%)	9 (16.1%)	4 (18.2%)	5 (14.7%)	
Step down, n (%)	3 (5.4%)	1 (4.5%)	2 (5.9%)	
Same, n (%)	44 (78.6%)	17 (77.3%)	27 (79.4%)	
Delta CCRS score				0.589
Step up, n (%)	16 (28.6%)	6 (27.3%)	10 (29.4%)	
Step down, n (%)	11 (19.6%)	3 (13.6%)	8 (23.5%)	
Same, n (%)	29 (51.8%)	13 (59.1%)	16 (47.1%)	
Delta CCRS class				0.478
Step up, n (%)	13 (19.1%)	6 (20.0%)	7 (18.4%)	
Step down, n (%)	6 (8.8%)	4 (13.3%)	2 (5.3%)	
Same, n (%)	49 (72.1%)	20 (66.7%)	29 (76.3%)	
Delta CCRS score				0.416
Step up, n (%)	19 (27.9%)	6 (20.0%)	13 (34.2%)	
Step down, n (%)	11 (16.2%)	5 (16.7%)	6 (15.8%)	

Same, n (%)	38 (55.9%)	19 (63.3%)	19 (50.0%)	
Delta IPSSM class				0.054
Step up, n (%)	32 (47.8%)	9 (31.0%)	23 (60.5%)	
Step down, n (%)	3 (4.5%)	2 (6.9%)	1 (2.6%)	
Same, n (%)	32 (47.8%)	18 (62.1%)	14 (36.8%)	
Delta IPSSM score				0.134
Step up, n (%)	50 (74.6%)	19 (65.5%)	31 (81.6%)	
Step down, n (%)	17 (25.4%)	10 (34.5%)	7 (18.4%)	
Delta IPSSR class				0.415
Step up, n (%)	26 (39.4%)	12 (41.4%)	14 (37.8%)	
Step down, n (%)	6 (9.1%)	4 (13.8%)	2 (5.4%)	
Same, n (%)	34 (51.5%)	13 (44.8%)	21 (56.8%)	
Delta IPSSR score				0.113
Step up, n (%)	38 (57.6%)	15 (51.7%)	23 (62.2%)	
Step down, n (%)	13 (19.7%)	9 (31.0%)	4 (10.8%)	
Same, n (%)	15 (22.7%)	5 (17.2%)	10 (27.0%)	

Table s2: abnormal karyotypes

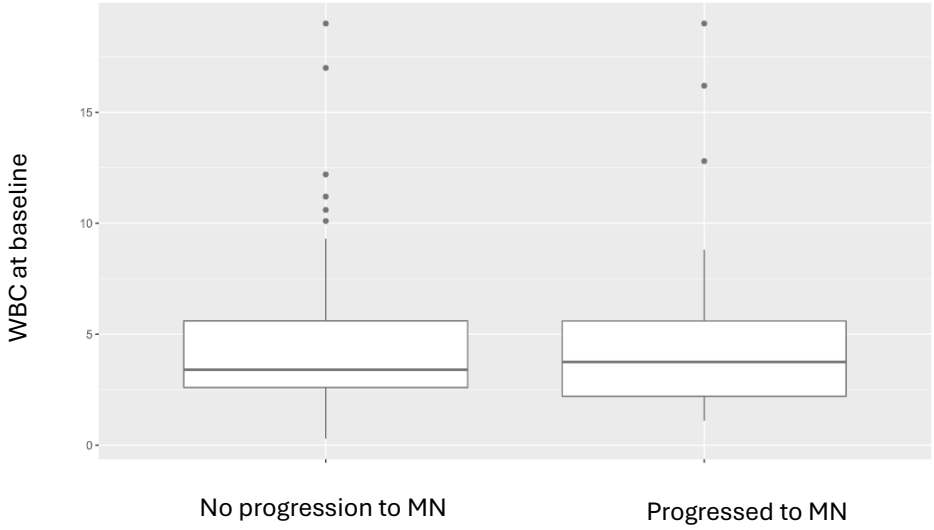
45,X,-Y,del(4)(q21q25)[20]/46,XY[1]
46,XY,del(20)(q11.2q13.3)[10]/56-65,sl,+1,+5,del(7)(q11.2q22),+9,+10,+11,+15,+22[cp2]/46,XY,del(7)(p11.2p15)[2]/46,XY[6]
47,XY,+8[11]/46,XY[9]
47,XX,+12,add(14)(q22)[8]/46,XX[2]
48,XX,+6,+8[4]/46,XX[16]
47,XY,+8[14]/46,XY[6]
46,XX,add(10)(q22)[4]/46,XX[16]
46,XX,del(20)(q11.2q13.3)[20]
46,X,t(X;12)(p11.4;p13.1)[20]
47,XX,+21[18]/46,XX[2
20q del
47,XY,+Y[6]/46,XY[14]
47,XY,+8[20]
47,XY,+8[20]
47,XX,+13[4]/46,XX[16]
46,XX,del(13)(q12q22)[5]/46,XX[15]
46,XY, der(6)t(1;6)(q21; p25)[17]/46,XY[3]
45,X,-Y[3]/46,XY[17]
47,XY,+8[7]/46,XY[13]
47,XX,+9[1]/46,XX[19]
47,XY,+8[16]/46,XY[4] Abnormal
47,XY,+8[15]/46,XY[5]
46,XX,add(9)(p13),del(20)(q11.2q13.3)[19]/46,XX[1]
46,XY,del(9)(q13q22)[20]
47,XY,+8[20]
46,XX,+1,der(1;21)(q10;q10)[20]
46,XX,del(5)(q13q33),t(10;13)(p10;q10)[3]/46,XX[17]
46,XY,del(20)(q11.2q13.1)[5]/46,XY[15]

46,XX,del(20)(q11.2q13.3)[20]
46,XY,del(20)(q11.2q13.1)[20]
46,XY,del(20)(q11.2q13.1)[20]
46,XY,del(20)(q11.2q13.3)[11]/45,X,-Y[4]/46,XY[5]
46,XY,t(1;3)(p36;q21)[3]/46,XY[17]
45,XY,del(5)(q13q33),psu dic(17;20)(p11.2;q11.2)[10]/46,XY[10]
46,XY,del(20)(q11.2q13.3)[12]/46,XY[8]
46,XY,del(20)(q11.2q13.3)[1]/46,XY[19]
47,XY,+8[14]/46,XY[6]
45,X,-Y[20]
45,X,-Y[20]
46,XY,del(20)(q11.2q13.3)[4]/46,XY[16].
46,XY,del(9)(q12q34)[11]/46,XY[9].
46,XX,del(20)(q11.2q13.3)[7]/46,XX[13] 46,XX,del(20)(q11.2q13.3)[7]/46,XX[13]
48,XX,+8,+9[2]/46,XX[18]
46,XX,del(20)(q11.2q13.3)[20]
47,XX,+6[3]/46,XX[17].
47,XXYc[20]
46,XY,del(20)(q11.2q13.3)[17]/46,XY[3]
45,X,-Y[20]
45,X,-Y[20]
46,XX,del(20)(q11.2q13.1)[1]/46,XX[19]
46,XY,del(20)(q11.2q13.1)[18]/46,XY[2]
45,X,-X[17]/46,XX[3]
45,X,-Y[11]/46,XY[9]
46,XY[17] with non-clonal chromosomal rearrangements and breakage
45,X,-Y[7]/46,XY[13]
45,X,-Y[13]/46,XY[7]
45,X,-X[18]/46,XX[2]
46,XX,add(8)(p11.2),add(17)(q11.2)[16]/46,XX[4]
47,XX,inv(3)(p11q29),+r?c[2]/47,XX,+r?c[18]

46,XY,del(13)(q12q22)[3]/46,XY[17]
47,XX,+8[16]/46,XX[4]
45,X,-Y[5]/46,XY[15]
47,XY,+8[20]
del 1q - del 20
46,XY,inv(2)(p11.2q13)?c[20]

Figure s1: Box plot for WBCs at time of CCUS diagnosis and at time of follow up (1st plot for baseline WBCs and 2nd plot for WBCs at follow up). We can see how the median WBC count was higher at baseline among patients who eventually progressed (3.7 vs 3, p=0.6), and at time of progression, the median WBC count was lower among patients who progressed compared to those who did not progress (2.7 vs 3.3, p=0.6)

1. At CCUS diagnosis:



2. At follow up: