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Site-specific biology of tongue base aggressive B-cell lymphomas revealed by molecular subtype classification

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Primary aggressive B-cell lymphomas of the tongue base represent an exceptionally rare extranodal localization, with available evidence limited to isolated case reports focusing on histopathological features and largely lacking molecular characterization or data on contemporary immunochemotherapy outcomes. [1–6]

Meanwhile, integrated molecular classification systems—including probabilistic models such as DLBclass and the clustering framework described by Chapuy et al.—have established that genetic subtype assignment in DLBCL provides clinically meaningful information beyond histology and cell-of-origin classification, informing outcome prediction and therapeutic stratification.[7–10]

Whether this biological framework retains discriminatory capacity in rare extranodal sites remains unexplored.

Here, we report four consecutive patients with primary DLBCL of the tongue base diagnosed at a single center between January and June 2025. The temporal clustering of cases prompted an integrated clinicopathological and molecular characterization, including targeted next-generation sequencing and DLBclass subtype assignment, to explore potential site-specific biological features in this rare presentation. This study was conducted in accordance with the Declaration of Helsinki. Given the retrospective and observational nature of the study and the use of anonymized data, formal ethical approval was not required under applicable institutional regulations.

Primary involvement was defined as the dominant site of disease at presentation, confirmed by clinical evaluation and FDG-PET/CT; patients with secondary tongue base involvement were excluded. Clinicopathological data were retrieved from institutional records. Histological diagnoses were established according to WHO-HAEM5 [5] and ICC criteria. [11]

Staging was performed with FDG-PET/CT and contrast-enhanced CT/MRI of the head and neck; treatment response was assessed per Lugano 2014 criteria.

Targeted DNA sequencing was performed on diagnostic FFPE biopsy specimens using the SOPHiA DDM™ Custom Lymphoma Panel. Variants were filtered at ≥100× coverage and VAF ≥1%. Molecular subtype classification was performed using DLBclass v1.0.0, a probabilistic neural-network classifier assigning cases to one of five genetic subtypes (C1–C5) defined by Chapuy et al. [8,12]

No formal hypothesis testing was performed given the descriptive nature of the study.

Baseline clinicopathological characteristics are summarized in Table 1 and Supplementary Table S1. Median age at diagnosis was 78 years (range 70–87); all patients presented with symptoms related to local tumor growth at the tongue base. At diagnosis, disease localization was confirmed by clinical evaluation and imaging studies, with the tongue base identified as the dominant site of involvement in all cases.

Cell-of-origin classification determined by the Hans algorithm[13], together with fluorescence In Situ Hybridization (FISH) analysis for *MYC*, *BCL2* and *BCL6* rearrangements showed heterogeneous features (Table 1). Two DLBCL patients presented with limited-stage

disease (Ann Arbor stage II), while the remaining two DLBCL patients presented with advanced stage at diagnosis.

We identified 21 coding variants across the cohort of tumor samples. Of these, 9 were synonymous and 12 were non-synonymous alterations, including 7 missense variants and 5 truncating variants (3 nonsense, 1 no-start site, and 1 frameshift), affecting 11 of the 73 genes included in the panel. In addition, 6 copy number alterations were detected (Supplementary Tables S1 and S2).

Molecular classification using DLBclass was successfully performed in all DLBCL cases. Two cases were assigned to the C1 genetic subtype, while two cases were classified as C3. Confidence threshold was high (> 0.7) in all but one case (Supplementary Tables S1 and S3). Molecular subtype attribution was further interpreted in the context of the genomic clustering framework described by Chapuy et al.

A consistent association was observed between DLBCL molecular subtype and pattern of disease dissemination at presentation. All DLBCL patients presented with bulky disease at the primary site, represented by a mass involving the base of the tongue.

However, relevant differences emerged in the extent of disease beyond the primary localization. Both patients classified as C1 DLBCL presented with disease confined to the tongue base, with or without involvement of regional lymph nodes, corresponding to limited-stage disease (Ann Arbor stage II). In contrast, both C3-classified DLBCL patients exhibited additional extranodal and/or nodal localizations at diagnosis, consistent with more disseminated and clinically aggressive disease.

Despite these differences in disease distribution and clinical behavior, Ki-67 proliferative index was comparably high across C1 and C3 cases, indicating that proliferative activity alone did not account for the observed differences in disease dissemination. These findings suggest that molecular subtype–specific biological features may influence the propensity for early dissemination beyond the primary bulky lesion.

Comprehensive molecular profiling revealed heterogeneous but subtype-concordant genomic landscapes across the DLBCL cases. C1-classified tumors displayed a non-GCB profile, and were characterized by alterations involving *BCL6*, the NF-kB regulators *NFKBIE* and *TNFAIP3*(*A20*), and *FBXW7*, a key tumor suppressor that mediates proteasome-dependent degradation of several oncoproteins, including components of the NOTCH2 signaling pathway. C1 tumors also harbored truncating mutations in genes involved in immune evasion (*B2M, CD58*).

In contrast, C3-classified tumors exhibited a GCB phenotype, associated with *BCL2* translocation and recurrent mutations in chromatin modifiers (*KMT2D, CREBBP*) as well as components of the BCR/phosphatidylinositol 3-kinase (PI3K) signaling pathway (*MEF2B, TNFSF14, GNA13*).

Notably, the observed mutational differences paralleled clinical presentation, reinforcing the association between molecular subtype, genomic background, and disease behavior in lymphomas arising from the tongue base.

All DLBCL patients received contemporary first-line immunochemotherapy. Three patients were treated with Polatuzumab vedotin–based immunochemotherapy (R-p-CHP), while one ultra-elderly patient received a reduced-intensity R-COMP regimen.

Both patients classified as C1 DLBCL achieved complete metabolic response after first-line treatment and remained in complete remission at last follow-up.

Among patients classified as C3 DLBCL, clinical outcomes were heterogeneous. One patient experienced rapid disease progression during R-Pola-CHP and died. The remaining C3-classified patient achieved complete metabolic response and was in complete remission at last follow-up.

This series provides among the first integrated clinico-molecular characterization of primary DLBCL arising from the tongue base, an extranodal localization for which biological data are virtually absent. Despite a shared presentation with bulky primary disease, molecular subtype assignment revealed a striking divergence in dissemination patterns: C1 cases presented with limited-stage disease and achieved durable remission, while C3 cases exhibited systemic spread and heterogeneous outcomes. The base of the tongue is a functionally sensitive site where even limited tumor growth prompts early symptoms, making delayed diagnosis an unlikely explanation for these differences. The observed divergence more plausibly reflects intrinsic subtype-specific biological programs governing early dissemination.

These findings align with large-scale genomic analyses demonstrating that C3-associated profiles are enriched for BCL2 rearrangements, chromatin remodeling defects, and alterations affecting immune surveillance — features associated with enhanced dissemination capacity. [12,14] The subtype-concordant mutational landscapes observed in our cohort further support the biological coherence of these classifications beyond conventional nodal presentations.

From a therapeutic standpoint, all patients received contemporary immunochemotherapy, predominantly Pola-R-CHP. Although sample size precludes any efficacy inference, the clinical trajectories are consistent with emerging evidence from the POLARIX biomarker analysis, which identified EZB/C3 and MCD/C5 subtypes as high-risk populations potentially benefiting from Pola-R-CHP over R-CHOP.[15]

Finally, the temporal aggregation of cases within a limited geographic area represents an intriguing observation but must be interpreted with caution. In the absence of formal epidemiological analysis and given the small sample size, no causal inference can be drawn regarding environmental or external etiological factors. Nonetheless, such observations may serve as hypothesis-generating signals warranting further investigation in larger, multicenter studies integrating molecular, clinical, and environmental data.

In conclusion, molecular subtype classification may retain biological and clinical discriminatory capacity even in exceptionally rare extranodal localizations. These findings support the integration of genomic profiling beyond cell-of-origin in the workup of DLBCL regardless of anatomical site, and provide a rationale for multicenter validation in rare extranodal presentations.

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Pt	Age	Diagnosis	COO	Ann Arbor Stage	ECOG PS	IPI	DE status	DH/TH	Mutated genes*	Molecular subtype	Bulky primary mass	First-line treatment	Best response	Outcome
1	78	DLBCL	non-GCB	IIB	0	2	DE	No	*B2M, BCOR, FBXW7*	C1	Yes	Pola-R-CHP	CR	Alive
2	87	DLBCL	non-GCB	IIB	2	2	DE	No (*BCL6*-R)	*CD58, GNA13, NFKBIE, TNFAIP3*	C1	Yes	R-COMP	CR	Alive
3	70	DLBCL	GCB	IVB	1	4	DE	No	*DDX3X, GNA13, BCL2, BRAF*	C3	Yes	Pola-R-CHP	PD	Dead
4	76	DLBCL	GCB	IVB	2	4	DE	No (*BCL2*-R)	*CREBBP, FOXO1, GNA13, KMT2D, MEF2B, TNFRSF4*	C3	Yes	Pola-R-CHP	CR	Alive

Abbreviations:
COO, cell of origin according to Hans algorithm; GCB, germinal center B-cell–like; non-GCB, non-germinal center B-cell–like; DE, double expressor - double expression of MYC (>40%) and BCL2 (>50%) by immunohistochemistry; DH/TH, double-hit/triple-hit; R, rearrangement; CR, complete remission; PD, progressive disease.

Tab 1. Integrated clinic-molecular characteristics and outcomes

Supplementary Table 1. DLBclass probabilistic subtype classification and FISH results

(A) FISH results for MYC, BCL2, and BCL6 rearrangements. (B) Posterior probabilities for each of the five genetic subtypes (C1–C5) generated by DLBclass v1.0.0, together with the predicted cluster assignment and associated confidence score. Confidence >0.7 indicates high-confidence assignment.

A. FISH results

ID	Diagnosis	MYC	BCL2	BCL6
Patient 1	DLBCL	normal	normal	normal
Patient 2	DLBCL	normal	normal	rearranged
Patient 3	DLBCL	normal	normal	normal
Patient 4	DLBCL	normal	rearranged	normal
ID	C1	C2	C3	C4

B. DLBclass subtype classification

ID	C1	C2	C3	C4	C5	Confidence	Predicted Cluster
Patient_1	0.7412	0.0431	0.1016	0.0402	0.074	0.7412	C1
Patient_2	0.7905	0.0108	0.1406	0.04	0.0182	0.7905	C1
Patient_3	0.0539	0.0277	0.5376	0.0903	0.2905	0.5376	C3
Patient_4	0.0018	0.0017	0.9908	0.0037	0.002	0.9908	C3

Supplementary Table 2. Somatic variants, insertions/deletions, and copy number alterations

(A) All coding variants (synonymous and non-synonymous) identified by targeted NGS using the SOPHiA DDM™ Custom Lymphoma Panel, including variant type, genomic coordinates, sequencing depth, variant allele frequency (VAF), predicted cDNA and protein consequence, and ClinVar/COSMIC annotation where available. Variants were filtered at ≥100× coverage and VAF ≥1%. (B) Copy number alterations (CNAs) detected in the cohort.

A. Somatic single-nucleotide variants and insertions/deletions

ID	Gene	Type	Consequence	Chromosome	Position	Depth	VAF (%)	cDNA	Protein	dbSNP	COSMIC
Patient_1	B2M	INDEL	frameshift	15	45003780	5183	13.2	c.43_44del	p.(Leu15Phefs*41)	rs2506037451	COSV62562847
Patient_1	BCOR	SNP	missense	X	39921444	3265	80.4	c.4376A>G	p.(Asn1459Ser)	rs199538037	COSV60698733
Patient_1	FBXW7	SNP	missense	4	153249384	7853	35.1	c.1394G>A	p.(Arg465His)	rs1057519895	COSV55891746
Patient_1	KMT2D	SNP	missense	12	49425854	5318	51	c.12634C>T	p.(Arg4212Trp)	rs760279999	COSV56426340
Patient_1	RHOA	SNP	missense	3	49413009	11628	8.7	c.14G>A	p.(Arg5Gln)	rs1057519953	COSV69041526
Patient_1	TBL1XR1	SNP	synonymous	3	176767818	9801	45.8	c.669A>G	p.(Pro223=)	rs61750378	COSV100763681
Patient_2	CD58	SNP	nonsense	1	117078668	7678	11.5	c.547C>T	p.(Gln183*)		
Patient_2	CD58	INDEL	frameshift	1	117078792	8146	4.8	c.397_422del	p.(Leu133Metfs*26)		
Patient_2	GNA13	SNP	missense	17	63052600	10549	9.3	c.112C>G	p.(Leu38Val)		
Patient_2	NFKBIE	SNP	missense	6	44233304	5815	51.2	c.197C>T	p.(Ala66Val)	rs779952301	
Patient_2	TNFAIP3	INDEL	frameshift	6	138199830	13777	21.3	c.1250_1251del	p.(Lys417Thrfs*11)		
Patient_2	BCL2	SNP	synonymous	18	60985618	8318	11.1	c.282C>T	p.(His94=)		
Patient_2	CREBBP	SNP	synonymous	16	3801729	8395	31.1	c.3777G>A	p.(Gln1259=)	rs368919135	
Patient_2	MAP2K1	SNP	synonymous	15	66777345	7822	47.3	c.711G>A	p.(Gly237=)	rs17586159	COSV61070996
Patient_3	DDX3X	SNP	no-start	X	41193508	7242	59.4	c.3G>A	p.(Met1?)	rs1555950665	
Patient_3	GNA13	INDEL	frameshift	17	63052467	16572	1.5	c.243_244del	p.(Glu82Glyfs*19)	rs771380711	COSV71475024
Patient_3	PIM1	SNP	synonymous	6	37139026	15313	30	c.639G>A	p.(Arg213=)	rs775749144	
Patient_4	BCL2	SNP	missense	18	60985803	9089	20.1	c.97G>C	p.(Gly33Arg)		COSV105209582
Patient_4	BCL2	SNP	missense	18	60985883	7316	18.6	c.17G>C	p.(Arg6Thr)		COSV61374585
Patient_4	BRAF	SNP	missense	7	140453154	5248	14.1	c.1781A>G	p.(Asp594Gly)	rs121913338	COSV56065695
Patient_4	CREBBP	SNP	missense	16	3781923	3579	45	c.4744A>G	p.(Ser1582Gly)	rs1363001625	
Patient_4	CREBBP	SNP	missense	16	3831230	6823	43.7	c.1651C>A	p.(Leu551Ile)	rs61753381	COSV52134580
Patient_4	FOXO1	SNP	missense	13	41240294	5907	19.5	c.56G>C	p.(Arg19Pro)	rs2137951108	COSV65426408
Patient_4	GNA13	SNP	missense	17	63052440	6464	30.2	c.271_272delinsCT	p.(Asn91Leu)		

ID	Gene	Type	Consequence	Chromosome	Position	Depth	VAF (%)	cDNA	Protein	dbSNP	COSMIC
Patient_4	GNA13	SNP	missense	17	63052641	9065	36.6	c.71A>G	p.(Glu24Gly)		
Patient_4	KMT2D	SNP	nonsense	12	49438067	7823	31.1	c.5104C>T	p.(Arg1702*)	rs886043414	COSV56407907
Patient_4	MEF2B	SNP	missense	19	19260079	6070	25.4	c.214T>A	p.(Tyr72Asn)		COSV50761560
Patient_4	TNFRSF14	SNP	missense	1	2489201	4905	22.5	c.106G>A	p.(Ala36Thr)	rs745993971	

B. Copy number alterations

ID	Diagnosis	Gene(s)	Chromosome	Consequence	Copy Number
Patient_1	DLBCL	CD79B	chr17	Gain	3.3
Patient_1	DLBCL	INPP5D	chr2	Gain	3.3
Patient_1	DLBCL	SF3B1	chr2	Gain	3.3
Patient_1	DLBCL	BCL2	chr18	Gain	4.8
Patient_2	DLBCL	TNFAIP3	chr6	Gain	3.4

Supplementary Table 3. DLBclass gene sample matrix (GSM)

Binary matrix of somatic alterations across the 73 genes included in the DLBclass classifier, used as input for probabilistic subtype assignment. Values reflect the number of detected alterations (0 = absent; 1 = one alteration; 2 = two or more alterations). N/A indicates genes not covered by the sequencing panel. DLBclass-predicted subtypes are indicated in the column headers.

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
ACTB	N/A	N/A	N/A	N/A
ATP2A2	N/A	N/A	N/A	N/A
B2M	2	0	0	0
BCL10	N/A	N/A	N/A	N/A
BCL11A	N/A	N/A	N/A	N/A
BCL2	0	1	0	2
BCL6	0	0	0	0
BCL7A	N/A	N/A	N/A	N/A
BRAF	0	0	0	2
BTG1	0	0	0	0
BTG2	N/A	N/A	N/A	N/A
CARD11	0	0	0	0
CCDC27	N/A	N/A	N/A	N/A
CD274	N/A	N/A	N/A	N/A
CD58	0	2	0	0
CD70	N/A	N/A	N/A	N/A
CD79B	0	0	0	0
CD83	N/A	N/A	N/A	N/A
CREBBP	0	1	0	2
CRIP1	N/A	N/A	N/A	N/A
CXCR4	0	0	0	0
DTX1	N/A	N/A	N/A	N/A
DUSP2	N/A	N/A	N/A	N/A
EBF1	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
EEF1A1	N/A	N/A	N/A	N/A
EP300	0	0	0	0
ETS1	N/A	N/A	N/A	N/A
ETV6	0	0	0	0
EZH2	0	0	0	0
FADD	N/A	N/A	N/A	N/A
FAS	N/A	N/A	N/A	N/A
GNA13	0	2	2	2
GNAI2	N/A	N/A	N/A	N/A
GRHPR	N/A	N/A	N/A	N/A
HIST1H1B	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
HIST1H1C	N/A	N/A	N/A	N/A
HIST1H1D	N/A	N/A	N/A	N/A
HIST1H1E	0	0	0	0
HIST1H2AC	N/A	N/A	N/A	N/A
HIST1H2AM	N/A	N/A	N/A	N/A
HIST1H2BC	N/A	N/A	N/A	N/A
HLA.A	N/A	N/A	N/A	N/A
HLA.B	N/A	N/A	N/A	N/A
HLA.C	N/A	N/A	N/A	N/A
HVCN1	N/A	N/A	N/A	N/A
IGLL5	N/A	N/A	N/A	N/A
IKZF3	N/A	N/A	N/A	N/A
IRF2BP2	N/A	N/A	N/A	N/A
IRF4	0	0	0	0

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
IRF8	N/A	N/A	N/A	N/A
KLHL6	N/A	N/A	N/A	N/A
KMT2D	2	0	0	2
KRAS	0	0	0	0
LTB	N/A	N/A	N/A	N/A
LYN	N/A	N/A	N/A	N/A
MAP2K1	0	1	0	0
MEF2B	0	1	0	2
MEF2C	N/A	N/A	N/A	N/A
METAP1D	N/A	N/A	N/A	N/A
MYD88	0	0	0	0
MYD88.L265P	0	0	0	0
MYD88.OTHER	0	0	0	0
NFKBIA	N/A	N/A	N/A	N/A
NFKBIE	0	2	0	0
NOTCH2	0	0	0	0
OSBPL10	N/A	N/A	N/A	N/A
PABPC1	N/A	N/A	N/A	N/A
PIM1	0	0	2	0
PIM2	N/A	N/A	N/A	N/A
POU2AF1	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
POU2F2	N/A	N/A	N/A	N/A
PRDM1	0	0	0	0
PTEN	0	0	0	0
PTPN6	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
RAC2	N/A	N/A	N/A	N/A
RHOA	2	0	0	0
SESN3	N/A	N/A	N/A	N/A
SF3B1	0	0	0	0
SGK1	N/A	N/A	N/A	N/A
SMG7	N/A	N/A	N/A	N/A
SOCS1	0	0	0	0
SPEN	N/A	N/A	N/A	N/A
STAT3	0	0	0	0
SV.BCL2	0	0	0	3
SV.BCL6	0	3	0	0
SV.MYC	0	0	0	0
TBL1XR1	1	0	0	0
TET2	0	0	0	0
TMEM30A	N/A	N/A	N/A	N/A
TMSB4X	N/A	N/A	N/A	N/A
TNFAIP3	0	2	0	0
TNFRSF14	0	0	0	2
TNIP1	N/A	N/A	N/A	N/A
TOX	N/A	N/A	N/A	N/A
TP53	0	0	0	0
TUBGCP5	N/A	N/A	N/A	N/A
UBE2A	N/A	N/A	N/A	N/A
X10Q23.31.DEL	0	0	0	0
X11P.AMP	N/A	N/A	N/A	N/A
X11Q.AMP	N/A	N/A	N/A	N/A
X11Q23.3.AMP	N/A	N/A	N/A	N/A
X12P.AMP	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
X12P13.2.DEL	0	0	0	0
X12Q.AMP	N/A	N/A	N/A	N/A
X13Q.AMP	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
X13Q14.2.DEL	N/A	N/A	N/A	N/A
X13Q34.DEL	N/A	N/A	N/A	N/A
X14Q32.31.DEL	N/A	N/A	N/A	N/A
X15Q15.3.DEL	N/A	N/A	N/A	N/A
X16Q12.1.DEL	N/A	N/A	N/A	N/A
X17P.DEL	N/A	N/A	N/A	N/A
X17Q24.3.AMP	N/A	N/A	N/A	N/A
X17Q25.1.DEL	N/A	N/A	N/A	N/A
X18P.AMP	N/A	N/A	N/A	N/A
X18Q.AMP	N/A	N/A	N/A	N/A
X18Q21.32.AMP	2	0	0	0
X18Q22.2.AMP	N/A	N/A	N/A	N/A
X18Q23.DEL	N/A	N/A	N/A	N/A
X19P13.2.DEL	N/A	N/A	N/A	N/A
X19P13.3.DEL	N/A	N/A	N/A	N/A
X19Q.AMP	N/A	N/A	N/A	N/A
X19Q13.32.DEL	N/A	N/A	N/A	N/A
X19Q13.42.AMP	N/A	N/A	N/A	N/A
X1P13.1.DEL	N/A	N/A	N/A	N/A
X1P31.1.DEL	N/A	N/A	N/A	N/A
X1P36.11.DEL	0	0	0	0
X1P36.32.DEL	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
X1Q.AMP	N/A	N/A	N/A	N/A
X1Q23.3.AMP	N/A	N/A	N/A	N/A
X1Q32.1.AMP	N/A	N/A	N/A	N/A
X1Q42.12.DEL	N/A	N/A	N/A	N/A
X21Q.AMP	N/A	N/A	N/A	N/A
X2P16.1.AMP	N/A	N/A	N/A	N/A
X2Q22.2.DEL	N/A	N/A	N/A	N/A
X3P.AMP	N/A	N/A	N/A	N/A
X3P21.31.DEL	N/A	N/A	N/A	N/A
X3Q.AMP	N/A	N/A	N/A	N/A
X3Q28.AMP	N/A	N/A	N/A	N/A
X3Q28.DEL	N/A	N/A	N/A	N/A
X4Q21.22.DEL	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
X4Q35.1.DEL	N/A	N/A	N/A	N/A
X5P.AMP	N/A	N/A	N/A	N/A
X5Q.AMP	N/A	N/A	N/A	N/A
X6P.AMP	N/A	N/A	N/A	N/A
X6P21.1.AMP	N/A	N/A	N/A	N/A
X6P21.33.DEL	0	0	0	0
X6Q.DEL	N/A	N/A	N/A	N/A
X6Q14.1.DEL	N/A	N/A	N/A	N/A
X6Q21.DEL	N/A	N/A	N/A	N/A
X7P.AMP	N/A	N/A	N/A	N/A
X7Q.AMP	N/A	N/A	N/A	N/A
X7Q22.1.AMP	N/A	N/A	N/A	N/A

Gene	Patient 1 (C1)	Patient 2 (C1)	Patient 3 (C3)	Patient 4 (C3)
X8Q12.1.DEL	N/A	N/A	N/A	N/A
X8Q24.22.AMP	N/A	N/A	N/A	N/A
X9P21.3.DEL	N/A	N/A	N/A	N/A
X9P24.1.AMP	N/A	N/A	N/A	N/A
X9Q.AMP	N/A	N/A	N/A	N/A
X9Q21.13.DEL	N/A	N/A	N/A	N/A
YY1	N/A	N/A	N/A	N/A
ZC3H12A	N/A	N/A	N/A	N/A
ZEB2	N/A	N/A	N/A	N/A
ZFP36L1	N/A	N/A	N/A	N/A
ZNF423	N/A	N/A	N/A	N/A