Cumulative incidence and factors associated with subsequent myeloid neoplasms in patients with nodal T-follicular helper cell lymphomas

T-follicular helper cell lymphomas (TFHL) are now understood to arise upon a background of clonal hematopoiesis (CH).[1] We and others have shown that divergent evolution of stem cells harboring CH mutations can result in both TFHL and myeloid neoplasms (MN) within the same patient.[1,2] As TFHL are typically treated with chemotherapy, there is a risk that selective treatment pressure allows CH clones to expand and evolve to MN. The frequency at which this occurs, as well as factors that may be associated with this phenomenon, have not been established. We assessed a large cohort of patients with TFHL to understand the incidence of this phenomenon and associated risks.

Patients with nodal TFHL who were sequentially treated at Memorial Sloan Kettering between 2012 and 2023 were identified. All cases underwent pathology review for diagnostic confirmation. TFHL were defined using consensus World Health Organization (WHO) and International Consensus Classification (ICC) pathology criteria, with at least two positive TFH markers to assign a TFH phenotype.[3,4] Cases with concomitant TFHL and MN at diagnosis, or cases in which the MN preceded the TFHL,[5] were excluded (N=12, described elsewhere[5]). In addition, patients with less than six months of follow up (who were still alive) were omitted (8 patients). Relevant patient- and treatment-related factors were collected. When available, next-generation sequencing results from the Memorial Sloan Kettering Integrated Mutation Profiling of Actionable Cancer Targets heme panel (MSK-IMPACT) were noted.[6] The cumulative incidence of developing a MN was calculated using the competing risks method treating death as a competing risk. Univariate analyses were performed to evaluate risks associated with developing a MN. All research was conducted under an institutional review board-approved retrospective research protocol.

A total of 208 patients were identified. Baseline characteristics are shown in Table 1. The median age of all patients was 69 years. Except for 8 patients who were managed by observation, all patients received upfront anthracycline-based induction (96%). Ninety-six patients (46%) received an etoposide-containing induction regimen, such as CHOEP (cyclophosphamide, doxorubicin, vincristine, etoposide, prednisone). Eighty-one patients received autologous stem cell transplant (ASCT) in first remission (39%). The median follow up time among survivors was 2.7 years (Interquartile Range [IQR]: 1.6-4.4). In terms of follow-up surveillance, patients were observed as per National Comprehensive Cancer Network (NCCN) recommendations, with clinical examinations and laboratory studies every three months for two years and then at least every six to 12 months, and surveillance imaging every six months for two years and then annually up to five years.

Among these 208 patients, 10 developed a MN, resulting in a cumulative incidence at two and five years of 1.5% and 5.3%, respectively (Figure 1). The median time to developing a MN was 2.2 years. The specific MN were acute myeloid leukemia (AML) (N=4), myelodysplastic syndrome (MDS) (N=4), chronic myelomonocytic leukemia (N=1), and chronic neutrophilic leukemia (N=1). All patients had received prior anthracycline-based therapy, 8 had received multiple treatments for TFHL, and 5 had received prior ASCT. No patients had received prior allogeneic transplant for lymphoma, though one patient proceeded with allogeneic transplant for AML. Except for 2 patients, all of these patients had undergone a baseline bone marrow (BM) biopsy at the time of TFHL diagnosis. Details of these patients are shown in *Online Supplementary Table S1*. Specifically, of the 8 patients with a baseline BM at the time of TFHL diagnosis, none had evidence of the MN and each demonstrated mature trilineage hematopoiesis without dysplasia. Patient 6 (an 81-year old female) was noted to have occasional small hypolobated megakaryocytes, and patient 10 (a 74-year old male) was noted to have a mildly hyperplastic myeloid lineage without dysplasia, both of which are consistent with expected secondary BM findings in TFHL.[7] Peripheral hematologic parameters at the time of TFHL diagnosis were largely unrevealing, with most patients having a normal white blood cell count, hemoglobin, and platelet count (*Online Supplementary Table S1*). Notable exceptions include patient 5 (a 72-year old male) with hemoglobin 7.3, though this patient had a concomitant diagnosis of multiple myeloma with 20% bone marrow involvement, and patient 9 (a 58-year old male) with hemoglobin of 6.4, though this patient had diffuse splenic involvement and positive direct Coombs testing, suggesting a paraneoplastic autoimmune hemolytic anemia associated with the lymphoma.

The results of mutational profiling for these patients are shown in Table 2, comparing the genetic profiles of each TFHL with the subsequent MN. Of particular note are the shared mutations and changes in variant allele frequency (VAF) within the MN. Apart from one patient without sequencing results, all patients had *TET2* mutations in the TFHL, and all patients with sequencing of both the lymphoma and MN shared at least one *TET2* mutation. Besides *TET2*, the only other shared mutation was *DNMT3A*. Notably,

among these cases, none of the TFHL harbored mutations in *TP53, SF3B1, SRSF2, ZRSR2, RUNX1, JAK2, IDH1*, or *PPMD1*, known CH mutations previously identified as conferring increased cumulative risk of MN.[8,9] Two patients had baseline genetic studies on peripheral blood or BM samples at the time of lymphoma diagnosis. Patient 1 (a 73-year old male) underwent BM testing with an unmatched 30-gene panel which detected no mutations (at that time, the BM was only minimally involved with lymphoma at <5%). Over eight years later, this patient developed MDS, which shared a *TET2* C1396R mutation with the preceding lymphoma. Of note, three years prior to the MDS diagnosis (and five years after the lymphoma diagnosis), unmatched MSK-IMPACT testing on a BM sample detected mutations in *U2AF1* Q157P (VAF: 6.6%) and *ASXL1* H782Lfs*2 (VAF: 36.5%); at that time, there was no definitive evidence of MDS. Three years later, upon formal MDS diagnosis, the same *U2AF1* and *ASXL1* mutations were detected, with a notably large increase in the VAF of the *U2AF1* variant to 31.7%. The second patient, patient 5 (a 72-year old male), underwent peripheral blood testing at the time of lymphoma diagnosis with an unmatched 49-gene panel. At the time of lymphoma diagnosis, peripheral blood testing detected mutations in *TET2* p.Y1128* (VAF: 5.1%) and *DNMT3A* p.R736C (VAF: 7.2%). Only a minute abnormal T-cell population was detected in the blood at this time (0.9% of total white cells), indicating that these mutations were present in the myeloid compartment. These same mutations were concomitantly present in the lymphoma at diagnosis at high VAF (*TET2* VAF: 45.8%, *DNMT3A* VAF: 47.8%) and were eventually detected in the MN at the time of MN diagnosis over two years later, again at high VAF (TET2 VAF: 35.8%, DNMT3A VAF: 39.6%). Of note,

Table 1. Cohort characteristics.

Variable, N	Total cohort N=208	No MN N=198	MN N=10
Diagnosis			
TFH, NOS	25	24	1
TFH, AITL	182	173	9
TFH, follicular	1	1	0
Age at lymphoma diagnosis, years, median (IQR)	69 (61-74)	68 (61-74)	72 (63-74)
Sex			
Male	112	105	7
Female	96	93	3
Stage			
I/II	17	17	0
III/IV	191	181	10
Induction			
CHOP	63	58	4
Etoposide-based	96	91	5
BV-CHP	30	30	0
Other	11	11	1
Observation	8	8	0
ASCT			
Yes	81	76	5
No	127	122	5
Number of lines of therapy, median (range)	2 (0-11)	2 (0-11)	3 (1-5)
Etoposide use (any time)	146	139	7
Other agents used			
azacitidine	18	16	2
EZH inhibitor	14	12	2
HDAC inhibitor	80	74	6
JAK inhibitor	21	19	2
lenalidomide	22	20	2
PI3K inhibitor	58	54	4

Other: includes investigational regimens, including lenalidomide-CHOEP, azacitidine-CHOP, duvelisib-CHOP, and BV-C(H)EP. The one patient in the MN group with Other as induction received lenalidomide-CHOEP. AITL: angioimmunoblastic T-cell lymphoma; ASCT: autologous stem cell transplant; BV-CHP: brentuximab vedotin, cyclophosphamide, doxorubicin, prednisone; CHOP: cyclophosphamide, doxorubicin, vincristine, prednisone; IQR: interquartile range; MN: myeloid neoplasm; N: number; NOS: not otherwise specified; TFH: T-follicular helper cell.

this patient's only intervening therapy was CHOP. Data for these patients are shown in *Online Supplementary Figure S1*. None of the other 8 patients had baseline sequencing performed on the blood and/or bone marrow at the time of lymphoma diagnosis, primarily due to normal bone marrow examination at baseline, and the fact that there had been no perceived need to sequence normal tissue.

To evaluate whether certain patient- and treatment-related factors were significantly associated with developing a MN, the effects of age, sex, receipt of ASCT, and receipt of etoposide were measured (*Online Supplementary Table S2*). On univariate analyses, no factor was significantly associated with subsequent MN (age >60 years, Hazard Ratio [HR]: 1.2, 95% Confidence Interval [CI]: 0.3-5.8; male sex, HR: 1.8, 95%CI: 0.5-6.8; CHOP induction *vs*. etoposide-containing, HR: 0.6, 95%CI: 0.2-21.1; ASCT, HR: 1.5, 95%CI: 0.4-5.2; etoposide use, HR: 0.9, 95%CI: 0.2-3.2).

Divergent clonal evolution of CH can lead to both TFHL and MN within the same patient. Here we reviewed a large cohort of patients with TFHL, showing that this is a relatively infrequent though not trivial event, with a cumulative incidence of 5.3% at five years. This is slightly less than or similar to estimates in patients with high-risk CH or clonal cytopenias of undetermined significance (and no lymphoma), though higher than estimates in patients with low-risk CH.[8,10] Given that CH is a well-established risk factor for developing MN,[10] and that cancer therapies

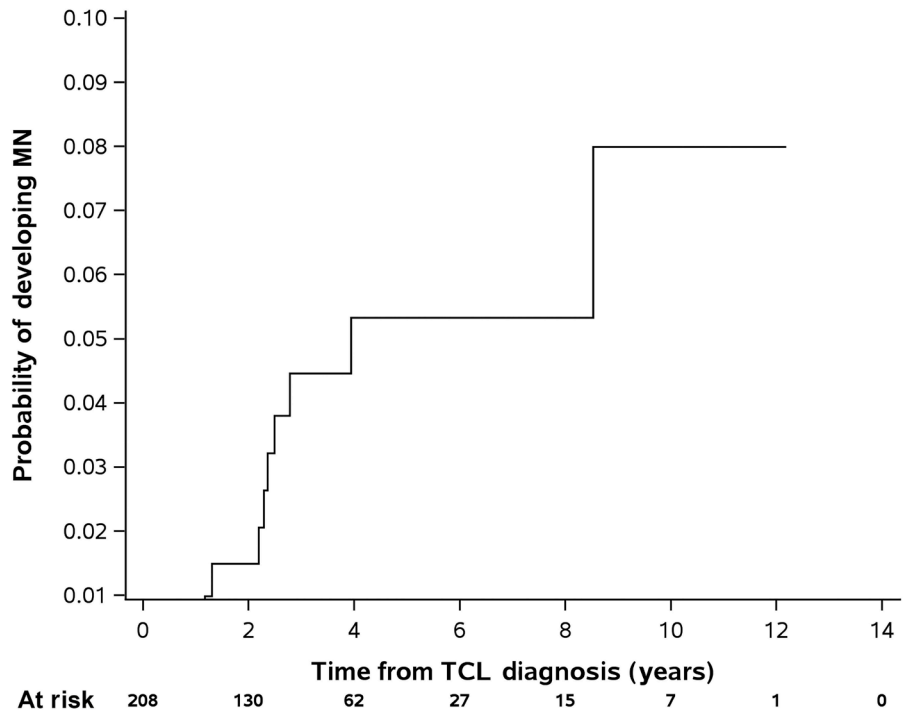


Figure 1. Cumulative incidence of myeloid neoplasm. Cumulative incidence plot of developing a myeloid neoplasm (MN) over time from T-cell lymphoma (TCL) diagnosis. Cumulative incidence calculated using the competing risks method, treating death as a competing risk.

Table 2. Genomic profiles of patients with T-follicular helper cell lymphoma and myeloid neoplasm.

Patient Age, years/sex	TFH genetics				Myeloid neoplasm genetics				
	Tissue source	Gene	Mutation	VAF	Dx	Tissue source	Gene	Mutation	VAF
73/M	Lymph node	*TET2*° *RHOA*	C1396R° G17V	0.171° 0.050	WHO: MDS-LB ICC: MDS, NOS	Bone marrow: MDS, +flow abnormal T-cell population (1.0% of WBC)	TET2° U2AF1 RUNX1 ASXL1 PTPN11	C1396R° Q157P T92Afs*31 H782Lfs*2 D61G	0.160[a] 0.317 0.223 0.311 0.021
72/M	Lymph node	*TET2*° *TET2*° *EPHA5*	C484*° L757Sfs*56° D136N	0.250° 0.230° 0.097	WHO: MDS-IB1 → AML, MR; ICC: MDS-EB → AML, NOS progression from MDS	Bone marrow: AML, no TFH lymphoma	*TET2*° *TET2*° *WT1*	C484* L757Sfs*56° S189*	0.449[b] 0.455[b] 0.408
63/F	Bone marrow, flow sorted abnormal T cells from bone marrow	*DNMT3A*° *TET2*° *TET2*° *NFE2* *ROBO1* *ARID1B* *DDX3X*	R771*° L1899Sfs*9° C1289Y° T318A G904R S914N V526A	0.510° 0.446° 0.506° 0.341 0.516 0.349 0.234	WHO: AML, MR ICC: AML with MR gene mutation	Bone marrow: AML and TFH lymphoma; genetics are flow sorted CD34+ myeloid blasts	*DNMT3A*° *TET2*° *TET2*° *RUNX1* *DNMT3A*	R771*° L1899Sfs*9° C1289Y° A329Sfs*271 C861Y	0.400[c] 0.320[c] 0.481[a] 0.621 0.406
74/M	Lymph node	*TET2*° *TET2* *TET2*° *SETBP1* *VAV1* *CUX1*	S577Pfs*3° C1289F F1300V L1421P° Y174C E555L	0.373° 0.243 0.072° 0.300 0.334 0.296	WHO/ICC: CMML-1	Bone marrow: CMML, +flow abnormal T-cell population (0.036% of WBC)	*SRSF2* *TET2*° *TET2*° *FBXO11* *CHEK2*	P95H S577Pfs*3° F1300V° Y692H D134G	0.479 0.426[a] 0.468[b] 0.437 0.443

Continued on following page.

Patient Age, years/sex	TFH genetics				Myeloid neoplasm genetics				
	Tissue source	Gene	Mutation	VAF	Dx	Tissue source	Gene	Mutation	VAF
72/M	Bone marrow, flow sorted abnormal T cells from bone marrow	*IDH2* *RHOA* *TET2°* *DNMT3A°* *TET2°* *KMT2B* *ATR*	R172K G17V Q810*° R736C° Y1128*° Q757* Q757*	0.406 0.459 0.463° 0.478° 0.458° 0.248 0.206	WHO: MDS-IB2 ICC: MDS/AML	Bone marrow: MDS, no TFH lymphoma	*RUNX1* *TET2°* *DNMT3A°* *TET2°* *BTG1* *BRCA2* *SETD5* *SPEN* *RET* *CEBPA* *MET* *MET* *NBN* *VAV2*	D198N Q810*° R736C° Y1128*° K29* A2306S Y987F P3008Q Q70K H260N P210Q R592K H328N G106*	0.042 0.440[a] 0.396[a] 0.358[a] 0.024 0.013 0.027 0.022 0.017 0.024 0.542 0.599 0.015 0.015
81/F	Lymph node	*RHOA* *TET2°* *TET2°* *TET2^°*	G17V N752Kfs*60° K1500Qfs*72° N598Ifs*3	0.195 0.180 0.194 0.007	WHO: AML, MR ICC: AML, NOS	Bone marrow: AML, +flow abnormal T-cell population (0.1% of WBC), genetics are flow sorted CD34+ myeloid blasts	*ATR* *TET2* *TET2^°* *TET2^°*	Q631* N598Ifs*3 N752Kfs*60° K1500Qfs*72°	0.697 0.717[b] 0.008[c] 0.008[c]
55/M	Lymph node	*TET2*	E1082*	0.408*	WHO: MDS, LB ICC: MDS, NOS	Not performed.	Not performed.	Not performed.	Not performed.
71/F	Lymph node	*IDH2* *RHOA* *TET2°* *DNMT3A°* *TET2* *HLA-A* *CCND3* *MYCN* *HDAC4* *HGF*	R172K G17V W1198*° Y724*° A379Vfs*6 X25_splice R271Pfs*53 S369G G362A S145G	0.146 0.147 0.452 0.364° 0.115 0.076 0.134 0.363 0.154 0.090	WHO/ICC: CNL	Bone marrow: CNL, +flow abnormal T-cell population (0.0043% of WBC), also 5% plasma cell neoplasm by CD138	*DNMT3A°* *JAK2* *TET2°*	Y724* V617F W1198*°	0.493[b] 0.412 0.792[b]
58/M	Not performed.	Not performed.	Not performed.	Not performed.	WHO: AML, MR ICC: AML with MR gene mutation	Not performed.	Not performed.	Not performed.	Not performed.
74/M	Lymph node	*IDH2* *RHOA* *TET2°* *TET2°* *DNMT3A°* *NCOR2* *SETD1B*	R172S G17V Q778*° D1384N° V296M° A989T E612*	0.063 0.070 0.420° 0.277° 0.410° 0.067 0.054	WHO: MDS-LB ICC: MDS, NOS	Bone marrow: MDS, +flow abnormal T-cell population (0.014% of WBC)	*TET2°* *TET2°* *DNMT3A°*	Q778*° D1384N° V296M°	0.515[a] 0.490[b] 0.520[b]

°Shared mutations between the TFH lymphoma and myeloid neoplasm. [a]Changes in variant allele frequency (VAF) within 10%. [b]Changes in VAF greater than 10%. [c]Changes in VAF less than 10%. ^These mutations are below the technical sensitivity of the performed assay though they were detected upon manual review. Dx: diagnosis; AML: acute myeloid leukemia; CNL: chronic neutrophilic leukemia; EB: excess blasts; F: female; IB: increased blasts; ICC: International Consensus Criteria; LB: low blasts; M: male; MDS: myelodysplastic syndrome; MR: MDS-related; NOS: not otherwise specified; TFH: T-follicular helper call; VAF: variant allele frequency; WBC: white blood cell; WHO: World Health Organization.

exert selective pressure on premalignant clones that can increase the risk of MN,[11-13] one may expect the incidence among patients with TFHL to be higher.

Several factors may explain the incidence observed in this cohort despite the uniformity of CH in TFHL and use of chemotherapies. First, there is a lack of mutations considered high-risk for progression to a MN in TFH lymphomas. For example, in a large cohort of healthy participants from the UK Biobank (N=438,890),[8] single mutations in *TET2* or *DNMT3A*, which are the most frequent in TFHL, carried a

low risk of subsequent MN (HR: 0.189, 95%CI: 0.147-0.243) compared to mutations in splicing factors, such as *SRSF2, SF3B1* and *ZRSR2* (HR: 13.77, 95%CI: 10.31-18.42) and AML-like genes, such as *FLT3* and *RUNX1* (HR: 9.26, 95%CI: 5.29-16.18). These high-risk mutations are generally not observed in TFHL.[13] Similarly, in a cohort of over 300 patients with Hodgkin lymphoma undergoing ASCT, while patients with *TP53* and/or *PPMD1* CH mutations at the time of ASCT had a very high incidence of MN (8-year cumulative incidence of 63.2%, 95%CI: 17.5-88.6), those with other mutations had a risk near to that observed in our cohort (8-year cumulative incidence of 5.8%, 95%CI: 1.0-17.2).[9] While *TP53* mutations are observed in TFHL, they are not very common and are more frequently encountered in non-TFH histologies, such as peripheral T-cell lymphoma, not otherwise specified.[14,15] Additional reasons for the incidence observed here are a high competing risk of death from lymphoma and the use of allogeneic transplant for relapsed lymphoma, which could, in theory, eliminate pre-existing recipient CH clones. In our cohort, 30 patients (14%) received an allogeneic transplant. Our series is limited in its retrospective nature and the low number of events, potentially hindering our ability to detect any significant factors associated with developing a MN. In addition, as mutational profiling had not been performed on all patients, we were not able to compare the mutational profiles of those patients who did and those who did not develop a MN. In particular, not being able to fully characterize the molecular makeup of each myeloid component at the time of lymphoma diagnosis hinders the ability to assess whether certain risk profiles (such as those calculable through the clonal hematopoiesis risk calculator[8]) hold true in this population. Still, as all patients in this cohort had TFHL, which are generally genomically similar,[14] we would expect that established genetic risk factors, such as the presence of high-risk mutations, high VAF within such mutations, and multiple high-risk mutations, to be applicable to patients with lymphoma.[8] As the patients in our series generally did not have these features, it remains somewhat elusive as to why MN developed. Moreover, recent work has shown that patients can actually develop MN before lymphoma or have a concomitant presentation in the absence of treatment pressures.[6] Collectively, our data and these findings appear to suggest that yet unrealized intrinsic genetic susceptibilities in addition to therapy-related selective pressure may be driving clonal evolution. Prospective studies that involve sequential sequencing to evaluate changes in VAF and capture additive mutational events may further elucidate this phenomenon.

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Contributions

KSL and RS identified and annotated the patients, analyzed the data, and drafted the manuscript; TC, KK, VI, BS and NG performed data and statistical analysis; MY, OCE, AD and WX reviewed pathology and molecular cases; WX and RS conceived and designed the study, identified and annotated the patients, supervised the data analysis, and revised the manuscript. All authors contributed to patient care, and reviewed and approved the final version of the manuscript for publication.

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Data-sharing statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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