CASE REPORT

Peripheral T-cell lymphoma mimicking classic Hodgkin's lymphoma in a patient presenting with fevers of unknown origin

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SUMMARY

A 52-year-old man presented to our hospital for further workup of fever of unknown origin after an extensive workup at an outside hospital had failed to reveal a diagnosis. At the outside hospital, he underwent excisional biopsy of the left supraclavicular lymph node, which showed non-necrotising granulomatous changes, and a bone marrow biopsy which showed a normocellular marrow. He was discharged without a diagnosis with recommendations to present to a tertiary hospital. During his admission, his hospital course was complicated by new direct hyperbilirubinaemia and eosinophilia, prompting liver and skin biopsies which showed CD30+ and CD3+ cells. He subsequently underwent left axillary lymph node biopsy, which was reported as 'classic Hodgkin's lymphoma'. With worsening lab values and T cells noted on liver and skin biopsies, excisional lymph node biopsy was sent to the National Institute of Health, where it was confirmed patient had peripheral T cell lymphoma.

BACKGROUND

Over the last several decades, classification and identification of non-Hodgkin's lymphoma (NHL) has evolved dramatically. Despite these advances in molecular medicine, many of the subclasses continued to remain a heterogeneous group unable to be separated based on immunophenotypic or genetic testing. In the 1950s, NHL was identified based on pathology cell size and shape,¹ and in the 1970s through the work of Lukes, Collins and Lennart, further subclassified into T and B cells.^{2,3} It was not until 1994 through the International Lymphoma Study Group, who proposed the REAL (revised European-American Classification of Lymphoid Neoplasms) classification which characterised T and B cell lymphomas based on major histological, immunological and genetic characteristics.⁴

Despite these advances, peripheral T cell lymphoma (PTCL) has been unable to be completely classified, and despite what is known about these subtypes such as anaplastic large cell lymphoma (ALCL), angioimmunoblastic T cell lymphoma (AITL) and others, the largest subgroup of PTCL remains the 'not otherwise specified' group. This demonstrates that there is much to be learnt regarding this large heterogeneous group.⁵ Over

the last few decades, several clinical studies have attempted to identify the clinical and pathologic features and prognostic importance of PTCL, not otherwise specified (PTCL-NOS), but the numbers of cases in these studies were generally small and findings found to be inconsistent or unconfirmed. We report a confirmed case of PTCL-NOS and the challenges that were presented in confirming this diagnosis due to its heterogeneous presentation based on the concomitant confounding presence of Reed-Sternberg-like cells.

CASE PRESENTATION

This patient is a 52-year-old Caucasian man who presented to our hospital for further workup of fever of unknown origin (FUO) after an extensive workup at an outside hospital had failed to reveal a diagnosis. He reported fevers and chills of 2 months duration, along with diffuse lymphadenopathy. At the outside hospital, he underwent an excisional biopsy of the left supraclavicular lymph node which showed non-necrotising granulomatous changes. Along with an elevated ACE, he was started on treatment for presumed sarcoidosis with prednisone 60 mg; however, after 3 weeks with no clinical response and persistent relapsing and remitting fevers, treatment was discontinued. He subsequently underwent an extensive infectious disease workup including lumbar puncture, blood and urine cultures, MRI of the brain and lumbar spine, *Cryptococcus*, *Bartonella*, *Ehrlichia* and *Babesia*, all of which were negative. Eventually he had a bone marrow biopsy, which showed a normocellular marrow with trilineage hematopoiesis without granulomatous lesions, with no overt evidence of acute leukaemia or high-grade myelodysplastic or myeloproliferative disorders. He was discharged without a diagnosis with recommendations to present to a tertiary hospital for further evaluation.

On admission to our hospital, a complete blood count showed a WBC count of 6000 (normal: 4000–10 000/mm³) with lymphopaenia, with an absolute lymphocyte count: 330/mm³ (normal: 1000–3200/mm³). An extensive infectious disease workup was repeated with several additional studies including Cytomegalovirus (CMV), Epstein-Barr virus (EBV), HIV, histoplasma, *Brucella*, *Blastomyces*, arboviruses, *Coxiella burnetii* and human T lymphotropic virus type I, all of which

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Figure 1 The excisional biopsy of left axillary lymph node shows diffuse effacement of normal nodal architecture and extracapsular infiltration of lymphoid cells.

were negative except for low-level CMV viraemia (viral load of 218 copies/mL), which was attributed to reactivation in the setting of illness. Endobronchial ultrasound with biopsy was performed to evaluate for malignant cells in the setting of his diffuse lymphadenopathy, which only showed few aggregates of histiocytes suggestive of granulomas. The patient's hospital course was complicated by development of relapsing and remitting fevers, maculopapular rash, severe pruritus, pancytopenia

Figure 2 There are scattered large atypical lymphoid cells with morphological features of Reed-Sternberg cells within a polymorphous background consisting of many lymphocytes intermixed with histiocytes, eosinophils and plasma cells.

requiring transfusions, decreased oral intake and hospital-induced delirium. Given this history of suspected sarcoidosis at the outside hospital, rheumatologic workup was also repeated with new labs including complement factors C3 and C4 and vasculitis factors cANCA and pANCA, which were all within normal limits. Due to low clinical suspicion for bacterial infection or sarcoidosis, both antibiotics and steroids were avoided.

After the first cycle of ABVD, the patient's direct hyperbilirubinaemia and eosinophilia continued to trend up, with the total bilirubin peaking at 33.6 mg/dL (normal: 0.0–0.2 mg/dL) and eosinophilia at 51.0% (reference range: 0%–8%). A repeat CMV DNA PCR showed 10570 copies/mL for which he was started on intravenous ganciclovir. With worsening lab values and clinical picture in the setting of T cells noted on liver and skin biopsies, the excisional lymph node biopsy was sent to the National Institutes of Health (NIH) for further analysis, which noted the presence of typical T cell markers CD4, CD5, CD7 and CD8, Reed-Sternberg-like cells with clonal T cell markers CD3, CD15 and CD30 and flow cytometry showing a small population (3%–5%) of T cells with dim CD3, confirming the diagnosis of PTCL with Reed-Sternberg-like cells (figure 1 and figure 2). CD21 showed absent dendritic cell meshwork.

Based on the new pathological diagnosis, cycle two of chemotherapy was changed to CHOEP (cyclophosphamide, doxorubicin, etoposide, vincristine and prednisone). Following his first cycle of CHOEP, the patient's total bilirubin (3.3 mg/dL) and eosinophilia (0.0%) improved, and he had significant clinical improvement, with decreased fatigue and increased oral intake. His CMV viraemia worsened, and his viral load peaked at 26256 copies/mL, thus requiring extended treatment with intravenous ganciclovir. On date of discharge, the CMV viral load was trending down, he was afebrile and was otherwise stable from a lab and clinical standpoint. He was discharged home with intravenous ganciclovir and home physical therapy. He was referred to a local oncologist to complete his chemotherapy treatments.

INVESTIGATIONS

Despite significant advancement in molecular medicine over the last several decades, PTCL-NOS exhibits heterogeneity, making it challenging to be distinguished based on immunophenotypic, histological assessment or genetic testing.⁵ There are patterns that subdivide PTCL into several categories with PTCL-NOS being the most frequent (25.9%), AITL making up the next largest (18.5%) and ALCL making up the third largest (12%).⁶ The International T Cell Lymphoma study published in 2008 reviewed 340 cases of PTCL-NOS in an attempt to better understand this undefined class and found that this class is still largely heterogeneous with much to be discovered.⁶ Overall survival and failure-free survival with PTCL-NOS at 10–15 years was 10%, with the overall age of diagnosis around 60, more predominant in males (male-to-female ratio of 1.9:1). A majority of patients (69%) had advanced staged disease (Stage III/IV) by the time of diagnosis. Only nodal disease was present in 38%, nodal and extranodal disease in 49% and extranodal disease only in 13%. The most common extranodal sites included skin (16%), subcutaneous tissue (6%) and lungs (8%).^{6,7}

DIFFERENTIAL DIAGNOSIS

In this case, the differential diagnosis was extremely broad given the patient's nonspecific symptoms. Lymphoma was initially lower on the differential on presentation to our hospital, due to non-specific findings noted on prior excisional biopsy of left supraclavicular lymph node and bone marrow biopsy performed

Figure 3 By immunohistochemical studies, the large Reed-Sternberg-like cells are MUM1-positive with variable reactivity for CD30, CD15, perforin and very weak CD45. The CD4/CD45-positive atypical lymphoid cells are variable in size with variably diminished CD3, CD5 and CD7 and a subset having perforin. The lymphoma cells are negative for CD20, PAX-5, CD10, BCL-6, PD-1, ALK- 1, HHV8 and EBER. The CD21 immunostatin shows absence of dendritic cell meshwork.

at the outside hospital. The initial aetiology on presentation was felt to be infectious versus rheumatologic. However, an extensive infectious workup was negative and the patient had been unresponsive to steroids at the outside hospital. After repeat rheumatology workup was negative, malignancy then moved higher on the differential. Hospital course was complicated by the development of direct hyperbilirubinaemia and eosinophilia prompting liver and skin biopsies, which noted the presence of T-cell clones, in particular CD3 and CD30. For these reasons, excisional left axillary lymph node biopsy was performed, which suggested ‘classic Hodgkin’s lymphoma, likely nodular sclerosis’ and patient was transferred to our oncology service where he was started on Cycle 1 of ABVD chemotherapy regimen. Because an atypical T-cell clone was identified in liver and skin biopsies, his pathology was sent out for consultation to the NIH. The final diagnosis returned as PTCL-NOS with Reed-Sternberg-like cells (figure 3).

TREATMENT

The patient received one cycle of ABVD for treatment of Hodgkin’s lymphoma. After clarification of his diagnosis, his chemotherapy regimen was switched to CHOEP for treatment of aggressive PTCL. The patient was discharged after completion of first cycle of CHOEP with clinical improvement and was scheduled to complete his treatment with an oncologist in his local area.

OUTCOME AND FOLLOW-UP

After treatment with one-half of a cycle of ABVD and then a full cycle of CHOEP chemotherapy and with treatment for CMV

viraemia, the patient showed clinical improvement. For the first time in over 1 month, his fevers resolved, his bilirubin improved and the eosinophilia resolved. He was discharged with home health to complete extended treatment course of intravenous ganciclovir and to complete his chemotherapy under the care of a local oncologist.

DISCUSSION

We have reported a case of PTCL-NOS, with Reed-Sternberg-like cells, demonstrating the evolution of presentation and the challenges presented in confirming the diagnosis. As discussed, PTCL-NOS, due to its inability to be distinguished based on immunophenotypic, major histological or genetic testing, remains heterogeneous in nature with confounding presentations.

According to Gomez-Gelvez and Smith, the presence of Reed-Sternberg-like cells in PTCL presents as one of the most challenging diagnosis to make, due to, in particular, the mixed inflammatory background, consisting of eosinophils, plasma cells, histiocytes and small lymphocytes without cytologic atypia found also in classic Hodgkin’s lymphoma. They noted that Reed-Sternberg-like cells were positive for CD30 and CD15 and also showed expression of CD3 and CD4, but negative for B cell markers (CD20, PAX-5, CD10 and BCL-6).⁸ This was consistent with the excisional lymph node biopsy in our case report as well as in liver and skin biopsies (figures 3, 4 and 5). Per their review,

Figure 4 Flow cytometry analysis on left axillary lymph node, peripheral blood and bone marrow samples. The flow cytometry analysis all detected a population of abnormal T-cells that express dim CD3, dim CD4 without CD8.

Figure 5 The flow cytometry analysis for TCR V-beta repertoire reveals T-cell clone expressing TCR-V-beta 9 in peripheral blood and lymph node samples. TCR, T cell receptor.

Table 1 Aetiologies mimicking peripheral T-cell lymphoma

Author	Year	Age	Gender	Aetiology
Colon-Otero <i>et al</i> ¹⁰	1986	44	F	Hodgkin's
Diez-Martin <i>et al</i> ¹¹	1991	69	M	Liver failure
Diez-Martin <i>et al</i> ¹¹	1991	33	M	Liver failure
Schwartz <i>et al</i> ¹²	1998	73	M	Hepatic disease
Falagas <i>et al</i> ¹⁴	2007	28	M	Cellulitis
Fragou <i>et al</i> ¹⁵	2009	23	F	Stroke
Kanner <i>et al</i> ¹⁶	2011	63	M	Ulcer
Cohen <i>et al</i> ¹³	2013	59	M	Cirrhosis
Fox <i>et al</i> ¹⁷	2014	53	M	PTLD
Coats <i>et al</i> ¹⁸	2016	32	F	FUO
Butler <i>et al</i>	2017	52	M	FUO, Hodgkin's

FUO, fevers of unknown origin; PTLD, Post-transplant lymphoproliferative disease.

they reported that these findings are more commonly associated with AITL, whereas rarely associated with PTCL-NOS. In fact, this diagnostic dilemma was noted in a case series of 11 patients where Reed-Sternberg-like cells of T-cell derivation were present.⁹

PTCL is a diagnosis that is often confused or mistaken for other entities. Aetiologies in the literature reported to mimic PTCL include Hodgkin's disease,¹⁰ cirrhosis and other hepatic diseases,^{11–13} cellulitis,¹⁴ stroke,¹⁵ ulcer,¹⁶ post-transplant lymphoproliferative disease (PTLD)¹⁷ and FUO¹⁸ (table 1). In addition, there are reports of PTCL having been diagnosed after initially mimicking alternate diagnoses, including Mycobacterium,¹⁹ EBV,^{20–22} natural killer lymphoma,²³ tuberculosis²⁴ and syphilis²⁵ (table 2). With Hodgkin's lymphoma in particular, Gaulard *et al* first noted the utility of combining histology and immunohistochemistry to reveal a clonal T-cell population to distinguish the aforementioned entities.²⁶ Further research has shown that the presence of an aberrant CD3(-dim) CD4(+) population by flow cytometry is displayed in PTCL of follicular helper type.²⁷ Using said flow cytometry and clonal PCR can even help distinguish the rare entity of a Hodgkin's lymphoma with expressing a T-cell antigen.^{28 29} This is of utmost importance as by eliminating mimicking aetiologies, more accurate diagnoses are obtained thereby leading to the appropriate treatment for the patient.

Interestingly, during initial presentation, the patient did not present with eosinophilia, but it later developed throughout his hospital stay and only dramatically trended down with initiation of CHOEP. It is well known that eosinophilia can be associated with either lymphomas or leukaemias, with T-cell-derived lymphomas being more commonly associated; however, in most of these case reports, malignancy did not develop until 30–42 months after the onset of hypereosinophilia and only rarely

Table 2 Peripheral T-cell lymphoma mimicking another aetiology

Author	Year	Age	Gender	Aetiology
Chen <i>et al</i> ¹⁹	2008	46	M	Mycobacterium
Chen <i>et al</i> ¹⁹	2008	63	F	Mycobacterium
Pan <i>et al</i> ²³	2011	55	M	NK lymphoma
Tamura <i>et al</i> ²⁰	2013	32	M	EBV
Epskamp <i>et al</i> ²¹	2015	50	M	EBV
Ranjan <i>et al</i> ²⁴	2015	21	M	TB
Yamashita <i>et al</i> ²⁵	2015	40	M	Syphilis
Zhang <i>et al</i> ²²	2016	25	M	EBV

EBV, Epstein-Barr virus; NK, natural killer; TB, tuberculosis.

Learning points

- ▶ The largest subgroup of peripheral T cell lymphoma remains the 'not otherwise specified group' (PTCL-NOS) due to its inability to be separated based on immunophenotypic or genetic testing.
- ▶ The presence of Reed-Sternberg-like cells in PTCL can confound the diagnosis, as they can appear similar to classic Hodgkin's lymphoma. Therefore, PTCL should always be considered in the differential diagnosis.
- ▶ Reed-Sternberg-like cells are typically positive for CD30 and CD15 and show expression for CD3 and CD4, but negative for B cell markers (CD20, PAX-5, CD10 and BCL-6). These markers are more commonly associated with angioimmunoblastic T cell lymphoma, however, can present in PTCL-NOS.
- ▶ PTCL is a diagnosis that is often confused or mistaken for other entities and therefore should be considered in the differential diagnosis for cases where aetiology does not fit or non-responsive to treatment, in particular Epstein-Barr virus, Mycobacterium, natural killer lymphoma, tuberculosis, syphilis, rhabdomyolysis, liver disease or cirrhosis, cellulitis, stroke, ulcer, post-transplant lymphoproliferative disease or fevers of unknown origin.

associated with eosinophilia presenting after the development of suspected malignancy, such as in our case.^{30 31}

While immunophenotypic and genetic testing has advanced in recent years, there is still much to be discovered in further classifying PTCL-NOS into more discrete categories, as our case demonstrates.

Contributors All authors: conception and design, acquisition of data or analysis and interpretation of data; drafting the article or revising it critically for important intellectual content; final approval of the version published; agreement to be accountable for the article and to ensure that all questions regarding the accuracy or integrity are investigated and resolved.

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