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Case Report

Peripheral T-Cell Lymphoma Presenting with Pulmonary Cavitations: A Rare Manifestation and Clinical Challenge

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Introduction:

Peripheral T-cell lymphoma (PTCL) is defined as a diverse group of aggressive lymphomas developing from mature-stage white blood cells called T-cells and natural killer (NK) cells. PTCL is classified as a subtype of non-Hodgkin's lymphoma (NHL), affecting two types of white blood cells: B-cells and T-cells. PTCL specifically affects T-cells resulting in abnormal growth and T-cells develop.

Primary T-cell lymphoma is a rare subtype of non-Hodgkin lymphoma, and when it involves the lungs, it can lead to several respiratory complications. Primary pulmonary lymphoma represents only 0.5-1% of all primary pulmonary malignancies, less than 1% of all the cases of non-Hodgkin's lymphoma (NHL) and 3-4% of all the extranodal manifestations of NHL [1]. Lung involvement is characterized by fever, cough, dyspnea, and bilateral pulmonary nodules, usually diagnosed by transbronchial biopsy or computed tomography (CT) guided needle biopsy and pathology.

In this report, we describe an uncommon case of a patient with diagnosis of PTCL who presented to our emergency room without respiratory symptoms and was found with symptoms and imaging slightly different from those described in previous literature.

Case Presentation:

We present a 48-year-old female with past medical history of epilepsy, achalasia with dilatation surgery, asthma, active smoker (14 pack years) and erosive GERD who presents to emergency room due to episodes of fever, hearing loss of left ear with associated tinnitus. Patient was admitted due to left sided coalesent

osteomastoiditis with associated extension into the intracranial compartment with associated subperiosteal inflammatory changes/phlegmon and superimposed cerebritis and/or pyogenic abscess within the left temporal lobe found on imaging. Incidental findings in CT Thorax without contrast demonstrated two lobulated large cavitary lesions with thick walls involving bilateral apices with internal cystic components, measuring approximately 8.4 cm AP x 7.6 cm transverse on the right and on the left 6.8 cm AP x 6.0 cm transverse (Figure 1 and 2).

Abdominal pelvic CT was remarkable for multiple bilateral renal cysts (Figure 3). Physical examination findings without tachycardia, normotensive, afebrile and adequate saturation at room air. Laboratories showed leukocytosis (15.1), normo-normo anemia (9.7), and thrombocytosis (900). Renal function test is normal (Table 1). Inflammatory markers including ESR and CRP were elevated and complement levels (C3 and C4) decreased (Table 2). Urinalysis without proteinuria or hematuria. Vasculitis markers including C-ANCA, P-ANCA, Anti-MPO and Anti-PR3 is negative. Rheumatoid factor is elevated however nonspecific. Infectious workup (Table 3) pertinent for positive Aspergillosis AG.

Since no specific diagnosis was found, bronchoscopy was scheduled for tissue diagnosis which was negative for infection and malignancy. Afterwards, fine needle biopsywas obtained from right sided cavitary lesion which finally provided diagnosis of Peripheral T- Cell Lymphoma. Immunohistochemistry showed positive CD2, CD3 and CD56 [Table 4]. PTCLs demonstrate a frequent loss of CD5 and CD7; CD2 and CD3 are the most common conventional markers expressed. Small studies have show that biopathologic factors associated with the worst outcome in PTC are high Ki-67 expression, Epstein-Barr virus (EBV) positivity, and CD15 staining [5]. EBV was found positive in our patient which has been proposed as a negative prognosticator in PTCL.

Stage IV peripheral T-cell lymphoma was confirmed per Ann Arbor Staging System (Figure 5). Patient was started on COEP(cyclophosphamide, vincristine, etoposide and prednisone) therapy(no anthracycline due to newly diagnosed heart failure with ejection fraction of 30- 35%) with G-CSF. Patient follow up noted without adequate response to treatment later with progression of the disease and other complications who later decided to proceed with hospice care.

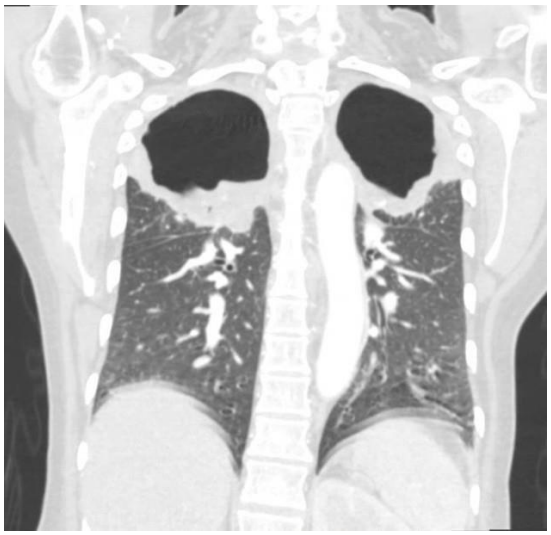

Figure 1: Coronal view of Chest CT showing two lobulated large perihilar cavitary lesions with thick walls involving bilateral apiceswith internal cysticcomponents.

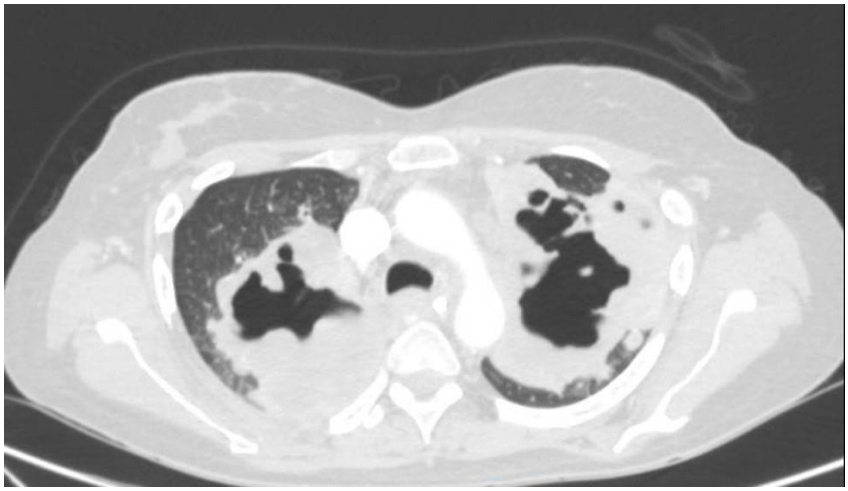

Figure 2: Axial view of Chest CT showing two lobulated largeperihilar cavitary lesions with thick walls involving bilateral apices with internal cystic components.

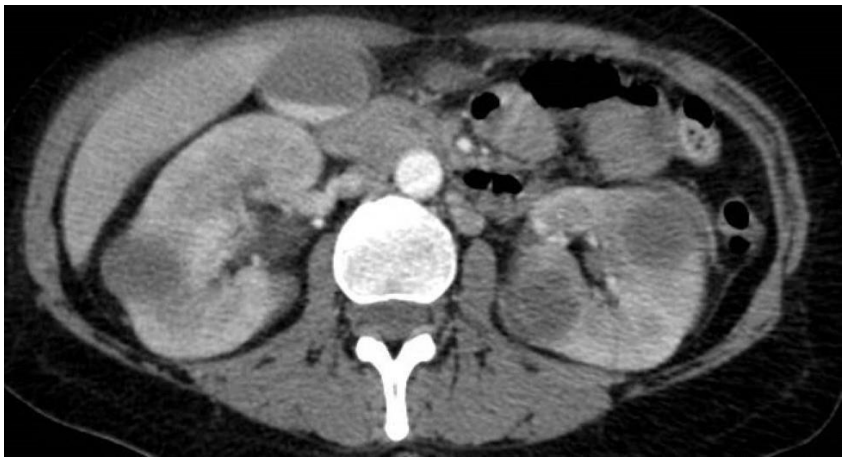

Figure 3: Multiple heterogeneous hypovascular lesions are seen scattered throughout both kidneys. The largest one is seen in the interpolar region of the left kidney, measuring approximately 3.3 cm in largest diameter. It appears to show extracapsular extension of the lesion into the adjacent pararenal fat.

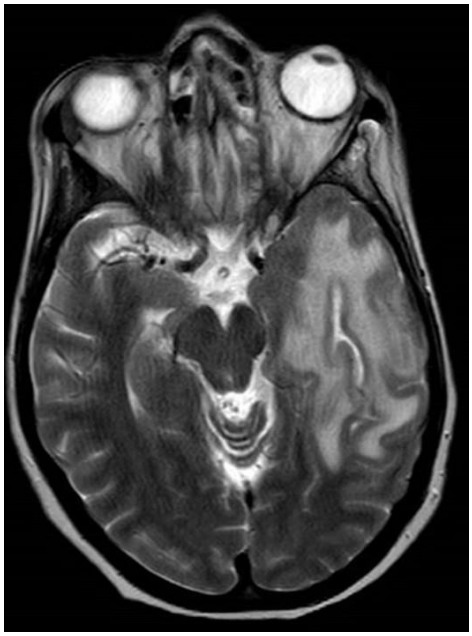

Figure 4: Multi septated T1 hypointense and T2 hyperintense peripherally enhancing collection with associated true restricted diffusion within the left temporal lobe with extensive associated vasogenic edema slightly and associated contrast enhancement involving the meninges, subarachnoid spaces, and portions of the cortex suggestive of seven post encephalitis.

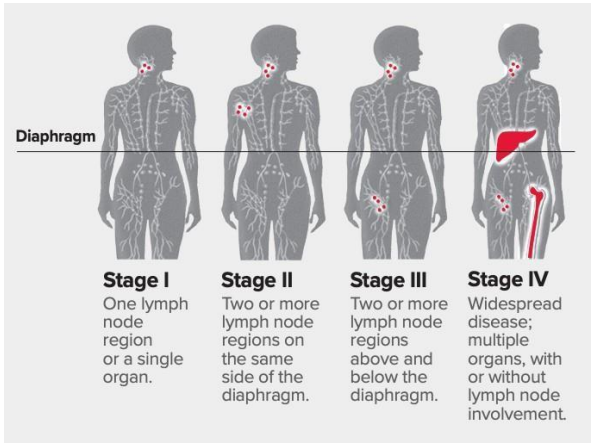

Figure 5: Ann Arbor Staging System for Lymphoma Staging

Tables:

Table 1: Initial Laboratories

CBC		CMP	ABGs
WBC	15.1 [H]	Na: 139	pH: 7.29
Hgb	9.7	K: 4.7	pCO2: 45
Hct	29.8	Cl: 102	pO2: 54
Plts	900 [H]	BUN: 28 [H]	HCO3: 21.5
		Cr: 0.63	FiO2: 21%
		Glu: 59	AST: 16
		Ca: 10.1 [H]	ALT: 28
		Alb: 3.5	AlkPhos: 84

Table 2: Rheumatologic Workup in Serum

Rheumatic factor: 158 [H]	Anti-MPO: <0.2 (NL: 0.0-1.0)	IgM:149
dsDNA: Negative	Anti-PR3: 0.9 (NL: 0.0-1.0)	IgG: 1443
ANA: Negative	Cryoglobulins: positive	IgG1: 633
c-ANCA: Negative	CD4 levels: 40 [L]	IgG2: 141 [L]
p- ANCA: Negative	CD8 levels: <4 [L]	IgG3: 20.7
Procalcitonin: 0.10 [H]	IgA: 479 [H]	SSA-Ab: Negative
CRP: 319 [H]	IgE: 702.0	SSB-Ab: Negative
ESR: 119 [H]		

Table 3: Infectious Workup

Blood culture anaerobic: Negative	MRSA nares: Negative
Blood culture aerobic: Negative	Sputum Culture: Negative
Urine culture: Negative	Urine Histoplasmosis AG: Negative
Tuberculin skin test: Negative	Aspergillosis AG: Positive
AFB: Negative 3/3	D-(1,3)-glucan: Negative
HIV: Negative	HAB Ab: Reactive
RPR: Non-reactive	HAV IgM/ HBsAg/HBsAb/ HBc IgG Ab/ HCV Ab: Negative
EBVIgG: Positive	Cryptococcal Antigen: Negative
HTLV-I/II Antibody: Negative	

Table 4: Immunohistochemistry

CD2: Positive	AE1/AE3: Negative
CD3: Positive	CAM5.2: Negative
CD56: Positive	Melan-A: Negative
Granzyme-B: Positive	WT-1: Negative
Chromogranin A: Negative	S100: Negative
ALK: Negative	PAS-F: Negative
Synaptophysin: Negative	CD99: Negative
CK 5/6: Negative	

Discussion:

Peripheral T-cell lymphomas (PTCLs) are a group of rare and often aggressive hematologic tumor developing from mature T cells and natural killer (NK) cells. They account for approximately 10 percent of nonHodgkin's lymphoma cases. PTCLs generally affect people older than 60 years, although they can occur at any time during adulthood and diagnosed more often in men than in women [2]. Usual presentation includes peripheral lymphadenopathy (87%) but can involve extranodal disease present in 62% including skin and gastrointestinal tract; lungs and central nervous system are less common [3].

Primary T-cell lymphoma is a rare subtype of non-Hodgkin lymphoma, and when it involves the lungs, it can lead to several respiratory complications. Pulmonary involvement in T-cell lymphoma is uncommon but can manifest in various ways, including cavitary lung lesions. This effect is seen secondary to tissue necrosis due to the aggressive infiltration of malignant cells [4]. Many mechanisms may contribute to cavitation in primary T-cell lymphoma, including direct invasion where the lymphoma can infiltrate lung tissue, causing necrosis and cavitation. Another mechanism may be secondary infection due to immunosuppression due to lymphoma or chemotherapy which can predispose patients to opportunistic infections such as fungal infections like Aspergillosis, or bacterial infections like tuberculosis, which can also lead to cavitation. Also, vascular involvement by tumors can compromise blood vessels, leading to infarction resulting in cavitary lesions.

It's prognosis for primary T-cell lymphoma with pulmonary involvement depends on the extent of disease, response to treatment, and presence of secondary complications like infections. Aggressive treatment is required, but outcomes can be variable depending on the lymphoma subtype and disease burden. For most of the subtypes of T-cell PPL, first line therapy is typically cyclophosphamide, hydroxydaunorubicin, oncovin, and prednisone (CHOP)-based chemotherapy [2]. Adult T-cell lymphoma has a poor prognosis because of life-threatening complications, such as infections and tumor progression.

The differential diagnosis for lung cavitations is broad, and etiologies can be classified based on the clinical history and the patient's symptoms. They can be caused by several conditions, including infections, malignancy, and trauma.

Our case shows a unique feature of PTCL as the disease presented as multiple lung cavitations with thick walls and internal cystic components. This radiologic appearance can also be present in such benign conditions as abscess, lung infarction, c-ANCA (anti- neutrophil cytoplasmic antibody)-associated granulomatous vasculitis and carcinomas. The thick wall and irregular inner margin are more frequently seen in malignant lesions, as was noted in our case.

Unfortunately, our patient had acatastrophic presentation with diffuse involvement and poor response to standard therapy including chemotherapy.

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