CASE REPORT

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Concomitant Lennert-type peripheral T-cell lymphoma with presumed smear-negative pulmonary tuberculosis based on clinical and radiological findings: a case report

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ABSTRACT

Lennert lymphoma (LL), a rare lymphoepithelioid variant of peripheral T-cell lymphoma (PTCL), is characterized by an indolent clinical course compared to other PTCL subtypes. The coexistence of LL and pulmonary tuberculosis (TB), particularly smear-negative TB, is extremely rare and presents unique diagnostic and therapeutic challenges. We report a case of a 76-year-old man with underlying type 2 diabetes mellitus who presented with progressive left inguinal swelling, night sweats, weight loss, and loss of appetite over four months. Examination revealed multiple bilateral cervical and inguinal lymphadenopathy. Laboratory tests showed anaemia, leukocytosis, and raised inflammatory markers. Histopathological examination of the inguinal lymph node demonstrated total effacement of the lymphoid architecture by malignant lymphoid cells, admixed with epithelioid histiocytes and Reed-Sternberg-like cells. Immunohistochemistry confirmed LL with a $CD3^+/CD4^+/CD8^-$ immunophenotype. Initial staging computed tomography (CT) revealed multi-organ lymphadenopathy and tree-in-bud nodules in the right upper lobe, suggestive of pulmonary TB. Repeated sputum examinations for acid-fast bacilli were negative. The patient was empirically treated for presumptive smear-negative pulmonary TB and LL. He was treated with cyclophosphamide, epirubicin, vincristine, etoposide, and prednisone chemotherapy and HRZE anti-tuberculous therapy. Empirical anti-tuberculous therapy showed clinical response radiologically; however, despite initial clinical improvement, diagnostic complexities and treatment interactions between lymphoma and TB.

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Introduction

Peripheral T-cell Lymphoma (PTCL) is an uncommon type of aggressive non-Hodgkin lymphoma. Lennert lymphoma (LL), a rare variant of PTCL, offers a better prognosis than other PTCL variants. Pulmonary tuberculosis (TB) is associated with immunodeficiency and B-cell lymphoma, which can cause T-cell immunodeficiency (1), and CD4+ T-cells are vital for defense against pulmonary TB (2). Previous studies suggest TB increases the risk of lymphoma, particularly B-cell non-Hodgkin lymphoma; however, a definite causal relationship cannot be established between T-cell lymphoma and TB (3). Reactivation of pulmonary TB has also been reported mainly in Hodgkin lymphoma, rarely in T-cell lymphoma (4). To the best of our knowledge, concurrent LL and pulmonary TB have never been reported in the literature. We report a case of concurrent LL, a rare subtype of nodal PTCL, and smear-negative pulmonary TB in a 76-year-old man with known type 2 diabetes.

Case Report

A 76-year-old male with underlying type 2 diabetes mellitus, under regular follow-up at a health clinic, presented with a large left inguinal swelling of four months' duration, associated with night sweats, loss of appetite, and weight loss. There was no history of vomiting, jaundice, or fever. Other than that, there were no significant comorbidities or prior surgical procedures. He was a social drinker and did not smoke cigarettes or take any recreational drugs in the past. Informed consent was obtained from the patient's spouse for the anonymous use and publication of clinical and imaging data.

On examination, he was alert, pale, afebrile, and without jaundice. The abdomen was soft and non-tender; the liver and spleen were not palpable. His vital signs were unremarkable: blood pressure 100/60 mmHg; pulse rate 80 beats per minute, regular and of good volume; respiratory rate 18 breaths per minute; and he was afebrile with a temperature of 37 °C. Multiple cervical lymph nodes were present, the largest at left level Va. There was enlargement of multiple inguinal lymph nodes, the largest in the left inguinal region measuring approximately 4×3 cm.

Initial complete blood count showed bicytopenia and leukocytosis with haemoglobin 9.4 g/dL, total white blood cell $14.74\times10^3/\mu L$, neutrophil $13.12\times10^3/\mu L$, lymphocyte $4.7\times10^3/\mu L$, monocytes $0.86\times10^3/\mu L$, and platelets $137\times10^3/\mu L$. Subsequently, the peripheral blood film showed moderate anaemia with RBC features of iron deficiency anaemia, leukocytosis with neutrophilia, likely secondary to infection, and thrombocytopenia. Iron studies favour iron deficiency anaemia, with a low serum iron level of 6.9 μmol/L, unsaturated iron-binding capacity of 24.9 μmol/L, total iron-binding capacity of 31.8 μmol/L, and transferrin saturation of 21.7%. Liver function tests were within normal limits, with total protein 81 g/L, albumin 41 g/L, globulin 40 g/L, total bilirubin 6.3 μmol/L, alkaline phosphatase 127 U/L, alanine transaminase (ALT) 14 U/L, and aspartate transaminase (AST)19 U/L. Lactate dehydrogenase was slightly elevated at 297 U/L, and C-reactive protein (CRP) was markedly elevated at 267 mg/L. Screening tests for hepatitis B (hepatitis B surface antigen), anti-hepatitis, and anti-human immunodeficiency virus were non-reactive.

An ultrasound-guided biopsy of the left inguinal lymph node was performed. Histopathological examination (HPE) of the lymph node showed total effacement of lymphoid architecture by malignant lymphoid cells that were closely intermixed with epithelioid histiocytes (Figure 1a) and scattered large Reed-Sternberg-like cells (Figure 1b). The malignant lymphoid cells display small-to-medium size, hyperchromatic nuclei with moderate nuclear pleomorphism, inconspicuous nucleoli, and scanty to clear cytoplasm. These malignant lymphoid cells were positive for CD3 (Figure 1c), CD5, and CD4 (Figure 1d), with downregulation of CD2 and CD7 (Figure 1e). They were negative for CD20, BCL6, CD79a, PAX5, CD8 (Figure 1f), CD23, CD10, PD1, CD56, CD30, ALK-1, CD15, TIA, perforin, and EBER ISH. The Ki-67 proliferative index was approximately 50%. Scattered large EBER-ISH-positive B-cells were present. The epithelioid histiocytes were highlighted by CD68 staining. The Ziehl-Neelsen stain was negative for acid-fast bacilli.

Initial computed tomography (CT) of the thorax, abdomen, and pelvis for staging (Figure 2) showed a left parotid mass, a pancreatic tail mass, and a large left thigh mass, accompanied by multiple abdominal and inguinal lymphadenopathy, likely representing multiorgan lymphoma involvement. In addition, the lung showed tree-in-bud nodules in the right upper lobe, suspicious for pulmonary tuberculosis. Conventional sputum acid-fast bacilli and auramine-stained smears were repeatedly performed and were negative for acid-fast bacilli; the mycobacterial culture was negative. CRP was elevated at 33 mg/L; however, erythrocyte sedimentation rate was not performed due to unavailability of the reagent at the time. Additional tests to confirm TB, such as polymerase chain reaction for TB, interferon-gamma release assay, and GeneXpert, were not available at our center at the time of the patient's presentation. Moreover, because of a low platelet count and severe anaemia throughout his illness, a biopsy of his lung lesion was not performed, as the procedure would likely cause more harm.

He was treated for advanced nodal PTCL, empirically treated for smear-negative pulmonary TB with lymph node involvement based on CT and HPE findings, and was started on cyclophosphamide, epirubicin, vincristine, etoposide, and prednisone (CHOEP) chemotherapy (cyclophosphamide, doxorubicin, vincristine, etoposide, prednisolone) for three cycles. Based on the imaging, microbiological tests, and the high incidence of pulmonary TB in this area, he was also empirically treated for smear-negative pulmonary TB with a two-month

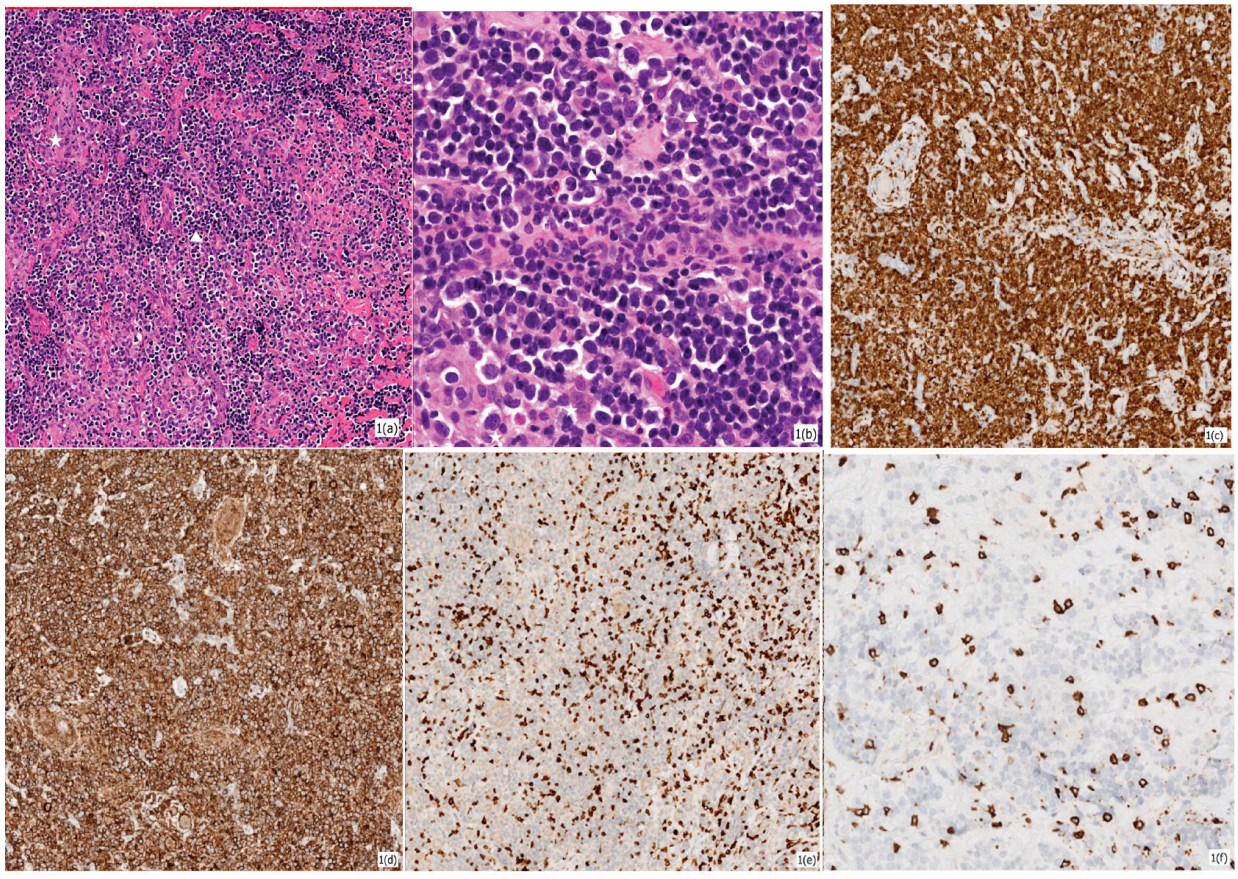

Figure 1. Histopathological examination of the left inguinal lymph node (a) showed malignant lymphoid cells (arrowhead) with epithelioid histiocytes in the background (star) [H&E staining (x100 magnification)]. (b) The malignant lymphoid cells display small to medium size, hyperchromatic nuclei with moderate nuclear pleomorphism, inconspicuous nucleoli, and scanty to clear cytoplasm (arrowhead) admixed with scattered large Reed-Sternberg-like cells (star) [H&E staining (x400 magnification)]. The malignant cells were diffusely positive for CD3 [(c) CD3 immunohistochemistry (x100 magnification)] and CD4 [(d) CD4 immunohistochemistry (x100 magnification)] with down-regulation of CD7 [(e) CD7 immunohistochemistry (x100 magnification)] but negative for CD8 [(f) CD8 immunohistochemistry (x200 magnification)]

H&E: Hematoxylin and eosin

course of anti-tuberculous treatment consisting of isoniazid, rifampicin, ethambutol, and pyrazinamide (EHRZ). He was then planned for a four-month maintenance phase of isoniazid and rifampicin (HR). As he was afebrile and had no other significant signs of infection, no antibiotic treatment was given before anti-tuberculous treatment.

However, after two months of isoniazid and rifampicin, the platelet count decreased to $82x10^3/\mu L$. Therefore, rifampicin was discontinued, and the combination of isoniazid, EHRZ was introduced. After nine days of treatment, the patient felt lethargic and unwell; his liver function tests showed elevated ALT and AST levels of 120 U/L and 84 U/L, respectively. As a result, pyrazinamide was withheld, and a combination treatment of isoniazid and ethambutol (HE) was given for a month due to transaminitis; the patient showed some improvement in symptoms. After consultation with a respiratory physician and an infectious disease consultant, the intensive combination treatment of isoniazid, rifampicin, EHRZ was reintroduced following five months of isoniazid and ethambutol treatment, which brought the total duration of anti-tuberculous treatment for his smear-negative TB to nine months.

During his follow-up, his diabetic control was not optimal, as the HbA1c readings were 7.5-7.7%. Repeated CT of the thorax, abdomen, and pelvis for disease monitoring (Figure 3) showed PTCL progression, including a new intraparotid node and enlarging pancreatic and left-thigh masses, although lung findings of smear-negative PTB had improved with disappearance of tree-in-buds on CT of the thorax. Salvage chemotherapy, gemcitabine, carboplatin, dexamethasone, was administered for two cycles, followed by 20 fractions of radiotherapy. However, disease progression continued with new bilateral inguinal lymphadenopathy. He was transitioned to palliative chemotherapy with etoposide and procarbazine. 16 months after initial diagnosis and treatment, he succumbed to the disease.

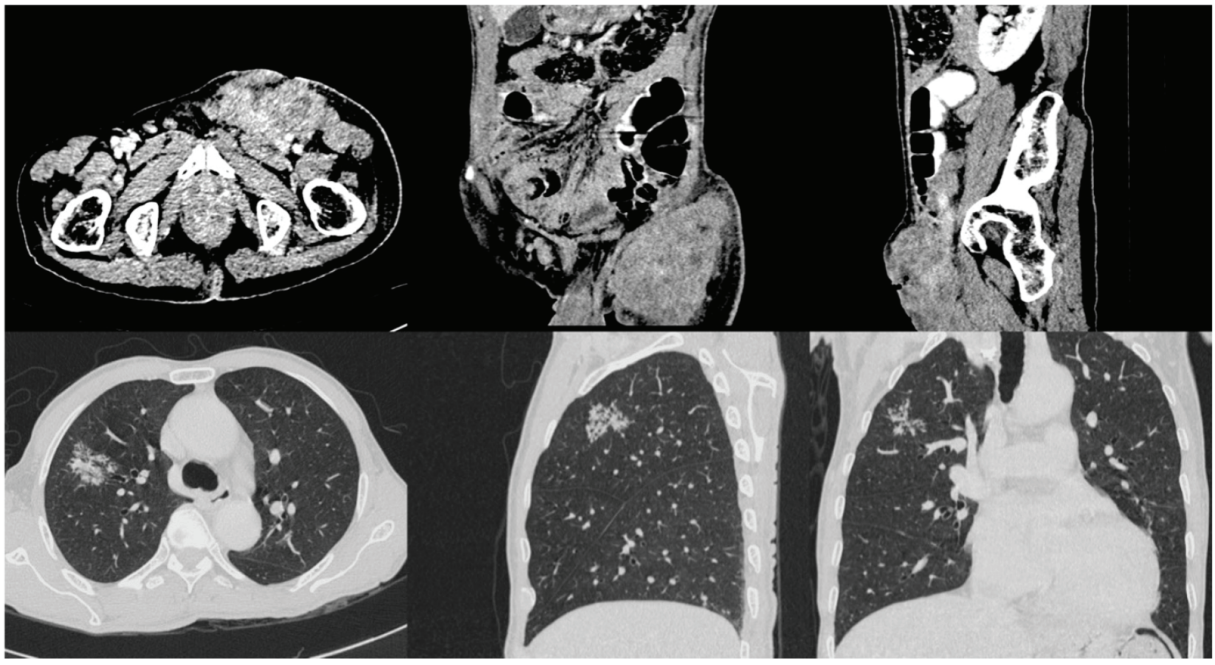

Figure 2. Initial CT scan of the thorax, abdomen and pelvis showed a left inguinal heterogeneously enhancing mass measuring 5.8 cm x11.2 cm x9.4 cm (AP x W x CC), with a central non-enhancing region likely due to necrosis. Anteriorly, there is superficial ulcerating extension of this mass, with poor fat plane with the adjacent vessel and muscles. Part of the left superficial femoral vein was compressed with partial encasement by this mass; however, no obvious filling defect seen. There were focal tree-in-buds densities in the right upper lobe however, no enlarged intrathoracic lymph nodes

CT: Computed tomography

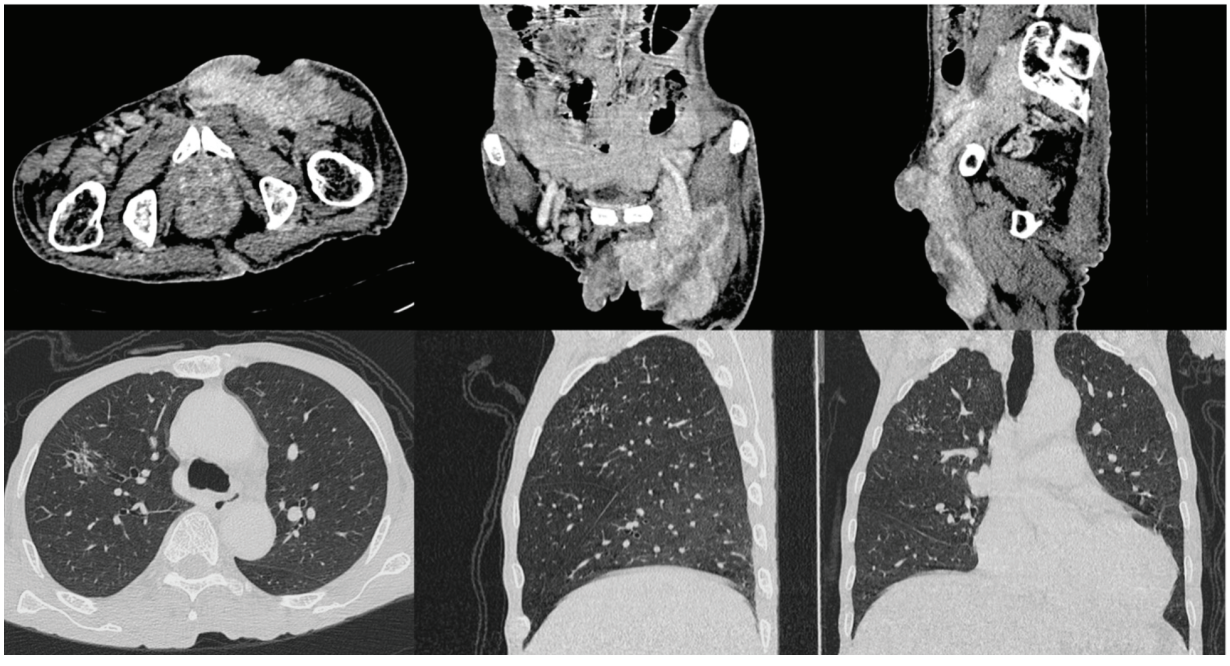

Figure 3. Repeated CT scan of thorax, abdomen and pelvis post chemotherapy showed the left inguinal heterogeneously enhancing mass was larger, measuring 6.1 cm x 14.5 cm x 16.7 cm (AP x W x CC), extending into the left pelvic region. This mass encases part of the left external iliac vein and part of the left common and superficial femoral veins, with no opacification of these vessels seen in the region of the mass. Resolving right upper lobe tree in bud densities also noted

CT: Computed tomography

Discussion

LL was first described by Lennert and Feller (5) in 1968 (6). Then, in 1988, Patsouris et al. (7) confirmed LL had a T-cell origin via immunohistochemical studies. However, only in 1992 was LL officially recognized as a lymphoma of T-cell origin, low-grade in the Kiel classification (5). Ultimately, the World Health Organisation classifies LL as a "lymphoepithelioid cell variant of the PTCL, not otherwise specified (PTCL/NOS)" (8,9).

Diabetes mellitus is a significant metabolic disorder that causes immunodeficiency, which leads to an increased risk of pulmonary TB. Macrophages are an important immunological defense against TB. The hyperglycemic state in diabetes mellitus leads to an increase in ROS and the proinflammatory cytokines interleukin (IL)-1 and IL-6, which, at high enough concentrations, can inhibit the function of macrophages (10). The proportion of patients with TB infection was higher among those with lymphoma. In a large study of 1,057 patients with lymphoma, it was found that TB infection exists in 11.9% of patients with T/NK-cell lymphoma, and among these patients, the PTCL variant was the second most common (3). The immunodeficiency caused by diabetes and LL likely contributed to TB infection in our patient.

Concurrent PTB and lymphoma are rarely encountered, and these two entities are difficult to distinguish on imaging alone. Patients with both diseases often present with lymph node enlargement on imaging. However, a study by Yang et al. (11) suggested that although there is little difference in the distribution patterns of the involved lymph nodes, their enhancement patterns differ. Peripheral enhancement of lymph nodes is seen in TB, and homogeneous lymph node enhancement points more towards untreated lymphoma (11). This imaging appearance of lymph nodes is also supported in a different study done by Tang et al. (12), in which they added that in cases of pulmonary TB, the location of the lymph nodes is often peri-hilar, whereas in lymphoma it often involves para-aortic lymph nodes. In a different study by Yang et al. (13) focusing on CT findings and survival of patients with PTCL, few imaging features may suggest PTCL, such as ill-defined margins, invasion of the surrounding structures, lesion heterogeneity, and presence of tumour necrosis, with poor survival seen in patients with the first two imaging features. According to Hare et al. (14), the most common imaging features of pulmonary lymphoma are multiple nodules or masses, usually with air bronchograms. However, it is not the mainstay for diagnosis, and hence multidisciplinary team involvement is needed to get an accurate diagnosis (14).

The diagnosis of nodal LL is typically made via histopathological evaluation of lymph nodes, which reveals effacement of lymphoid architecture by tumour cells composed of atypical lymphocytes and epithelioid histiocytes, sometimes admixed with plasma cells, eosinophils, as well as large Reed-Sternberg-like cells (8,9). Most LL cases showed an immunophenotype of $CD3^+/CD4^-/CD8^+$ (8,9,15); however, a small percentage of patients may present as $CD3^+/CD4^+/CD8^-$ or $CD3^+/CD4^-/CD8^-$ (16). Among the $CD3^+$ LL phenotype, $CD4^-/CD8^+$ is more common (about 38% of all PTCL) and $CD4^+CD8^+$ is rare (about 8% of all PTCL). LL with $CD4^-/CD8^8$ had better survival than other $CD8^+$ lymphomas (15). It is also noted that among LL cases, 31% of patients exhibited EBER-positive non-neoplastic B-cells (17). A large multi-center study revealed that PTCL/LL with EBER-positive non-neoplastic B-cells was linked to worse overall survival, particularly in patients younger than 60 years, and was consistent with an aggressive form of lymphoma (18). In our case, similar morphological features were seen, with total effacement of lymphoid architecture by tumour cells composed of atypical lymphocytes and epithelioid histiocytes, admixed with plasma cells, eosinophils, and large Reed-Sternberg-like cells. The immunophenotype, however, follows the latter, which is a $CD3^+/CD4^+/CD8^-$ immunophenotype with the presence of EBER-positive non-neoplastic B-cells, which carry a worse prognosis.

Although tree-in-buds appearance is classically described for endobronchial spread of *Mycobacterium TB*, it is also recognized as a CT manifestation of various entities such as infection (bacterial, fungal, viral, parasitic), congenital disorders (cystic fibrosis, Kartagener syndrome), idiopathic disorders (obliterative bronchiolitis, panbronchiolitis), aspiration or inhalation of foreign substances, immunologic abnormalities or connective tissue disorders (19). In addition, it may also appear in metastatic diseases such as renal cell carcinoma (most common), liver, prostate, breast, ovarian, stomach cancers, and Ewing's sarcoma (20). It is also possible in intravascular and Mantle cell lymphoma, as reported by Manglani et al. (21) and Albacar Ingla et al. (22). In our case, because PTB is endemic in our setting and the patient presented with a tree-in-bud appearance, we empirically treated our patient for smear-negative PTB, and upon completion of treatment, the disappearance of the tree-in-bud appearance may indicate a response to treatment.

The principle of management for cases of concomitant lymphoma and TB infection is challenging and requires multidisciplinary effort (23). Most of the cases of LL were treated with a combination of CHOEP regimen chemotherapy (24,25). However, this regimen can have significant interactions with anti-tuberculous treatment, particularly with rifampicin, as it affects the metabolism of several chemotherapy drugs, including vincristine, via CYP3A4 (26). Although LL was considered more favourable than other variants of PTCL (9), unfortunately, despite intensive treatment, comorbid diabetes and TB infection were difficult to treat and led to the demise of our patient. We report this case in the hope of providing additional references for clinicians and pathologists to guide future work on the management of this rare variant of PTCL and the concomitant smear-negative pulmonary TB. However, as this is a single-case report, the generalizability

of the findings and the response to treatment might be limited; the management of similar concurrent disease entities should be individualized, with involvement of a multidisciplinary team.

Conclusion

This case highlights the rare coexistence of LL and smear-negative pulmonary TB in a patient with type 2 diabetes mellitus. The overlapping clinical and radiological features of these two conditions posed significant diagnostic and therapeutic challenges. Early recognition of such concomitant presentations is crucial, as both diseases require timely and carefully coordinated management to minimize complications and drug interactions. Multidisciplinary collaboration remains essential in optimizing outcomes for patients with dual diagnoses of lymphoma and TB, particularly in those with underlying immunocompromised states.

Ethics

Informed Consent: Informed consent was obtained from the patient's spouse for the anonymous use and publication of clinical and imaging data.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.F.A., M.S.A.S., V.S.S., S.L.L., Concept: M.F.A., A.N.M.Y., Design: M.F.A., A.N.M.Y., Data Collection or Processing: M.F.A., M.S.A.S., V.S.S., S.L.L., Analysis or Interpretation: M.F.A., M.S.A.S., V.S.S., S.L.L., Literature Search: M.F.A., A.N.M.Y., M.S.A.S., Writing: M.F.A., A.N.M.Y., M.S.A.S.

Conflict of Interest: The authors declared no conflict of interest.

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