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Tuberculosis in the Context of Idiopathic CD4 Lymphocytopenia: A Case Report and Literature Review

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Patient: Male, 42-year-old
Final Diagnosis: Tuberculosis and idiopathic CD4 Lymphocytopenia
Symptoms: Cough • fever • shortness of breath • weight loss
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: Idiopathic CD4 lymphocytopenia is a rare immunodeficiency syndrome characterized by a persistent reduction in CD4 lymphocyte count to fewer than 300 cells/μL or less than 20% of total T lymphocytes on multiple occasions. This diagnosis is made in the absence of HIV infection or other known immunodeficiencies. Herein, we present a case report of idiopathic CD4 lymphocytopenia in an adult patient, contributing to the limited literature on the management challenges of this rare immunodeficiency.

Case Report: A 42-year-old male patient presented with shortness of breath, productive cough, subjective fever, night sweats, and unintentional weight loss of 6 kg over 1 month. Imaging showed a loculated right-sided pleural effusion with an air-filled cavity. The diagnosis of active pulmonary tuberculosis was confirmed based on positive tuberculosis diagnostic tests. In addition, his complete blood count showed marked lymphopenia; however, his human immunodeficiency virus (HIV) result test was repeatedly negative. In the context of investigations for lymphopenia, lymphocyte subset analysis showed a low CD4 lymphocyte count of 103.8 cells/μL, in the absence of known etiologies such as other immunodeficiencies, drug-induced leukopenia, or immunomodulatory therapy. The CD4 lymphocyte count remains below 200 cells/μL on consecutive analyses, even after successful completion of anti-tuberculosis therapy. Consequently, a diagnosis of idiopathic CD4 lymphocytopenia with pulmonary tuberculosis as an opportunistic infection was made.

Conclusions: This case highlights the importance of high clinical suspicion for idiopathic CD4 lymphocytopenia in patients presenting with lymphocytopenia and a negative history of HIV/AIDS. Further research is needed to formalize management guidelines for patients with idiopathic CD4 lymphocytopenia.

Keywords: Idiopathic CD4 Lymphocytopenia • Lymphocyte Count • Opportunistic Infections • Tuberculosis Disease
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Introduction

In the late 1980s, the physicians observed that some rare patients presented with low CD4 lymphocyte counts and opportunistic infections without evidence of human immunodeficiency virus (HIV) infection or any other recognized immune deficiency disorder. Based on clusters of patients, the U.S. Centers for Disease Control and Prevention (CDC) defined such cases of unexplained CD4 T-lymphocyte depletion under a distinct clinical entity, namely idiopathic CD4 lymphocytopenia (ICL), with the following diagnostic criteria [1]:

- CD4 T-lymphocyte count less than 300 cells/μL or fewer than 20% of total T lymphocytes on at least two separate tests, 6 weeks apart.
- No evidence of HIV infection.
- No other underlying cause for the low CD4 count, such as immunosuppressive drugs, malignancies, or other immune disorders.

ICL is diagnosed by ruling out other conditions that can cause CD4 lymphocytopenia/ depletion [2]. The pathogenesis of this rare immunodeficiency syndrome remains unclear; it appears to be a heterogeneous immunological disorder often associated with deficiencies of natural killer cells, cytotoxic T lymphocytes (CD8), and/or CD19 cells [3,4]. ICL heterogeneity is reflected in the variety of immunological status, clinical manifestations, severity, and long-term prognosis among affected patients [4,5]. Those patients are usually identified when the CD4 lymphocyte count falls below 200 cells/μL, and clinical manifestations become apparent [2].

The clinical presentations of ICL mimic those of acquired immunodeficiency syndrome (AIDS). Although most patients present with opportunistic infections, malignancies, autoimmune disorders, or neurological manifestations, some remain asymptomatic and appear otherwise healthy [3,4,6].

The opportunistic infections frequently reported in ICL patients include cryptococcosis, human papillomavirus infections, non-tuberculous mycobacterial infections, tuberculosis (TB), candidal infections, pneumocystis pneumonia, varicella-zoster virus infections, histoplasmosis, and aspergillosis. The reported malignancies include Kaposi's sarcoma, squamous cell carcinoma, basal cell carcinoma, lymphomas, and vulvar intraepithelial neoplasia [4,6]. The main documented autoimmune disorders are Sjogren's disease, psoriasis, sarcoidosis, autoimmune hemolytic anemia, and idiopathic thrombocytopenic purpura [6]. Increased CD8 lymphocytes are found in patients with autoimmune disorders; however, profoundly low CD4 lymphocytes (< 100 per μL) are associated with a decreased risk of autoimmunity [4,7]. Unexplained neurological demyelinating disorders were also reported in ICL patients, including progressive multifocal leukoencephalopathy [3,8].

Given the rarity of ICL cases, estimated to affect only 0.0002% of adults worldwide [9], we contribute this case report to the limited body of literature, highlighting the diagnostic and management challenges. In addition, we review previously reported cases of TB occurring in the context of ICL.

Case Report

A 42-year-old Sudanese expatriate male residing in Saudi Arabia presented to the emergency department with a history of recurrent chest infections over the past year (**Figure 1**). He was in his usual state of health until 2 weeks prior to presentation, when he developed a dry cough, especially at night, that progressed to a productive cough with moderate amounts of yellowish sputum associated with worsening dyspnea and subjective fever. He also reported night sweats and unintentional weight loss of 6 kg over 1 month. He had no history of contact with sick people or TB patients.

On examination, he was conscious, alert, and oriented; he appeared pale and ill. His temperature was 36.8 °C; blood pressure was 107/67 mm Hg; heart rate was 91 beats per minute; respiratory rate was 24 breaths per minute; and oxygen

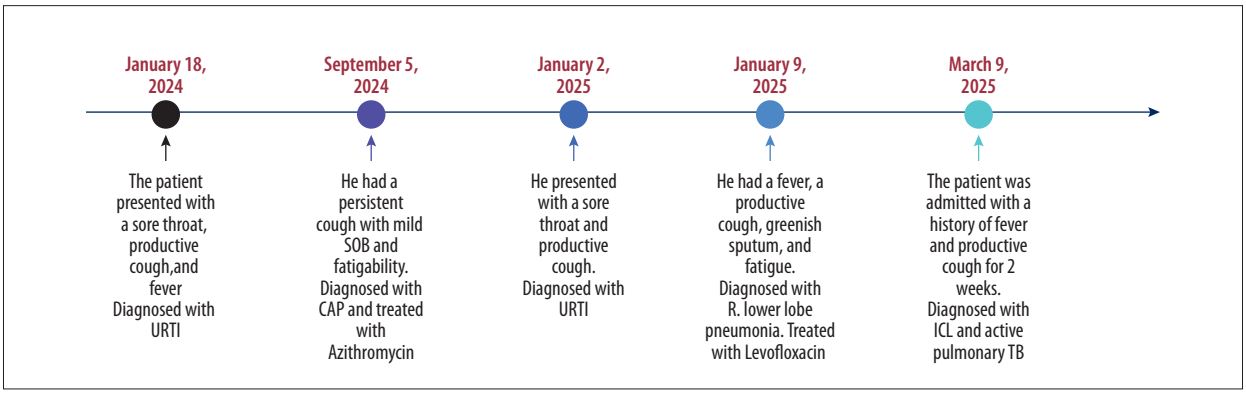

Figure 1. The clinical timeline of recurrent chest infections.

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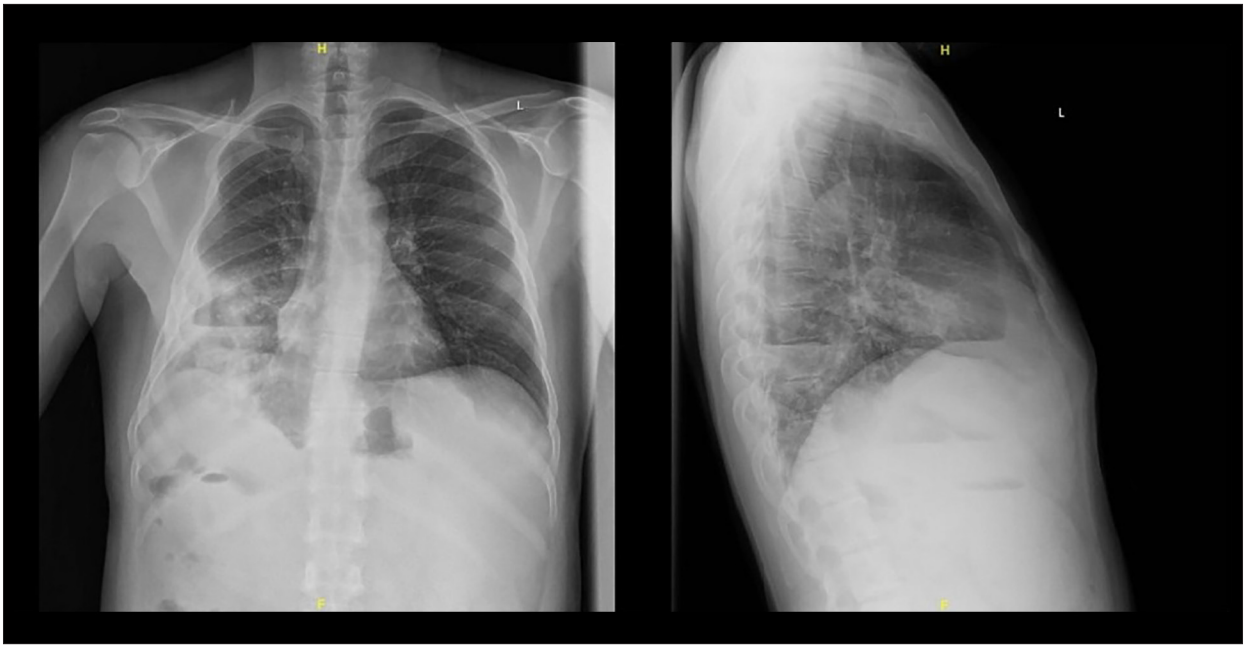

Figure 2. Chest posterior-anterior and lateral X-rays showed right lower-zone consolidation, pleural effusion, and cavity formation.

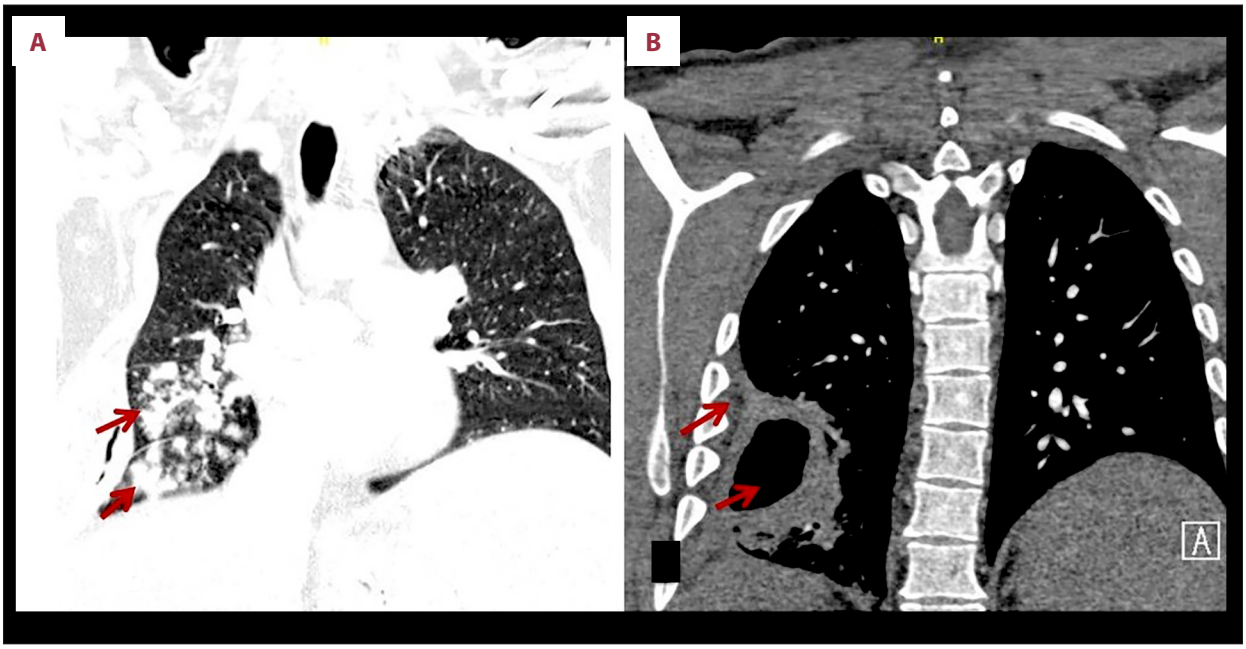

Figure 3. Chest CT shows nodular consolidation in the right middle and lower lobes (arrows) (**A**). In addition to a loculated right-sided pleural effusion with an air-filled cavity (arrows) (**B**).

saturation was 89% to 93% on room air. Chest examination revealed decreased air entry and diminished breath sounds in the right lower zone, with fine crackles. The remainder of the physical examination was within normal limits.

The initial lab results on March 9, 2025, revealed a white blood cell count of 2100/µL, with an absolute neutrophil count of 1500/µL and a lymphocyte count of 297/µL, indicating leukopenia and severe lymphopenia. Hemoglobin was 14.2 g/dL, and platelets measured 72 000/µL. Liver function tests were within normal limits, except for a low total protein of 5.8 g/dL and a mildly elevated gamma-glutamyl transferase at 70 U/L. Blood urea nitrogen was 6 mg/dL, creatinine was 0.57 mg/dL, potassium was 4.2 mmol/L, and sodium was 136 mmol/L. In addition, C-reactive protein was high at 5.65 mg/dL, erythrocyte sedimentation rate was 6 mm/h, and procalcitonin was

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Table 1. Trends in CD4+ cells, CD8+ cells, and the CD4: CD8 ratio during (June 16 and 25, 2025) and following the completion of tuberculosis therapy (October 2, 2025, December 4, 2025, and April 6, 2026).

Test	Reference range	June 16, 2025	June 25, 2025	October 2, 2025	December 4, 2025	April 6, 2026
WBC (cells/µL)	4000-11 000	1600	2100	2500	2900	2800
Lymphocytes absolute (cells/µL)	690-2540	228.0	252.2	381.1	349.2	384.3
CD4+ T Cells%	31-60	35.5	27.9	22.8	23.6	21.1
CD4+ T Cells absolute (cells/µL)	410-1590	103.8	105.2	134.2	147.0	153.7
CD8+ T Cells%	13-41	39.8	36.3	38.1	31.7	32.2
CD8+ T Cells absolute (cells/µL)	190-1140	116.2	137	223.8	197.3	234.3
CD4: CD8 ratio	≥1.0	0.9L	0.8	0.6	0.7	0.7

0.06 ng/mL. All virology workup results were negative, including HIV, hepatitis B and C viruses, herpes simplex virus, Epstein–Barr virus (EBV), cytomegalovirus (CMV), and parvovirus B19. Serologic tests for brucellosis, toxoplasmosis, and syphilis were also negative. Immunoglobulin fractions (IgM, IgG, IgA, and IgE) were within normal limits.

Chest X-ray showed right lower-zone consolidation, pleural effusion, and cavity formation (**Figure 2**). Further, chest computed tomography (CT) with contrast revealed a loculated right-sided pleural effusion in the right lower zone with an air-filled cavity, along with adjacent focal consolidation and an air bronchogram (**Figure 3**).

Given the high suspicion of TB, a full TB panel was sent, consisting of 3 early-morning sputum samples collected on three consecutive days for acid-fast bacillus (AFB) staining, mycobacterial culture, and TB PCR testing. The AFB staining was positive for all three samples. TB PCR and mycobacterial culture also turned positive for *Mycobacterium tuberculosis*, confirming the diagnosis of active pulmonary TB. The patient was started on an anti-tuberculosis regimen that included Isoniazid 300 mg PO OD, Rifampicin 600 mg PO OD, Ethambutol 1200 mg PO OD, and Pyrazinamide 1500 mg PO OD, in addition to Pyridoxine 40 mg PO OD on March 16, 2025. The initial 4-drug regimen was continued for 3 months when AFB sputum turned negative. On June 16, 2025, he started the anti-TB continuation phase (Isoniazid 300 mg PO OD, Rifampicin 600 mg PO OD, and Pyridoxine 40 mg PO OD) for a further 3 months. He completed anti-tuberculosis treatment on September 16, 2025, with complete resolution of associated clinical manifestations and negative AFB sputum results. However, the patient remained persistently lymphopenic. On June 16, 2025, CD4/CD8 counts were ordered, which revealed a low absolute T-cell count of 228.0 cells/µL, a low absolute CD8+ count of 116.2 cells/µL, and a low absolute CD4+ count of 103.8 cells/µL.

Persistent CD4 lymphocytopenia was subsequently demonstrated in the next four consecutive measurements, all showing an absolute CD4 count below 200 cells/µL. These were obtained on June 25, 2025, during anti-tuberculosis treatment, and on October 2, 2025, December 4, 2025, and April 6, 2026, after successful completion of anti-tuberculosis therapy (**Table 1**).

Therefore, the diagnosis of ICL was made in the absence of secondary causes of lymphocytopenia, including HIV infection, other immunodeficiencies, or drug-induced leukopenia. The patient was started on trimethoprim-sulfamethoxazole (TMP/SMX) for Pneumocystis pneumonia (PCP) prophylaxis as part of the follow-up care plan.

Discussion

In this challenging case, the diagnosis of ICL was established through close follow-up for more than 1 year. The depletion of CD4 lymphocytes, which can occur in the context of active tuberculosis, was initially suspected; however, the persistence of a very low CD4 lymphocyte count for more than 6 months after completion of TB treatment, together with the absence of any identifiable secondary etiology, affirms the diagnosis of ICL.

ICL cases have been reported worldwide [10], including the Americas, Europe, Africa, Australia, and the Middle East. The most available data regarding ICL epidemiology are derived from small case series and reports involving diverse geographic and ethnic populations, with no consistent racial or demographic patterns identified despite reported genetic variants and familial occurrence [7,11,12]. A review of the literature reveals only 1 case report of ICL, in a 9-year-old patient from Saudi Arabia, which was initially misdiagnosed as primary ciliary dyskinesia [13], and no adult cases of ICL had been documented prior to the recent case described in a Sudanese expatriate residing in Saudi Arabia.

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Table 2. Reported TB cases in the context of ICL in the literature.

Paper type	Publication year	TB Patient number	TB manifestations	Associated opportunistic infections/ malignancies	Outcome	Reference
Retrospective cohort study (24 patients)	2017	3 patients, not specified whether they were non-tuberculous or tuberculosis	No data	No data	No data	Yarmohammadi and Cunningham-Rundles [3]
Retrospective cohort study (40 patients)	2014	2 TB patients and one with non-tuberculous infection	1 – Extrapulmonary tuberculosis (CNS TB) 2 – Not defined	Not defined	One patient died	Régent et al [4]
Longitudinal cohort study (91 patients)	2023	5 patients with non-tuberculous infections	No data	No will defined	No data	Lisco et al [7]
Case report	2009	1	Disseminated TB	None	Treated successfully	Thoden et al [9]
Case report	2014	1	Pulmonary TB	None	Treated successfully	Said et al [10]
Case report	1999	2	Disseminated tuberculosis	1- Herpes zoster 2- None	Both treated successfully	De Socio et al [17]
Case report	1992	1	Disseminated TB	Kaposi's sarcoma	Treated successfully	Castro et al [18]
Case report	2000	1	Extrapulmonary TB (intestinal tuberculosis)	Esophageal candidiasis	Treated successfully	Hirasaki et al [19]
Brief case report	2001	1	Disseminated TB	Cryptococcal pneumonia and Cryptococcemia	The patient was treated for 24 weeks with improvement	Zaharatos et al [20]
Case report	2011	1	Pulmonary TB	Epidermodysplasia verruciformis-like skin eruption and *Nocardia farcinica* brain abscesses	Died	Göktay et al [21]
Case report	2022	1	Pulmonary TB, tuberculous pleuritis, and meningitis TB	None	Treated successfully	Triwicaksana et al [24]
Case report	2015	1	Disseminated TB	None	Treated successfully	Sikri et al [25]
Case report (2 patients)	2013	2	Pulmonary TB	1 – Kaposi's sarcoma 2 – None	Died	Ollé-Goig et al [26]
Case report	2016	1	Extrapulmonary TB	None	Treated successfully	Ramalingam et al [27]
Case report	1995	1	Disseminated TB	None	Treated successfully	Neukirch and Kremer [28]
Case report	1998	1	Disseminated TB (miliary TB)	None	Treated successfully	Suzuki et al [29]

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ICL is a heterogeneous immunodeficiency syndrome first recognized by the CDC as a distinct clinical entity in 1992 [1]. However, after more than 3 decades, the accumulated knowledge remains limited, based mainly on case reports or case series, with fewer than 500 ICL cases reported globally. The etiology of ICL remains unknown. Given the clinical manifestations' similarity to those seen in AIDS patients, an infectious etiology was initially investigated and excluded [5]. Various immunologic defects have been associated with ICL; however, these associations have not yielded a robust hypothesis for ICL pathogenesis. Loss of CD4 lymphocytes due to increased activation and turnover, as well as accelerated apoptosis, has been suggested [6]. Perez-Diez et al proposed autoantibodies as the primary mechanism of CD4 lymphocyte depletion [12]. Induction of CD4 T-lymphocyte apoptosis by overexpression of the cell-surface death receptors Fas and Fas/CD95 was also reported [14]. In addition, some mutations have been identified in ICL patients, suggesting a genetic etiology for specific ICL variants [4,7,12]. Furthermore, Lin et al reported a familial occurrence of ICL in 2 young siblings [11]. However, the mean age of ICL patients in published reviews and ICL cohort studies was above 40 years [3,4,6,7], which may support an acquired etiology of ICL or, at least, the late-onset of an intrinsic immunodeficiency defect that leads to permanent CD4 depletion.

CD4 lymphocytopenia resulting from viral infections, such as EBV, CMV, and coronavirus disease 2019 (COVID-19), as well as from severe sepsis, tuberculosis, or other infections, is well established in the literature. Nonetheless, CD4 cell depletion attributable to infectious diseases is often reversible, with counts typically normalizing after recovery, as observed in TB patients.

Among TB patients, Kony et al reported that CD4 lymphocyte counts were less than 300 cells/μL in 62 (14.4%) of 430 HIV-negative patients diagnosed with active TB and indicated that extrapulmonary TB cases were more common among patients with counts less than 300/μL [15]. Also, Al-Aska et al observed that CD4 and CD8 lymphocyte counts were significantly lower in TB patients with HIV-negative tests compared with normal individuals, and profound CD4 lymphocytopenia is frequently associated with disseminated TB [16]. They also reported that CD4 lymphocyte counts tended to normalize after recovery. In our case, the patient showed profound CD4 lymphocytopenia in 5 consecutive tests—2 during anti-tuberculosis therapy and 3 after completion of the therapy course. In addition, partial improvements in CD4 and CD8 lymphocyte counts occur gradually as the patient recovers (**Table 1**). These findings support the diagnosis of ICL and, at the same time, demonstrate the effects of active TB on CD4 and CD8 lymphocyte counts, consistent with previous reports in the literature.

Opportunistic infections in the context of ICL are well described. However, the frequency of infections associated with ICL is primarily determined by the epidemiology of the causative agents and the extent of immunodeficiency. The most commonly reported opportunistic infections were human papillomavirus-related infections and cryptococcal infections [3,4,7]. Regarding mycobacterial infections, Ahmed et al identified 258 ICL cases from 1989 to 2012 in their review and found that 44 ICL patients (25 with non-tuberculous mycobacterial infections and 19 with tuberculosis) had mycobacterial infections [6]. Régent et al reported 2 cases of tuberculosis and 1 case of non-tuberculous mycobacterial infection [4]. Yarmohammadi and Cunningham-Rundles identified mycobacterial infections in three patients, without specifying whether they were non-tuberculous infections or tuberculosis [3]. Finally, Lisco et al reported that, among their cohort of 91 ICL patients, five had non-tuberculous mycobacterial infections; however, no cases of tuberculosis were identified [7]. The main findings from the studies mentioned above on tuberculosis were presented alongside the reviewed case reports in **Table 2**. In addition to our case, we identified reports of 17 documented active TB cases among ICL patients. Multifocal or disseminated TB was the most common form of TB in ICL patients (52.9%), while pulmonary TB was reported in only four (23.5%) of ICL patients with TB. Other opportunistic coinfections or malignancies presented with TB were reported in 6 patients (35.3%), including herpes zoster, cryptococcosis, Kaposi's sarcoma, nocardiosis, and esophageal candidiasis [17-21]. Four ICL patients (23.5%) diagnosed with TB died. The duration of anti-tuberculosis treatment varied, with some patients receiving treatment for up to 2 years [17].

Regarding ICL management, there are currently no specific guidelines. However, treatments and preventive measures for opportunistic infections generally follow the same protocols used for opportunistic infections in patients with HIV/AIDS [2,3]. The same concept was applied to our patients as well. He was initiated on TMP/SMX prophylaxis with regular follow-up.

In terms of targeted therapy, some researchers have explored the use of interleukin-2, interleukin-7, and interferon-γ to increase the CD4 lymphocyte count, with promising results [2,22,23]. In certain cases, allogeneic hematopoietic stem cell transplantation has been employed as a definitive therapeutic option for patients with ICL [2].

Conclusions

This case report contributes to the limited literature on the presentation pattern of ICL, underscores the diagnostic complexity and challenges of distinguishing ICL from transient CD4 depletion associated with active tuberculosis, and highlights the importance of follow-up findings demonstrating persistent CD4 lymphocytopenia after completion of anti-tuberculosis therapy.

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In tuberculosis patients with unexplained lymphocytopenia or who do not respond to standard treatment, early immunological evaluation should be considered to facilitate timely recognition of underlying immune deficiency. The available literature indicates that TB, as an opportunistic infection in ICL patients, frequently presents as multifocal or disseminated infection, often concomitant with other opportunistic infections, and has a poor prognosis with a high mortality rate.

Recognition of this entity is clinically important because confirmation of the ICL diagnosis has direct implications for prophylaxis against opportunistic infections. Further research is required to establish evidence-based recommendations for the evaluation and management of ICL.

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Institution Where Work Was Done

Dammam & King Fahad Hospital of the University, Al Khobar, Saudi Arabia.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying data.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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