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COEXISTING AUTOIMMUNE HAEMOLYSIS AND PNEUMOCYSTIS JIROVECII PNEUMONIA IN AN ELDERLY COPD PATIENT: A CASE REPORT

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ABSTRACT

Pneumocystis jirovecii pneumonia (PCP) is a life-threatening opportunistic infection, mostly seen in individuals with overt immunosuppression, such as those living with HIV/AIDS. However, recent trends show increasing incidence in individuals without classical risk factors, prompting the need for broader clinical awareness. We present a complex and ultimately fatal case of PCP in an elderly man with chronic obstructive pulmonary disease (COPD), whose clinical course was complicated by newly diagnosed autoimmune haemolytic anaemia (AIHA).The patient, a 72-year-old man, arrived with progressively worsening breathlessness, fatigue, and pallor, initially attributed to a COPD exacerbation. He had no history of immunosuppressive therapy, HIV, or known immunodeficiency. However, laboratory investigations revealed severe anaemia, and a positive Direct Coombs Test confirmed AIHA. A chest CT scan raised concern for a complex pulmonary infection in an already compromised lung with findings not typical of PCP alone, but possibly exacerbated by the underlying COPD and emerging immune dysfunction from AIHA. Ultimately, microscopic examination of induced sputum stained with Giemsa and toluidine blue O confirmed P. jirovecii infection. Although, trimethoprim-sulfamethoxazole and corticosteroids were promptly initiated, the patient succumbed to progressive respiratory failure within six days. This case underscores the importance of considering PCP in non-HIV patients, particularly those with chronic lung disease and immune dysfunction. Although rare, AIHA in COPD can further weaken immune defences, heightening the risk of severe infections. The diagnostic challenge posed by clinical overlap with bacterial pneumonia and tuberculosis in non-HIV PCP underscores the need for early empirical treatment, prompt recognition, and targeted therapy to improve outcomes.

Keywords: Pneumocystis jirovecii, PCP, Autoimmune haemolytic anaemia, chronic obstructive pulmonary disease, Giemsa and toluidine blue O and Trimethoprim–sulfamethoxazole

INTRODUCTION

Pneumocystis jirovecii is a globally widespread opportunistic fungus that can cause life-threatening pneumonia in individuals with weakened immune systems [1]. Pneumocystis jiroveciipneumonia (PCP) primarily occurs in immunocompromised individuals, including those with human immunodeficiency virus (HIV), recipients of solid organ or hematologic transplants, individuals on prolonged high-dose corticosteroids, and patients undergoing immunosuppressive therapies like chemotherapy or biologics for cancer or autoimmune diseases [2]. While immunocompetent individuals are often exposed to the P. jirovecii, they usually experience either asymptomatic infections or mild symptoms [3]. In cases of HIV-associated PCP, over 90% of infections are seen in patients with CD4+ T-cell counts below 200 cells/mm3, and PCP is classified as an AIDS-defining illness [1]. The introduction of early and effective antiretroviral therapy (ART), along with primary prophylaxis using trimethoprim–sulfamethoxazole (TMP-SMX), has significantly reduced PCP incidence in people with HIV. However, PCP now disproportionately affects non-HIV immunocompromised individuals [4]. Previous studies comparing mortality rates between HIV-positive and HIV-negative patients with PCP demonstrated that non-HIV patients experienced significantly higher mortality rates (33–71%) compared to HIV-positive patients receiving highly active ART (13–18%). This higher mortality rate among non-HIV patients is largely attributed to delayed diagnosis and more severe respiratory failure [5].

The diagnosis of PCP is a complex, multifactorial process requiring multiple approaches that combine clinical, imaging, and laboratory assessments [6]. Clinically, patients typically present with progressive dyspnoea, nonproductive cough, and fever, necessitating a high index of suspicion, particularly in immunocompromised individuals [1]. According to the updated 2019 EORTC/MSGERC consensus definitions of invasive fungal infection, Proven PCP is established through clinical and radiologic evidence coupled with microscopic identification of P. jirovecii in tissue or respiratory specimens using conventional or immunofluorescence staining. Probable PCP requires the presence of appropriate host factors and compatible clinical-radiologic features, along with detection of P. jirovecii DNA by quantitative PCR or β-D-glucan (BDG) in serum, provided that other fungal infections and false positives are excluded. Cases lacking microbiological confirmation but exhibiting consistent clinical and radiological findings are classified as possible PCP, a less definitive category mainly used in research contexts. [7].However, it is critical to note that current diagnostic methods, particularly qPCR and BDG assays, cannot reliably differentiate between active infection and mere colonization with P. jirovecii. Although qPCR thresholds and elevated BDG levels are proposed to aid in this distinction, standardized diagnostic cut-offs remain unresolved and are not universally accepted [7]. This diagnostic uncertainty poses a significant challenge in both clinical and research settings, especially when making treatment decisions in patients with underlying chronic lung conditions or immunosuppression.

Additionally, diagnosing PCP in patients with chronic respiratory diseases presents challenges due to overlapping symptoms and typically low fungal loads, which complicate detection through standard diagnostic methods. Pulmonary diseases remain among the leading causes of morbidity and mortality worldwide, with PCP and chronic obstructive pulmonary disease (COPD) representing two distinct yet significant challenges in respiratory medicine [8]. Interestingly, Pneumocystis has been found to have a high prevalence among COPD patients, contributing to increased disease severity even when the fungal load is minimal [9]. Recent studies using an elastase-induced COPD rat model have shown a link between COPD severity and Pneumocystis infection, with increases in inflammation and mucus-related markers [10]. These findings strengthen the hypothesis that a mild Pneumocystis infection may intensify inflammation or other pathological features, such as fibrosis, in respiratory diseases like COPD, either directly or as a contributing factor [11]. Furthermore, studies on human COPD patients infected with P. jirovecii have shown that the presence of this fungus is associated with elevated levels of pro-inflammatory cytokines, such as TNFα, IL-6, and IL-8 [12]. Additionally, P. jirovecii colonization has been identified in COPD patients, correlating with acute exacerbations,

and has been linked to the activation of a strong Th1 inflammatory response in COPD patients or TH2 and TH17 inflammatory responses [13]. Moreover, additional evaluations and continued investigations are required to understand the clinical implications of Pneumocystis involvement in the progression and development of chronic respiratory diseases.

Here, we present a complex case of the interplay of chronic respiratory disease, autoimmune anaemia, and opportunistic infection in elderly patients. The patient's COPD exacerbation, and coexisting autoimmune haemolyticanaemia (AIHA), and PCP illustrate how chronic lung conditions combined with immune dysregulation can complicate both the diagnosis and management of severe opportunistic infections.

CASE PRESENTATION

A 72-year-old Indian male with a history of smoking-related COPD presented with repeated episodes of abdominal pain and increasing respiratory distress. The patient had not been on any regular maintenance therapy for COPD prior to admission. This was attributed due to limited access to healthcare services and inconsistent follow-up, as he had not been under regular medical care for his condition. He mentioned occasional use of over-the-counter
remedies for respiratory symptoms though denied the use of prescribed bronchodilators, inhaled corticosteroids, or other COPD-specific medications. There was no documented history of primary immunodeficiency, HIV, or immunosuppressive therapy.However, a more comprehensive evaluation of the patient's immune status could not be performed due to resource limitations. He was referred to our tertiary care hospital from another facility due to unmanageable worsening hypoxia and respiratory decline. The patient was not on any previous medication; however, he continues to experience increased fatigue, shortness of breath, and a persistent dry cough, despite several days of treatment at our tertiary care facility. On detailed physical examination, the patient exhibited reduced vesicular breath sounds with a prolonged expiratory phase. Furthermore, the physical examination also revealed notable pallor and icterus. On abdominal palpation examination mild hepatomegaly was observed without significant splenomegaly.

The patient's initial laboratory results indicated a BUN/creatinine ratio of 13.5/0.9, which was within the normal range, suggesting adequate renal function and no significant prerenal azotaemia. Sodium was slightly low at 132 mmol/L, suggesting mild hyponatremia, which could potentially contribute to nonspecific symptoms such as fatigue or confusion if clinically symptomatic. Bicarbonate was at 22 mmol/L, within a lower normal range, hinting at a possible subtle acid–base disturbance but not severe enough to show overt metabolic acidosis. Liver function tests revealed aspartate transaminase at 48 U/L, alanine transaminase at 110 U/L, and an elevated lactate dehydrogenase (LDH) level of 883 U/L. The WBC count is 11,000/µL, with neutrophils at 83% and lymphocytes at 12%, and a chest X-ray revealed diffuse bilateral pulmonary consolidations, which aided in the differential diagnosis, most likely indicating pneumonia.

The patient presented with critically low haemoglobin (6 g/dL), indicating severe anaemia that required urgent intervention. Testing with a Direct Coombs Test revealed a strong
positive result (4+), pointing toward AIHA as the cause of this rapid red blood cell (RBC) destruction. Due to the immediate need for stabilization, the medical team proceeded with a transfusion of carefully matched packed red blood cells (PRBCs), which successfully raised the haemoglobin to 9.3 g/dL. However, despite this initial improvement, the patient continued to experience active haemolysis, evidenced by an elevated LDH level of 833 U/L. Additionally, a reticulocyte counts of 6.84% (Reticulocyte Index of 3) indicated increased RBC production as the body attempted to compensate for the ongoing cell destruction.

Blood cultures for bacteria and fungi were collected immediately upon the patient's admission and remained negative throughout the duration of the hospitalization, indicating the absence of detectable infection in the bloodstream. Likewise, sputum cultures showed no microbial growth, suggesting there was no active bacterial or fungal infection in the respiratory system. The atypical laboratory findings with active symptoms, also suggested a possible infection with a non-culturable

organism. Given the difficulty in collecting a sputum sample, obtaining an accurate specimen for proper diagnosis proved challenging. Likewise, the lack of testing for potential viral causes in the current situation presented a significant obstacle in sampling. Without these tests, doctors face a greater challenge in reaching an accurate diagnosis, as they cannot exclude or verify the possibility of a viral infection influencing the patient's symptoms.

Figure Legend

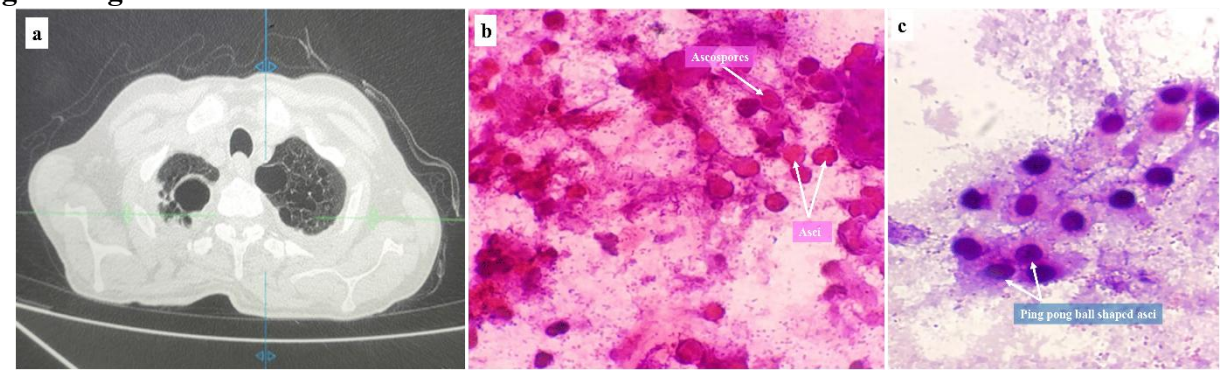

FIG 1 (**a**) CT scan of the patient's chest showing a large cavity in the right upper lobe abutting the mediastinal pleura, with adjacent small soft tissue lesions. Centrilobular and paraseptal emphysema are visible in the left upper lobe. (**b, c**) Pneumocystis jirovecii visualized under 1000× magnification using Giemsa and Toluidine Blue O stains, (**b**) Giemsa stain highlighting cysts containing intra-cystic ascospores arranged in a circular pattern within a distinct ascus wall. (**c**) Toluidine Blue O stain demonstrating multiple round, well-outlined asci (spore-containing cysts), showing the characteristic "ping pong ball-like" appearance of P. jirovecii cysts.

The patient's overall condition deteriorated, with worsening dyspnoea. To address this, the medical team intensified treatment by adding two classes of broad-spectrum antibiotics: meropenem (1 g IV every 8 hourly), a carbapenem antibiotic, and levofloxacin (750 mg IV once daily) a fluoroquinolone. Additionally, steroids were started to help control the patient's inflammatory response.The clinical management of this patient's progressively worsening condition presented several challenges, especially given the lack of response to initial treatment. The case took a pivotal turn following a computed tomography (CT) scan of the chest, which revealed a large cavity in the right upper lobe abutting the mediastinal pleura, surrounded by small soft tissue lesions, along with centrilobular and paraseptal emphysema in the left upper lobe (Fig. 1a). Although these findings initially suggested chronic cavitary infections or structural lung disease associated with COPD, they did not fully account for the patient's deteriorating respiratory status. Clinically, he continued to experience persistent dry cough, fatigue, increasing shortness of breath, and episodes of abdominal pain, all of which failed to improve despite appropriate empirical management. This mismatch between radiologic findings, lack of organism isolation upon culture and clinical progression raised the suspicion of an underlying opportunistic infection, particularly PCP, despite the absence of its classic radiographic features.

The patient's condition worsened further, necessitating transfer to the intensive care unit (ICU) for ventilatory support and close monitoring. Based on high clinical suspicion, empirical treatment with oral (nasogastric) trimethoprim-sulfamethoxazole (TMP-SMX) 15-20 mg/kg/ day was initiated which is the treatment of choice in case of PCP. In parallel, fluconazole (400 mg/day) was administered to address the possibility of other concurrent fungal infections. Given signs of immune dysregulation and suspected PCP-associated inflammation, intravenous methylprednisolone (40 mg/day) was also started following TMP-SMX treatment modality. To elaborate, following the challenges in obtaining a standard sputum sample, induced sputum was collected. This subsequently underwent detailed microscopic examination, as it was the only specimen available for analysis. Preferred staining methods, such as Giemsa stain, highlighted cysts containing intra-cystic

ascospores arranged in a circular pattern within a distinct ascus wall (Fig. 1b). Subsequent staining with Toluidine Blue O, observed under 1000× magnification, revealed multiple round, well-defined asci (ascospore-containing cysts) exhibiting the characteristic 'ping pong ball-like' appearance of P. jirovecii cysts, suggestive of PCP (Fig. 1c). Despite intensive care and the timely initiation of appropriate treatment, the patient's condition continued to progressively decline. He survived for six days following the initiation of TMP-SMX therapy but ultimately succumbed to progressive respiratory failure and complications from both PCP and AIHA.

DISCUSSION

This case highlights the importance of considering opportunistic infections like PCP in elderly patients with COPD, particularly those with underlying immune dysfunction. In this patient, a combination of age-related immune decline, structural lung damage from COPD, and coexisting AIHA significantly increased susceptibility to infection, complicating both diagnosis and management. Although PCP is commonly associated with profound immunosuppression, it can also occur in individuals with less recognized risk factors such as autoimmune or chronic inflammatory diseases [14]. Notably, this atypical case was diagnosed through induced sputum staining, as BAL, the recommended sample, could not be obtained due to the patient's worsening clinical condition. The immunosuppressive effect of corticosteroid therapy for AIHA further contributed to the development of PCP, emphasizing the need for heightened clinical vigilance in similar scenarios. The clinical presentation of PCP often overlaps with various respiratory conditions, making early diagnosis particularly challenging. Common symptomssuch as fatigue, dyspnoea, cough, malaise, and hypoxiaare non-specific and frequently seen in diseases like tuberculosis, bacterial pneumonia, Legionella infection, COVID-19, and acute respiratory distress syndrome [15]. This significant overlap in clinical features leads to diagnostic ambiguity, a recurring issue in clinical practice, resulting in PCP being frequently misdiagnosed or overlooked. Compounding the issue, the diagnosis becomes even more complex in resource-limited settings where access to advanced diagnostic tools such as PCR, BDG assays, and Gomori-Grocott Methenamine Silver (GMS) staining is constrained. Although PCR is highly sensitive for detecting P. jirovecii, it cannot reliably distinguish colonization from active infection, potentially leading to false positives. Quantitative PCR, which estimates fungal load, offers better specificity, but its utility is hampered by the lack of universally accepted threshold values. To improve diagnostic accuracy, it has been recommended to combine qPCR results with serum BDG levels ≥80ng/L detection in ≥2consecutive serum samples [16], and specific qPCR cut-offs have been proposed to help differentiate infection from colonization [17]. Nevertheless, BDG remains a non-specific fungal marker and can yield false positives in the presence of other fungal infections. Similarly, microscopy-based techniques such as Giemsa staining, though commonly used, are operator-dependent, susceptible tofalse positives, and less informative without bronchoalveolar lavage (BAL) samples [18]. A recent study compared mortality rates among patients diagnosed with PCP following allogeneic hematopoietic cell transplantation using microscopy versus PCR alone on BAL samples. The findings indicated no statistically significant difference in mortality between the two groups. However, microscopy-positive cases exhibited a trend toward higher ICU admission rates ($p = 0.05$) this likely reflects a higher fungal load or more severe disease at presentation rather than a fundamental difference in diagnostic accuracy. This underscores the need for prompt and effective management strategies based on disease severity rather than diagnostic methods alone [19]. In response to these challenges, the 2019 EORTC/MSGERC consensus definitions advocate for a comprehensive diagnostic framework that incorporates host immune status, clinical presentation, imaging findings, and quantitative fungal burden [7]. Such an integrated diagnostic approach is particularly valuable for distinguishing true fungal infection from colonization, a distinction that remains particularly challenging in individuals with chronic lung diseases such as COPD. Pneumocystis jirovecii is frequently detected in this population; however, its clinical significance remains uncertain. Reported prevalence rates of Pneumocystis jirovecii colonization in patients with COPD vary widely, ranging

from 16% to 55%, highlighting both population-specific variability and differences in diagnostic criteria [20]. Although biomarker-based diagnostics have advanced, the lack of standardized cut-off values continues to hinder consistent interpretation across clinical settings. Colonized individuals may act as reservoirs for transmission and are at increased risk of developing PCP if immunosuppressed. Emerging evidence also suggests that even low levels of P. jirovecii in the lungs may contribute to persistent inflammation and structural remodelling, potentially accelerating COPD progression and perpetuating the cycle of disease [21].Additionally, COPD itself is known to impair pulmonary defences, further heightening the risk of fungal infections like PCP [22].Experimental evidence from both human and animal studies suggests that colonization may provoke systemic and localized inflammatory responses, contributing to structural lung damage and progressive decline in pulmonary function [10]. However, the direct causal relationship between colonization and COPD progression remains to be fully elucidated, highlighting the need for further research [23]. Smoking, a major risk factor for COPD, has also been associated with heightened susceptibility to fungal colonization and accelerated lung function decline. Interestingly, data from non-HIV populations indicate that Pneumocystis colonization may exacerbate COPD severity even in individuals without corticosteroid use or a smoking history [24], suggesting a more complex and potentially underrecognized role of this pathogen in COPD pathogenesis.

The occurrence of AIHA alongside COPD presents difficult clinical challenges, particularly during blood transfusions. The Direct Antiglobulin Test (DAT or Coombs test) is the gold standard for diagnosing AIHA by detecting red cell-bound autoantibodies or complement and guiding clinical management. Treating AIHA in the setting of chronic respiratory disease requires a delicate balance between correcting hypoxia from anaemia and minimizing transfusion-related risks [25]. This complexity demands a multidisciplinary approach involving pulmonologists, haematologists, and transfusion medicine specialists to ensure tailored and effective care. Immunosuppressive therapy, particularly corticosteroids, is often essential for controlling the autoimmune process and reducing transfusion dependence. The present case of coexisting COPD, AIHA, and PCP underscores the intricate interaction between chronic lung disease, immune dysregulation, and opportunistic infections. It illustrates the diagnostic uncertainty and therapeutic hurdles posed by overlapping clinical presentations. These challenges highlight the pressing need for the formulation of specific international or national guidelines to manage AIHA in the context of pneumonia and COPD, ensuring timely and evidence-based intervention in such complex clinical scenarios.

TMP-SMX remains the first-line treatment for PCP, with therapeutic success often confirming the diagnosis retrospectively [26]. However, TMP-SMX is not without risks. One rare but significant adverse effect is haemolyticanaemia, which occurs due to oxidative stress-induced RBC destruction. This complication is more pronounced in patients treated for PCP and other infections, such as urinary tract infections and nocardiosis, with TMP-SMX [27].Historically, sulfamethoxazole was considered highly risky for patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency due to their heightened vulnerability to oxidative damage. However, recent evidence suggests that the risk of haemolyticanaemia may be lower than previously thought, especially at prophylactic doses [28]. The 2022 G6PD Clinical Pharmacogenetics Implementation Consortium (CPIC) guidelines reported minimal evidence linking TMP-SMX prophylaxis to haemolyticanaemia in G6PD-deficient patients [29]. Nevertheless, factors such as dosage and individual patient characteristics play a critical role in determining risk. In this case, the patient's immediate need for TMP-SMX therapy to manage suspected PCP took precedence over the risk of haemolysis. Despite appropriate treatment, some patients may experience worsening respiratory failure due to the severity of PCP. In this scenario, the patient's underlying AIHA significantly complicated clinical management, as ongoing haemolysis heightened the risk of anaemia-induced hypoxia, particularly in the setting of respiratory compromise. This necessitated close haematological monitoring and supportive care throughout the course of treatment. Emerging treatments such as echinocandins are being explored as alternatives or adjuncts to TMP-SMX for PCP. Echinocandins, including caspofungin, micafungin, and anidulafungin, target the β-(1,3)-D-glucan in Pneumocystis cyst

walls, effectively addressing the cystic forms of the pathogen. However, their reduced efficacy against trophic forms limits their use as standalone treatments. Current evidence suggests that echinocandins may be useful in combination with TMP-SMX, particularly in patients who cannot tolerate standard therapies [30]. Rezafungin, a newer echinocandin, shows promise for prophylactic use but requires further investigation [31].

This case underscores the critical importance of early diagnosis and timely treatment of PCP in non-HIV patients, particularly those with chronic conditions such as COPD and coexisting immune dysfunctions like AIHA. Despite the absence of an initial history of primary immunodeficiency or immunosuppressive therapy, the emergence of AIHA followed by PCP strongly suggested a background of unrecognized immune dysregulation. However, a significant limitation in this case was the lack of comprehensive immunological testing, including CD4+ T-cell counts or immunoglobulin levels, which might have helped define the degree of immunosuppression and clarified the patient's predisposition to opportunistic infections.Furthermore, the absence of comprehensive virological testing, including for SARS-CoV-2 and other respiratory viruses, limits the ability to definitively exclude the possibility of a viral co-infection or antecedent viral illness contributing to the patient's clinical deterioration and susceptibility to opportunistic infection.

Compounding these limitations was the absence of standardized molecular diagnostics such as PCR, qPCR, and Beta-D-glucan testing, which are now regarded as critical tools for confirming P. jirovecii infection. In our setting, the diagnosis instead relied on a pragmatic, integrative clinical approach, combining the patient's progressive hypoxemia, COPD history, recent AIHA diagnosis, and rapid clinical decline. Although CT is often a supportive imaging modality for PCP, this patient lacks the characteristic radiological features likeground-glass opacities, increased interstitial markings etc, making the clinical decision-making process even more complex [32]. Nonetheless, empirical treatment for PCP was initiated, reflecting a real-world, resource-constrained approach where high clinical suspicion must sometimes override the absence of definitive diagnostic confirmation. This strategy, though imperfect, aligns with best practices in such settings, where delayed treatment can lead to rapid deterioration and high mortality.Notably, mortality in severe PCP remains alarmingly high among non-HIV immunocompromised patients, exceeding 50% in ICU-managed cases and surpassing 20% even among severe cases outside the ICU, underscoring the stakes of timely recognition and intervention [33]

The incidence of PcP is increasing in an ever-diverse at-risk population, which will continue to expand with advancements in clinical care. While guidelines are available for the management of well recognized at-risk cohorts, there is a growing need for guidance in other patient populations [34]. Conducting rigorous and comprehensive assessments of patients undergoing B cell-targeted immunotherapy. These measures are essential to enhancing therapeutic efficacy while ensuring patient safety and quality of life [35,36].

In conclusion, this case underscores the complex interplay between chronic pulmonary disease, autoimmune pathology, and opportunistic infection in elderly, non-HIV immunocompromised host. The convergence of COPD, AIHA, and suspected PCP illustrates how layered immunological vulnerabilities, both disease-related and iatrogenic, can predispose patients to severe opportunistic infections, even in the absence of classic risk factors. The clinical trajectory, marked by rapid deterioration and ICU-level care, highlights the importance of early recognition and empirical management of PCP in similar contexts. Moreover, the lack of microbiological confirmation reflects a broader diagnostic challenge in resource-constrained settings, emphasizing the need for practical and accessible tools for immune profiling and fungal diagnostics. Our case advocates for heightened clinical vigilance, broader differential diagnoses, and investment in surveillance strategies to improve outcomes in non-HIV patients with atypical or overlapping immunocompromising conditions.

Abbreviations

PCP: Pneumocystis jirovecii pneumonia; HIV: Human Immunodeficiency Virus; ART: Antiretroviral Therapy; TMP-SMX: Trimethoprim-Sulfamethoxazole; COPD: Chronic Obstructive Pulmonary Disease; AIHA: Autoimmune HaemolyticAnaemia; LDH: Lactate Dehydrogenase; RBC: Red Blood Cells; PRBCs: Packed Red Blood Cells; IV: Intravenous; ICU: Intensive Care Unit; GMS: Grocott's Methenamine Silver (stain); qPCR: Quantitative Polymerase Chain Reaction; BAL: Bronchoalveolar Lavage; HRCT: High-Resolution Computed Tomography; G6PD: Glucose-6-Phosphate Dehydrogenase; CPIC: Clinical Pharmacogenetics Implementation Consortium.

Author Contributions

G.U.: writing - original draft, review and editing, data curation,A.S.: Investigation, data curation, writing - review and editing, D.P.S.:data curation,writing - review and editing, R.Y.: writing - review and editing, R.K.V.:conceptualization, data curation, writing - review and editing, NA: formatting, editing and plagiarism check . All authors have read and agreed to the published version of the manuscript.

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Ethics approval and consent to participate

Not applicable

Informed Consent Statement

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

Conflicts of Interest

There are no conflicts of interest.

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