

A case of *Pneumocystis jirovecii* pneumonia in an immunocompetent patient

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Case Report

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Abstract

Background

Pneumocystis jirovecii pneumonia (PJP) in immunocompetent hosts remains relatively rare and is not yet fully understood, posing unique challenges for clinical diagnosis and treatment.

Case Report

A 40-year-old immunocompetent female sales clerk working in a shopping mall clothing department presented with fever, chills, and generalized muscle soreness. She showed a poor response to initial treatment with quinolone antibiotics. Subsequent chest CT and electronic bronchoscopy were performed, and bronchoalveolar lavage fluid was collected. Gomori methenamine silver staining revealed Pneumocystis jirovecii (scanty). The patient was ultimately treated with trimethoprim-sulfamethoxazole tablets (0.8 g orally, four times daily), which alleviated her fatigue and muscle pain, leading to significant symptomatic improvement and discharge.

Conclusion

This case suggests that PJP may occur not only in immunocompromised patients but also in those with normal immune function. Importantly, this report further highlights that long-term exposure to relatively enclosed or toxic environments may cause injury to respiratory epithelial cells. Moreover, high-intensity work combined with persistent psychological stress can predispose individuals to chronic fatigue, leading to dysregulation of the neuro-endocrine-immune network and impaired local pulmonary immune defense.

Clinical Data

A 40-year-old female, employed as a clothing salesperson at a shopping mall, was admitted to our hospital on April 1, 2026, with a chief complaint of fever and chills for four days. The main clinical manifestations included chills and fever, with a maximum body temperature of 38.5°C, accompanied by generalized myalgia and occasional non-productive cough. The patient also experienced intermittent shortness of breath upon exertion, which resolved at rest. No nausea, vomiting, headache, or dizziness was reported. The patient had self-medicated with over-the-counter cold remedies without improvement, prompting presentation to our hospital.

Physical examination on admission: Body temperature 36.3°C, pulse rate 120 beats per minute, respiratory rate 21 breaths per minute, blood pressure 122/78 mmHg. The patient was alert and in good spirits, with supple neck. The chest was symmetric without deformity. Pulmonary percussion revealed clear notes bilaterally. Breath sounds were clear in both lungs, with no significant dry or moist rales auscultated. Cardiac and abdominal examinations revealed no positive findings. Neurological examination was unremarkable. No obvious edema was observed in the lower extremities. Personal history: The patient has one daughter and normal menstrual history. She has a 10-year history of rhinitis,

but during the course of disease she experienced no nasal congestion or rhinorrhea. There is no history of hypertension, diabetes mellitus, infectious diseases, surgery, or trauma. No food or drug allergies were reported. She has no smoking or alcohol consumption history, and no history of extramarital sexual contact. Chest computed tomography (CT) revealed bilaterally symmetric ground-glass opacities with partial consolidation, suggestive of *Pneumocystis jirovecii* pneumonia (formerly termed *Pneumocystis carinii* pneumonia; Fig. 1A and Fig. 1B). Electrocardiogram and echocardiography revealed no abnormalities. HIV test was negative, as were respiratory panel (six common pathogens) and SARS-CoV-2 nucleic acid tests. Routine blood tests and biochemical profiles were unremarkable. Upon admission, the initial diagnosis was “1. Bilateral pulmonary infection; 1.1 Viral pneumonia?” Antiviral therapy with oseltamivir phosphate (75 mg orally twice daily), anti-infective treatment with levofloxacin and sodium chloride injection (100 ml intravenously once daily), and symptomatic management including acetaminophen (0.3 g orally) for fever and fluid resuscitation were administered. However, the therapeutic response was poor. To further elucidate the etiology, electronic bronchoscopy was performed, which showed patent bilateral bronchi with a small amount of thin white secretions. Bronchoalveolar lavage was subsequently performed in the lateral segment of the right middle lobe. Bacterial culture of the bronchoalveolar lavage fluid was negative. Subsequent Gomori methenamine silver (GMS) staining revealed a small number of *Pneumocystis jirovecii* (Fig. 1C and Fig. 1D). Complete immunological workup, including immunoglobulin levels (IgG, IgA, IgM), rheumatoid factor, autoantibodies, and galactomannan (GM) test, were all negative. Peripheral blood T lymphocyte subset analysis (Table 1) revealed no significant abnormalities. Based on the clinical presentation and the above laboratory findings, the final diagnosis was established as *Pneumocystis jirovecii* pneumonia. The patient was treated with trimethoprim-sulfamethoxazole (TMP-SMX) at a dose of 0.8 g orally four times daily. After two days of treatment, fever resolved, and fatigue and myalgia subsided. The patient’s symptoms improved markedly, and she was discharged. She was instructed to continue the same regimen for an additional three weeks after discharge.

Table 1
Results of peripheral blood T lymphocyte subset analysis (%)

Parameter	Result	Reference range (adults)
CD3 + T cells / lymphocytes (%)	64	50–84
CD3 + CD4+ T cells / lymphocytes (%)	45	27–51
CD3 + CD8+ T cells / lymphocytes (%)	30	15–44
CD4 + T cells / CD8 + T cells ratio	1.95	1.4–2.5

Discussion

Analysis of causes of *Pneumocystis jirovecii* pneumonia (PJP) in immunocompetent hosts: PJP has traditionally been regarded as an opportunistic infection affecting individuals with acquired immunodeficiency syndrome (AIDS), those receiving long-term corticosteroid therapy, or patients on

immunosuppressive treatments. However, PJP can indeed occur in immunocompetent individuals, most commonly due to reactivation of latent infection, local host defense defects, or occult immune dysfunction.

1. Reactivation of latent infection

Pneumocystis jirovecii pneumonia (PJP) is ubiquitous in the environment. Most individuals acquire subclinical infection during childhood, and the pathogen can persist as a long-term latent reservoir in the lungs, contributing to a high rate of latent infection in the general population. Reactivation of latent infection is considered the primary mechanism underlying the development of PJP in immunocompromised patients [1]. Kushima et al. [2] further demonstrated in a retrospective study that the rate of airway colonization by Pneumocystis jirovecii was as high as 18.4% among HIV-negative patients who did not receive trimethoprim-sulfamethoxazole (TMP-SMX) prophylaxis, whereas prophylactic use of TMP-SMX completely eradicated colonization and prevented the development of pneumonia. This suggests that even in immunocompetent hosts, the presence of occult T-cell dysfunction, localized pulmonary defense defects, or subclinical immune abnormalities may still lead to reactivation of latent Pneumocystis organisms within the airways, resulting in overt disease. Therefore, in patients presenting with unexplained interstitial pneumonia and negative results for conventional pathogens, it is advisable to actively detect Pneumocystis jirovecii DNA using polymerase chain reaction (PCR) and to assess colonization status. For those with confirmed colonization or a high clinical suspicion of PJP, timely initiation of TMP-SMX therapy or prophylaxis can significantly improve outcomes.

2. Local host defense defects

Even in the absence of clear abnormalities in systemic immune function, impairment of local defense mechanisms in the respiratory tract may create conditions conducive to breakthrough infection with PJP. Pulmonary epithelial cells serve as the first line of defense against the invasion of various airborne pollutant particles [3] and also function as key regulatory cells in the process of airway mucus clearance. Through their functions of secretion, substance transport, and regulation of water–salt ion balance, pulmonary epithelial cells work in concert with ciliated cells and goblet cells, in conjunction with the mucus blanket structure, to establish the mucociliary clearance system (MCC), thereby exerting airway defense functions [4]. The mucociliary clearance system is a crucial innate defense mechanism of the lung, primarily composed of the protective mucus layer, the airway surface liquid layer, and the cilia structures on the surface of ciliated cells. Cilia, as specialized organelles, can expel inhaled particles and pathogenic microorganisms adhered to the mucus layer out of the airways through rhythmic beating [5]. Chronic smoking impairs airway mucociliary clearance function and reduces macrophage-dependent clearance capacity, thereby decreasing the efficiency of pathogen elimination from the lower respiratory tract [6]. Patients with chronic bronchitis, asthma, or chronic obstructive pulmonary disease (COPD) often exhibit airway structural remodeling, increased mucus secretion, and local immune microenvironment dysregulation [7]. These alterations not only facilitate colonization by Pneumocystis

organisms but also increase the risk of transition from colonization to overt infection. Following recent viral pneumonia (e.g., influenza, COVID-19, respiratory syncytial virus infection) or upper respiratory tract infection, the virus can induce transient T-cell dysfunction and alveolar epithelial barrier damage [8]. Even after resolution of the infection, local immune restoration may take weeks to months, a period that constitutes a “window of opportunity” for reactivation of *Pneumocystis* organisms. In addition, long-term exposure to air pollution, dust, or occupational hazards can induce chronic airway inflammation and impair mucosal immunity. Advanced age, frailty, malnutrition, and chronic fatigue further exacerbate the deterioration of local defense functions through systemic inflammaging, inadequate protein synthesis, and dysregulation of the neuro-endocrine-immune network [9, 10]. These factors often act in an additive or synergistic manner, placing the host in a “locally susceptible” state, which represents an important potential mechanism for the development of PJP in immunocompetent individuals.

3. Occult immune dysfunction

Some patients with so-called “immunocompetent” PJP actually have mild cellular immune deficits that are difficult to detect by routine laboratory tests. Certain individuals exhibit mild abnormalities in lymphocyte subsets, such as relatively low CD4 + T-cell counts (300–500/ μ L) or an inverted CD4+/CD8 + ratio, which may result from prior viral infections or genetic polymorphisms. These alterations are sufficient to shift *Pneumocystis jirovecii* from colonization to invasive disease [11]. In addition, a variety of underlying diseases can cause “occult immunosuppression.” For example, patients with poorly controlled diabetes mellitus exhibit defects in neutrophil chemotaxis and impaired T-cell proliferation. Chronic liver disease can lead to T-cell, natural killer (NK) cell, and macrophage dysfunction through cirrhosis-associated immune dysfunction (CAID). In patients with chronic kidney failure, accumulation of uremic toxins can suppress lymphocyte activity and induce immune cell apoptosis. Early or undetected malignancies may secrete immunosuppressive factors, creating a locally tolerant microenvironment [12]. Some undiagnosed autoimmune diseases (e.g., Sjögren's syndrome, early-stage systemic lupus erythematosus) are often associated with subclinical lymphocyte dysfunction, and the mortality rate associated with PJP in these patients remains relatively high, ranging from approximately 19% to 76% [13]. Collectively, the above factors can enable PJP to breach immune defenses and cause disease in otherwise “normal” hosts.

Conclusion

The patient in this case was a middle-aged female with no history of HIV infection, diabetes mellitus, hematologic disorders, or immune-related underlying diseases. Lymphocyte subset analysis revealed no abnormalities, and overall assessment indicated no systemic immunodeficiency. The patient had long been engaged in clothing sales in a relatively enclosed shopping mall environment, where volatile organic compounds released from new garments may cause injury to respiratory epithelial cells. In addition, high work intensity, prolonged occupational standing load, and sustained psychological stress predisposed the patient to chronic fatigue, leading to dysregulation of the neuro-endocrine-immune network and impairment of local pulmonary immune defense. The cumulative interplay of multiple risk

factors resulted in local host defense defects and occult immune dysfunction, ultimately culminating in opportunistic infection by *Pneumocystis jirovecii*.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the 940th Hospital of Joint Logistics Support Force of the Chinese People's Liberation Army (Approval No. 2025KYLL110). The study was conducted in accordance with the principles of the Declaration of Helsinki, and written informed consent was obtained from the patient's guardian.

Consent for publication

Written informed consent for the publication of this case report and related images was obtained from the patient. A copy of the consent document is available for review by the journal editors.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

D.G. and G.M. drafted the main manuscript. Y.X. and J.D. collected and analyzed the data. G.M. and Y.X. prepared the figures and tables. J.D. and Y.X. performed literature retrieval and revised the manuscript critically. All authors have read and approved the final manuscript.

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Availability of data and materials

The datasets generated and analyzed during this study are not publicly available, but can be obtained from the corresponding author upon reasonable request.

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Figures

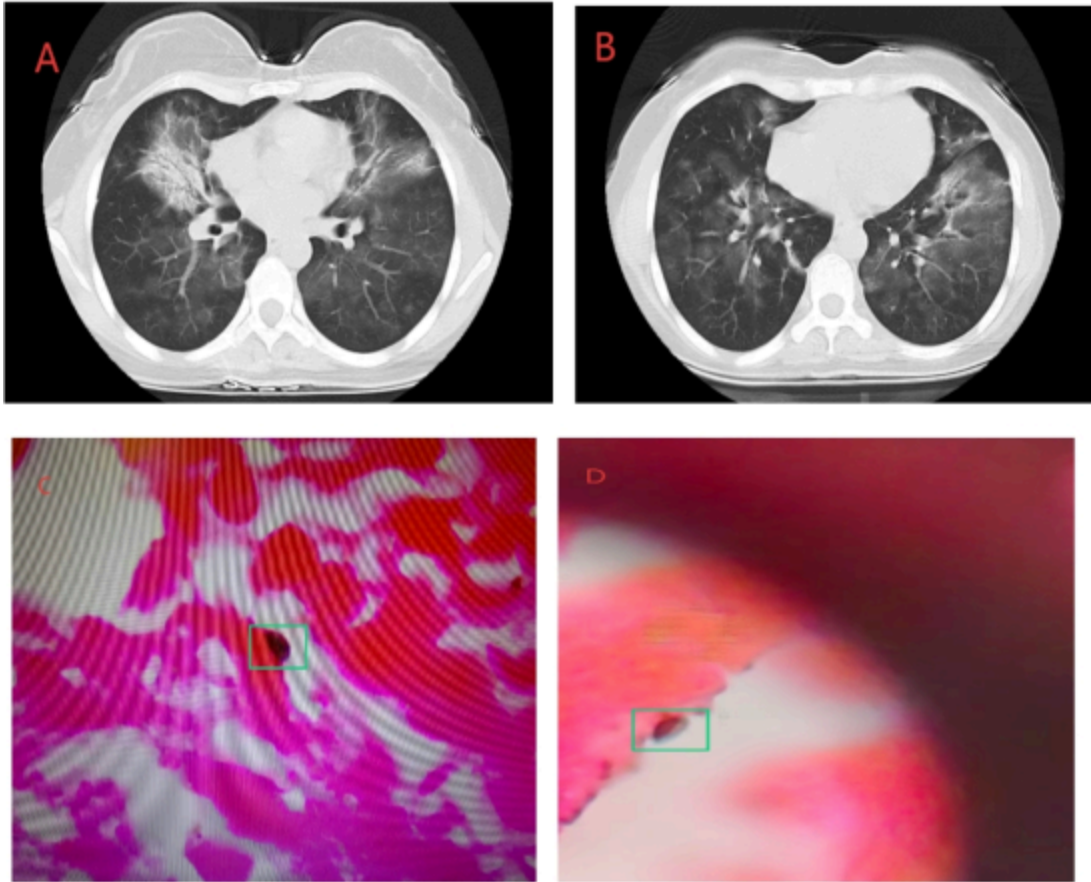


Figure 1

(A, B) Chest computed tomography (CT) showing bilaterally symmetric ground-glass opacities with partial consolidation. (C, D) Bronchoalveolar lavage fluid from the lateral segment of the right middle lobe; Gomori methenamine silver (GMS) staining reveals a small number of *Pneumocystis jirovecii* organisms.