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CASE REPORT

Invasive Pulmonary Aspergillosis in an Immunocompetent Patient Following Occupational Exposure to Iron Oxide Dust: A Long-Term Follow-Up Case Report

Chenwei Zhou[1], Leru Chao[2], Cui Guo[3], Yanjuan Zhou[1]

[1]Department of Respiratory and Critical Care Medicine, Wujin Hospital Affiliated with Jiangsu University, Changzhou, People's Republic of China; [2]Department of Pathology, Wujin Hospital Affiliated with Jiangsu University, Changzhou, People's Republic of China; [3]Department of Clinical Laboratory, Wujin Hospital Affiliated with Jiangsu University, Changzhou, People's Republic of China

Correspondence: Yanjuan Zhou, Department of Respiratory and Critical Care Medicine, Wujin Hospital Affiliated with Jiangsu University, Changzhou, People's Republic of China, Tel +86 13961165259, Email swallowjuan@163.com

Purpose: Invasive pulmonary aspergillosis (IPA) is a life-threatening fungal infection that predominantly occurs in immunocompromised hosts. However, IPA may also develop in immunocompetent individuals following substantial environmental or occupational exposure, though such cases are rare and often underrecognized. To our knowledge, there are limited reports of IPA associated with iron oxide dust exposure in otherwise healthy individuals, and long-term follow-up data are scarce.

Case Presentation: The patient was a 59-year-old male with no history of immunosuppression, who presented with cough, fever, and chest pain one week after heavy occupational inhalation of iron oxide dust. Initially misdiagnosed with bacterial pneumonia, he showed no improvement after empirical antibiotic therapy. Further investigation revealed purulent secretions on bronchoscopy; microbiological culture of sputum and bronchoalveolar lavage fluid grew *Aspergillus fumigatus*; serological tests showed elevated galactomannan (index 1.47) and β-D-glucan (359 pg/mL); and initial chest computed tomography(CT) demonstrated bilateral multisegmental infiltrates. Based on these findings, along with histopathological examination of lung tissue, the patient was diagnosed with invasive pulmonary aspergillosis. He was subsequently treated with voriconazole combined with caspofungin, along with a short course of glucocorticoids to modulate the inflammatory response. Follow-up CT at 10 years post-discharge showed complete radiological resolution of the pulmonary lesions.

Results: The patient's symptoms improved significantly after antifungal therapy. A follow-up chest CT at 10 years showed complete resolution of lung lesions without residual fibrosis or structural damage.

Conclusion: This case highlights that IPA should be considered in immunocompetent patients with significant occupational or environmental exposures, even in the absence of classic risk factors. Early diagnosis through combined microbiological, serological, and histopathological evaluation is crucial. Long-term follow-up demonstrates that favorable outcomes can be achieved with appropriate antifungal therapy and patient adherence.

Keywords: invasive pulmonary aspergillosis, immunocompetent, occupational exposure, iron oxide dust, long-term follow-up

Introduction

Aspergillus, a fungal genus ubiquitous in soil, decaying organic matter, household dust, building materials, ornamental plants, floral arrangements, food, and water,[1] is a frequent cause of pulmonary infections. Invasive pulmonary aspergillosis (IPA), which is caused by *Aspergillus* species, is a serious fungal infection associated with high morbidity and mortality rates, primarily affecting immunocompromised individuals. A recent global estimate indicates that over 2.1 million people develop IPA annually, with a crude mortality of approximately 1.8 million deaths.[2] The most common etiological agent is *Aspergillus fumigatus*, followed by *A. flavus* and *A. niger*.[3] IPA predominantly affects

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immunocompromised individuals, including those with prolonged neutropenia, hematological malignancies, solid organ or hematopoietic stem cell transplantation, acquired immunodeficiency syndrome (AIDS), chronic granulomatous disease, or those receiving prolonged high-dose corticosteroid therapy.[4] The incidence of IPA has been increasing, partly due to the increasing number of organ transplantations and expanded use of immunosuppressive therapies. The clinical presentation of IPA is highly variable and non-specific, making it challenging to diagnose.[5] Recent advances have improved our understanding of global epidemiology, diagnostic approaches, and treatment strategies for IPA. This study reports a case of IPA in an otherwise healthy male patient, with the aim of exploring the clinical characteristics, diagnostic pathways, and long-term outcomes of IPA in non-immunosuppressed hosts, to provide valuable insights for clinical management.

Case Presentation

A previously healthy 59-year-old male, who worked at a metal processing factory, presented with cough, fever (39°C), and pleuritic chest pain one week after substantial inhalation of iron oxide dust during grinding tasks without protective equipment. He denied any history of immunosuppression, diabetes, malignancy, or chronic lung disease. His medical history was unremarkable, with no prior episodes of recurrent infections or known allergies. On examination, he exhibited tachypnea and decreased breath sounds bilaterally, without cyanosis or clubbing. Laboratory investigations revealed a normal leukocyte count (6.96×10^9 /L; normal: $4.0–10.0 \times 10^9$ /L) with marked neutrophilia (86.5%), elevated C-reactive protein (301 mg/L; normal: <5 mg/L), procalcitonin (5.23 ng/mL; normal: <0.05 ng/mL), and erythrocyte sedimentation rate (80 mm/h; normal: 0–20 mm/h). Serological tests for human immunodeficiency virus (HIV), syphilis, hepatitis B/C, and autoantibodies (including antinuclear antibodies and anti-double-stranded DNA antibodies) were negative, ruling out immunosuppressive conditions. Initial chest computed tomography (CT) demonstrated bilateral multi-segmental infiltrates (Figure 1A and B). Empirical antibiotic therapy with moxifloxacin failed to improve symptoms, prompting further evaluation. Bronchoscopy revealed purulent secretions (Figure 2). Sputum smears and bronchial brushings showed true hyphae and oval spores. Cultures of both sputum and bronchoalveolar lavage fluid (BALF) yielded *Aspergillus fumigatus* (graded 2+), exhibiting velvety, light gray to grayish-green colonies with irregular edges. Lactophenol cotton blue staining revealed intertwined blue hyphae with spherical conidial heads bearing radially arranged spines and scattered conidia (Figure 3). Serological tests were positive, with serum galactomannan index of 1.47 (normal: <0.5) and β-D-glucan of 359 pg/mL (normal: <60 pg/mL). Histopathological examination of lung biopsy confirmed tissue invasion by septate hyphae with acute-angle branching consistent with *Aspergillus* species (Figure 4). Comprehensive immunological assessment, including HIV testing, immunoglobulin levels, lymphocyte subsets (CD3+: 72.28%; CD4+: 48.00%; CD8+: 22.38%; NK cells: 8.26%; B lymphocytes: 18.38% [reference: 5–18%]), confirmed an intact immune system with no humoral or autoimmune deficiencies. Based on integrated microbiological, serological, and histopathological findings, IPA was diagnosed. The patient was initially treated with oral voriconazole (loading dose of 400 mg every 12 hours, followed by 200 mg every 12 hours). However, his condition showed no significant improvement. Intravenous voriconazole was then promptly initiated (loading dose of 400 mg every 12 hours on day 1,

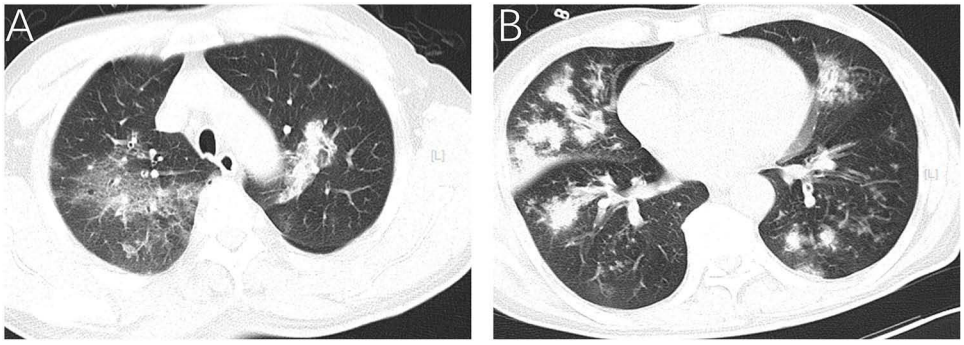

Figure 1 CT Multiple patchy consolidations with ground-glass opacities are seen in the bilateral upper lobes of the lungs (**A**). The middle and lower lobes show nodules and patchy increased-density opacities, with ill-defined borders and heterogeneous density. (**B**). [L] indicates the left side of the lung.

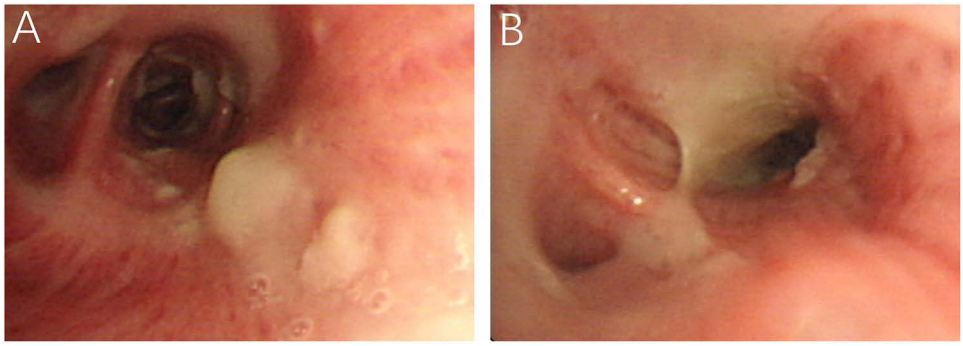

Figure 2 Tracheal mucosa with massive adherent purulent secretions, mild hyperemia and edema, suggesting airway inflammatory exudation (**A**). Partial obstruction from purulent secretion accumulation in the segmental bronchial lumen, consistent with invasive pulmonary aspergillosis (IPA)-related airway involvement (**B**).

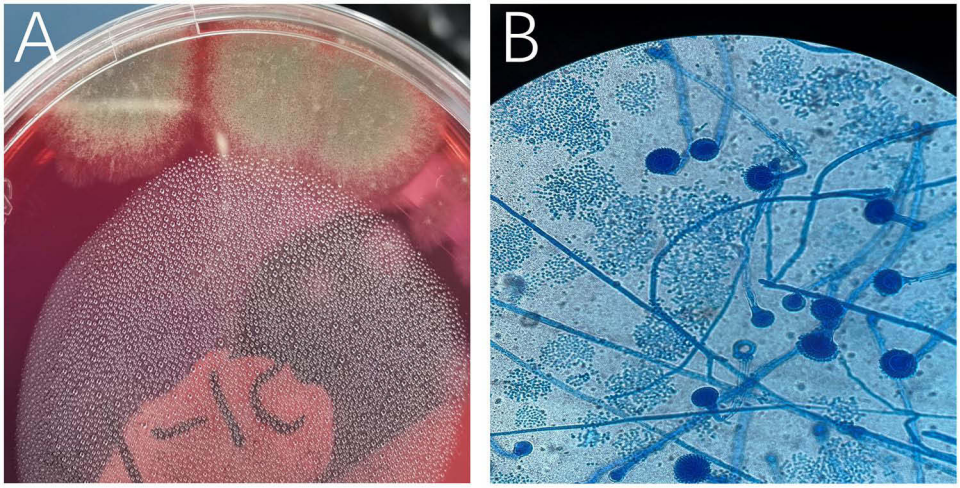

Figure 3 BALF culture Colonial morphology of the isolate on blood agar plate following incubation (**A**). Lactophenol cotton blue staining of the fungus, demonstrating hyphae and spores under light microscopy (×400) (**B**).

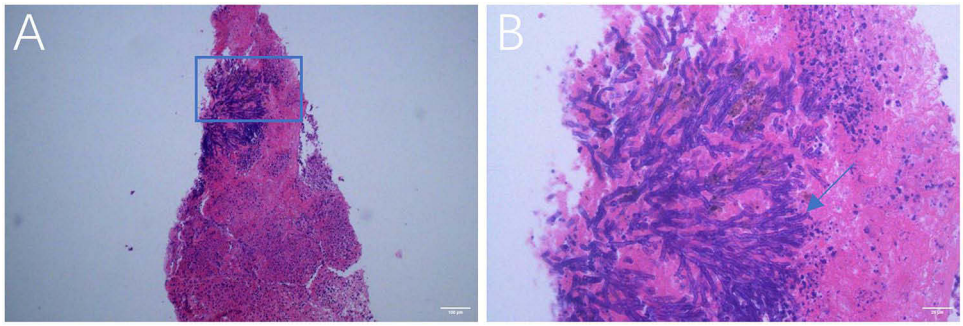

Figure 4 Hematoxylin-eosin staining Low-magnification view shows a focal aggregation of fungal hyphae (boxed area) (×100) (**A**). High-magnification view reveals septate hyphae with acute-angle branching, characteristic of *Aspergillus* invasion (Arrow) (b 400×) (**B**).

followed by 200 mg every 12 hours) in combination with caspofungin (70 mg loading dose, then 50 mg daily). Following this combination therapy, the patient's cough improved compared to before, although recurrent fever persisted. A short course of oral prednisone (15 mg twice daily, tapered over 2 weeks) was initiated to address suspected hyperinflammation. Clinical improvement was observed within 10 days, with resolution of fever and chest pain, and decreasing inflammatory markers (C-reactive protein from 301 mg/L to 121 mg/L; procalcitonin from 5.23 ng/mL to 0.33 ng/mL). The patient was discharged after 3 weeks with instructions to continue oral voriconazole (200 mg every 12 hours)

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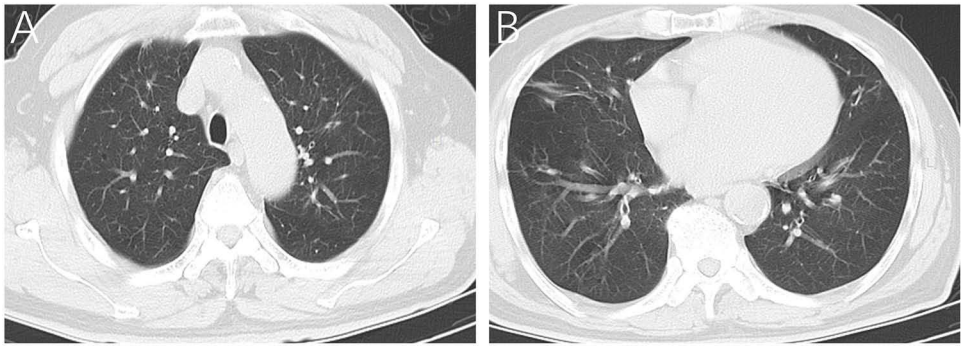

Figure 5 Chest CT at 10-year follow-up. The bilateral upper lung fields show normal lucency, with no evidence of patchy consolidations, ground-glass opacities, nodules, or fibrous streaks (**A**). The bilateral middle and lower lung fields also appear normal, with complete resolution of all previous lesions (**B**). [L] indicates the left side of the lung.

for 2 months, followed by itraconazole (200 mg twice daily) for 4 months. At the 6-month follow-up, symptoms had fully resolved, and serum markers normalized. Remarkably, a 10-year follow-up chest CT showed complete radiological resolution without residual fibrosis, cavitation, or bronchiectasis (Figure 5A and B).

Discussion and Conclusion

Aspergillus species are among the most common fungal pathogens causing pulmonary infections, with a particularly high incidence in immunocompromised patients, such as those undergoing chemotherapy for hematological malignancies, organ transplant recipients, and individuals on long-term corticosteroid therapy.[6] In this case, the patient had no prior history of immunodeficiency or high-risk factors such as viral infection. The only notable factor was significant exposure to iron oxide dust. This suggests that environmental exposure may play an important role in the development of atypical IPA in a non-immunocompromised host.[7,8] The mechanisms underlying IPA in immunocompetent individuals are complex and may involve environmental exposure, disruption of local defense mechanisms (eg, structural lung abnormalities, trauma, or surgery), subtle immune deficiencies or genetic predispositions (eg, congenital immune defects, acquired immune dysregulation), as well as fungal virulence and host–pathogen interactions (eg, highly virulent strains, host genetic polymorphisms). The temporal relationship between heavy iron oxide dust exposure and symptom onset suggests that it may have facilitated *Aspergillus* invasion by disrupting mucociliary clearance or inducing local inflammation. Consistent with this observation, two other case reports have also confirmed that non-immunocompromised individuals can develop IPA following high-concentration *Aspergillus* exposure: one involved a 67-year-old female who developed IPA after cleaning a damp old house,[9] and another reported a 45-year-old male waste recycler with IPA due to long-term residence in a dark and humid environment.[10] These cases collectively indicate that environmental exposure to high levels of *Aspergillus* spores—regardless of the specific exposure source (damp housing, waste recycling sites, or occupational dust)—can overcome the host's normal immune defenses and induce IPA.

The clinical presentation of IPA is often non-specific, primarily including cough, fever, and chest pain, which can be easily mistaken for bacterial pneumonia or pulmonary tuberculosis.[5] In non-immunosuppressed patients, diagnosis is often delayed due to lower clinical suspicion. The detection of *Aspergillus* in sputum cultures has limited diagnostic value, as it may represent colonization rather than true infection.[11] However, sputum cultures may still have some utility in immunosuppressed patients or other high-risk populations. The GM assay is an important serological marker for diagnosing IPA, although its utility is reduced in non-hematological patients.[12] In contrast to serum detection, GM assay in BALF shows significantly higher diagnostic sensitivity, particularly in patients with underlying pulmonary diseases such as chronic obstructive pulmonary disease or bronchiectasis.[13] However, bronchoscopy is often contraindicated in patients with IPA due to severe hypoxemia or thrombocytopenia. Recent studies suggest that GM testing on non-invasive proximal airway samples, such as induced sputum and tracheal aspirates, may be more sensitive than BALF testing, as it allows better recovery of *Aspergillus* antigen aggregates.[14]

The (1,3)-β-D-glucan (BDG) has high sensitivity but low specificity for fungal infections and cannot differentiate between fungal species; it is therefore recommended to combine BDG with GM testing to improve diagnostic accuracy.[3]

However, BDG testing in BALF has limited diagnostic value and is not recommended as a routine diagnostic method.[15] Imaging plays an important role in the diagnosis of IPA, although findings are often non-specific. Chest CT commonly shows patchy or nodular opacities, cavitary lesions, or both; however, the halo sign and air crescent sign are observed in fewer than 20% of cases.[16] Recently, immunoPET/MR imaging using an *Aspergillus fumigatus*-specific monoclonal antibody (mAb JF5), has shown promising specificity in murine models, effectively distinguishing aspergillosis from bacterial infections or inflammatory responses, though clinical applications require further investigation.[17] Additionally, blood-based polymerase chain reaction (PCR) methods are not widely used clinically due to their high cost and complex sample processing.[18] Currently, histopathological demonstration of fungal hyphal invasion remains the gold standard for diagnosis.[19]

Notably, false negatives in conventional diagnostic tests are common in non-immunocompromised patients with invasive pulmonary aspergillosis (IPA), further increasing the difficulty of diagnosis. For example, in a case reported by Bu et al,[9] sputum culture, bronchoalveolar lavage fluid (BALF) culture, serum (1,3)-β-D-glucan (BDG) assay, and BALF galactomannan (GM) assay were all negative. The diagnosis was ultimately confirmed by CT-guided percutaneous lung biopsy, which revealed free *Aspergillus* masses and septate hyphae. Similarly, in the patient described in Ao et al's study,[10] sputum culture and serum BDG assay were negative, and the diagnosis was established based on the identification of *Aspergillus* in BALF and detection of *Aspergillus* by next-generation sequencing (NGS). These cases, together with the patient in the present study (who had negative initial bacterial cultures and required a combination of sputum/BALF culture, serological testing, and histopathological examination for diagnosis), collectively emphasize that no single test is sufficient for diagnosing IPA in non-immunocompromised hosts. A comprehensive diagnostic approach integrating environmental exposure history, dynamic imaging, multi-site microbial detection, and histopathological examination is crucial.

Aspergillus species were isolated from both the sputum culture and bronchoalveolar lavage fluid (BALF) culture of this patient, and both the (1,3)-β-D-glucan (BDG) test and galactomannan (GM) test were positive. In addition, the patient's C-reactive protein and procalcitonin showed no significant decrease during the initial antibacterial treatment, but exhibited a marked downward trend after the initiation of antifungal therapy. Although procalcitonin is typically associated with bacterial infections, it may also be elevated in IPA, particularly in critically ill patients.[20] In the present case, the initial elevation likely reflected a robust inflammatory response to fungal invasion, while its decline following antifungal therapy indicated control of the infectious process. Combined with clinical symptoms, imaging findings, and a clear history of iron oxide dust exposure, the patient was diagnosed with invasive pulmonary aspergillosis (IPA). Subsequent lung biopsy confirmed that the infectious pathogen was *Aspergillus fumigatus*, further clarifying the diagnosis.

Regarding treatment, voriconazole has replaced amphotericin B as the first-line therapy for IPA.[21] Isavuconazole and posaconazole have shown non-inferior efficacy to voriconazole with better safety profiles. However, voriconazole remains a preferred agent due to extensive clinical experience, guideline recommendations, and cost-effectiveness.[22] A notable advantage of isavuconazole is that it does not require therapeutic drug monitoring, unlike voriconazole and posaconazole, suggesting potential for broader use.[23] When azoles are contraindicated or not tolerated, lipid formulations of amphotericin B may be used.[18]

The choice of voriconazole as the core therapy in our case aligns with its status as the first-line agent in clinical guidelines, which is also supported by other case reports: both Bu et al and Ao et al used voriconazole as the primary antifungal drug, with clinical improvement observed in the early stage of treatment.[9,10] However, Ao et al also noted that monotherapy with voriconazole may be insufficient for severe IPA—their patient required the addition of caspofungin to achieve symptom control,[10] which is consistent with our decision to switch to voriconazole-caspofungin combination therapy when the patient's lesions progressed despite initial voriconazole monotherapy. This suggests that combination antifungal therapy may be a viable option for non-immunocompromised IPA patients with suboptimal response to monotherapy, even though guidelines typically do not recommend combination therapy as first-line treatment.

The use of glucocorticoids in IPA remains controversial. Following *Aspergillus* infection, glucocorticoids may increase the risk of infection dissemination due to their immunosuppressive effects. In this case, the patient had a persistent fever, and antifungal therapy with voriconazole and caspofungin did not lead to significant improvement,

suggesting a potential excessive inflammatory response. For non-immunosuppressed patients with invasive pulmonary fungal disease(IPFD) who develop an excessive inflammatory response during effective antifungal therapy, adjunctive treatment with prednisone/prednisolone can be considered while continuing active antifungal therapy.[24] Therefore, a short course of prednisonewas administered to control the excessive inflammatory response and was gradually tapered once the fever was controlled. Special attention should be paid to interactions drug interactions when combining glucocorticoids with antifungal agents: for instance, dexamethasone may induce CYP3A4 activity, the enzyme responsible for voriconazole metabolism, potentially reducing voriconazole plasma levels and leading to treatment failure.[25] Therefore, therapeutic drug monitoring of voriconazole is recommended during co-administration to ensure efficacy and minimize interaction risks.

Drug interactions involving voriconazole require particular vigilance, as demonstrated by a case reported by Chen et al: a patient with Philadelphia chromosome-positive acute lymphoblastic leukemia developed pulmonary arterial hypertension after co-administration of voriconazole and reduced-dose dasatinib.[26] This was attributed to voriconazole's inhibition of CYP3A4, which increased dasatinib plasma concentrations despite dose reduction. This case, together with the potential interaction between voriconazole and glucocorticoids in our patient, highlights that voriconazole—while effective—has a high risk of pharmacokinetic interactions with other drugs. Therapeutic drug monitoring is not only necessary for combination with glucocorticoids but also for any co-administration with CYP3A4 substrates (such as dasatinib), inhibitors, or inducers, to balance efficacy and safety.

Another critical lesson from previous cases is the importance of treatment adherence for long-term prognosis. Ao et al reported a fatal outcome: their patient was discharged with oral voriconazole but discontinued the drug irregularly, leading to disease recurrence and death 1 week later.[10] In contrast, our patient achieved complete resolution of lesions at 10-year follow-up, which may be attributed to strict adherence to the prescribed antifungal regimen and regular follow-up. This contrast underscores that even with effective initial treatment, non-adherence can negate therapeutic gains, and long-term management (including patient education on medication adherence) is integral to improving IPA prognosis.

This case highlights the importance of considering the diagnosis and treatment of IPA even in patients without classic immunosuppressive risk factors. Future studies should further explore the relationship between environmental exposures and the pathogenesis of IPA, as well as optimize the clinical use of non-invasive diagnostic techniques.

Institutional Review Board Statement

This study was approved by the Ethics Committee of Wujin Hospital Affiliated with Jiangsu University (Approval No. 2025-SR-245). Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The committee confirmed that the study complied with the Declaration of Helsinki.

Abbreviations

IPA, Invasive pulmonary aspergillosis; CT, Computed tomography; BALF, bronchoalveolar lavage fluid; BDG,(1,3)-β-D-glucan; PCR, polymerase chain reaction.

Data Sharing Statement

The original contributions presented in the study are included in the article; further inquiries can be directed to the corresponding author.

Informed Consent Statement

Written informed consent for publication of their details was obtained from the patient.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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