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Severe COVID-19 Pneumonia, Opportunistic *Candida krusei* Infection, and Acute Respiratory Distress Syndrome with Pulmonary Arterial Hypertension Treated with Bosentan: A Case Report

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 73-year-old
Final Diagnosis: Pulmonary hypertension • syndrome (CARDS) • *Candida krusei* infection
Symptoms: Acute respiratory failure
Clinical Procedure: —
Specialty: Critical Care Medicine • Infectious Diseases
Objective: Unusual clinical course

Background: Despite global vaccination efforts, COVID-19 still necessitates effective treatments for severe cases that can quickly escalate to life-threatening complications, such as acute respiratory distress syndrome (ARDS) and secondary pulmonary arterial hypertension (PAH). Here, we present the clinical journey of a 73-year-old Ecuadorian man who developed severe COVID-19 pneumonia complicated by an opportunistic *Candida krusei* infection and ARDS, subsequently progressing to long-term PAH, managed with bosentan, an endothelin 1 (ET-1) antagonist.

Case Report: The patient, vaccinated with 2 doses of CoronaVac, experienced severe COVID-19 complications, including ARDS and secondary PAH, further complicated by a *C. krusei* infection. Despite prompt mechanical ventilation and intensive care, his condition rapidly deteriorated. Clinical evaluation confirmed COVID-19-associated ARDS, secondary PAH, and *C. krusei* infection through bronchoalveolar lavage. The therapeutic approach combined bosentan (125 mg twice daily) with dual antifungal therapy, leading to significant stabilization and eventual discharge. Post-discharge assessments showed persistent cardiopulmonary dysfunction, consistent with post-COVID-19 syndrome.

Conclusions: This case highlights critical COVID-19 complications in a vaccinated patient. While vaccination may provide substantial protection, COVID-19 pneumonia treated with corticosteroids can increase the risk of opportunistic infections like *C. krusei*, and ARDS can lead to pulmonary fibrosis and PAH. This case underscores the need for research on therapeutic strategies for complex COVID-19 cases and emphasizes comprehensive, personalized care for managing COVID-19 complications and sequelae.

Keywords: Bosentan • COVID-19

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Introduction

Pulmonary hypertension encompasses a spectrum of conditions characterized by elevated pressure in the pulmonary vasculature, leading to compromised lung function and increased cardiac strain [1]. The 6th World Symposium on Pulmonary Hypertension redefined pulmonary hypertension as a mean pulmonary arterial pressure exceeding 20 mmHg, coupled with pulmonary vascular resistance of 3 or more Wood units for pre-capillary pulmonary hypertension forms [2].

The emergence of COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has fundamentally transformed our understanding and management of pulmonary hypertension. SARS-CoV-2 primarily targets the respiratory system through the angiotensin-converting enzyme 2 receptor expressed in lung tissue, including alveolar type II cells and endothelial cells [3]. This interaction triggers the angiotensin II and angiotensin 1-7 pathways, leading to dysregulated pulmonary constriction and inflammatory responses [3].

Of particular concern, patients with COVID-19 face increased susceptibility to secondary opportunistic infections, including fungal pathogens such as *Candida krusei*. This pathogen exhibits multidrug resistance, predominantly affecting immunocompromised individuals and necessitating specific antifungal treatment, due to inherent fluconazole resistance [4]. Diagnostic confirmation requires blood cultures, while management often includes echinocandins or voriconazole [5].

COVID-19 pneumonia manifests with respiratory distress, hypoxemia, and bilateral infiltrates on imaging, potentially progressing to ARDS. The presence of opportunistic infections exacerbates these issues, creating complex management challenges, particularly in patients on corticosteroids or broad-spectrum antibiotics, which increase susceptibility to fungal infections [6].

ARDS, a frequent complication of viral pneumonia, is characterized by widespread alveolar-capillary damage, non-cardiogenic pulmonary edema, and hypoxemia. Current management strategies prioritize lung-protective ventilation and, in severe cases, can necessitate extracorporeal membrane oxygenation [7].

The pathophysiology of PAH encompasses complex interactions among chronic lung conditions, cardiac dysfunction, and immune responses, particularly relevant in infections such as SARS-CoV-2. Definitive evaluation of PAH requires right heart catheterization, complemented by echocardiography and pulmonary function tests [8]. Contemporary PAH management incorporates vasodilators like endothelin receptor antagonists, notably bosentan, which selectively blocks ET-1 receptors in pulmonary vessels, thereby reducing vascular constriction and remodeling [9]. Clinical evidence suggests that the dual-targeting mechanism of bosentan offers promising therapeutic potential for COVID-19-associated PAH [9].

This report describes a 73-year-old man with severe COVID-19 pneumonia, opportunistic infection with *C. krusei* and ARDS who developed long-term PAH requiring treatment with bosentan, an ET-1 antagonist.

Case Report

A 73-year-old man presented to the Emergency Department with acute respiratory distress. Despite previous immunization with 2 doses of CoronaVac vaccine (Sinovac Biotech, Beijing, China), with the last dose administered on July 31, 2021, and a medical history significant for hypertension, the patient developed severe COVID-19 symptoms.

Initial clinical evaluation revealed marked respiratory distress, with significant hypoxemia (SpO2 86% on room air), tachypnea, fever, and tachycardia. Immediate oxygen supplementation via nasal cannula at 3 L/min improved oxygen saturation to 90%. Laboratory evaluation demonstrated significant hematological alterations, including leukopenia (white blood cells: 4160 cells/mm^3), lymphopenia (lymphocytes: 350 cells/mm^3), neutrophilia (neutrophils: 3490 cells/mm^3), and anemia (hemoglobin: 10.5 g/dL, hematocrit: 31.5%). Additional findings included renal function impairment (urea: 88 mg/dL, creatinine: 2.13 mg/dL) and hepatic dysfunction (aspartate aminotransferase: 54 U/L, alanine aminotransferase: 36 U/L). Inflammatory markers were notably elevated: lactate dehydrogenase (268 U/L), IL-6 (222.7 pg/mL), D-dimer (1.17 µg/mL), ferritin (1556 ng/mL), and procalcitonin (1.06 ng/mL).

Arterial blood gas analysis demonstrated pH 7.4, pCO2 29.7 mmHg, pO2 79 mmHg, HCO3 18.6 mmol/L, and SaO2 95.9% on supplemental oxygen. Initial chest radiography revealed bilateral ground-glass opacities affecting 50% of lung parenchyma (**Figure 1A**). Clinical deterioration on day 2 necessitated escalation to high-flow nasal cannula (45 L/min, FiO2 50%), maintaining SaO2 at 98%. Initial echocardiographic assessment indicated mild pulmonary hypertension.

Disease progression was documented by chest computed tomography on day 3, revealing extensive bilateral ground-glass opacities and consolidation affecting 75% of lung parenchyma, predominantly in peripheral distribution across upper and lower lobes (**Figure 1B**). The patient experienced fluctuating oxygen saturation (85%-95%) over the subsequent 48 h. Despite initiation of dexamethasone therapy, progressive respiratory failure necessitated Intensive Care Unit (ICU) transfer for mechanical ventilation on day 6.

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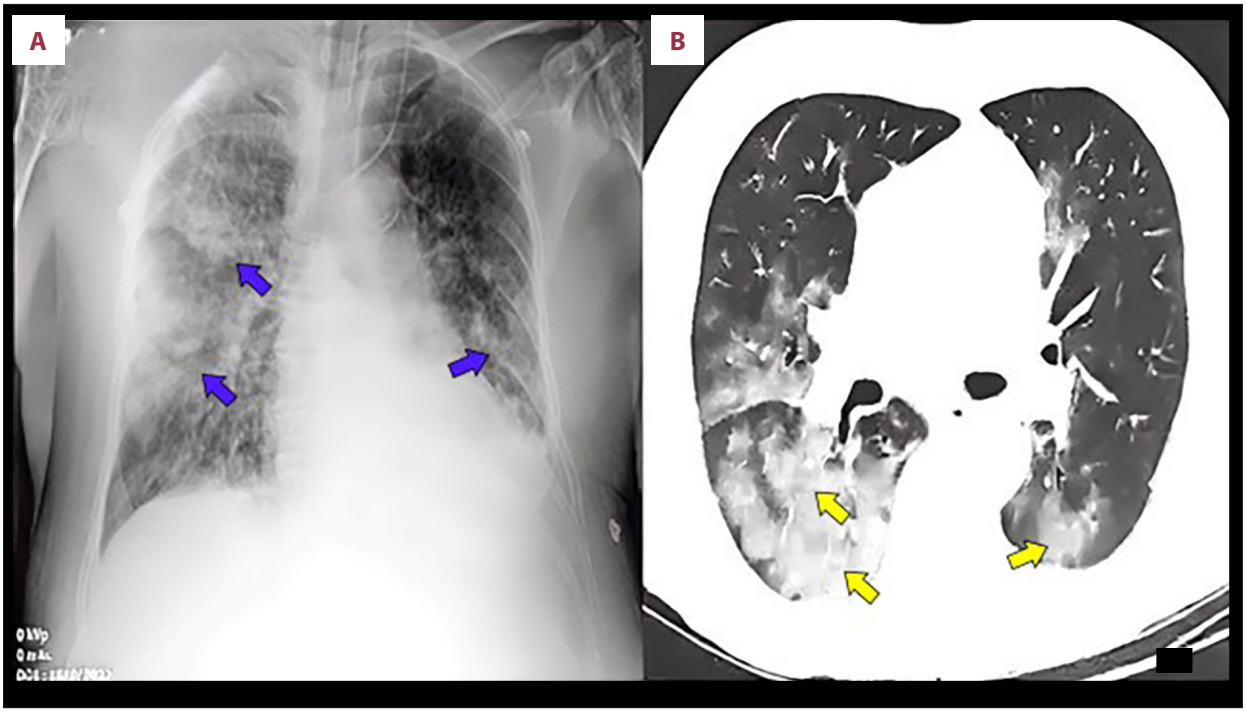

Figure 1. Radiological progression of COVID-19 pneumonia. (A) Anteroposterior chest radiograph demonstrating extensive bilateral infiltrates with ground-glass appearance (blue arrows), affecting approximately 50% of lung fields, characteristic of early COVID-19 pulmonary involvement. (B) Chest computed tomography scan revealing progression to 75% parenchymal involvement, characterized by peripheral consolidations (yellow arrows) and ground-glass opacities (green arrows), typical of severe COVID-19 pneumonia.

Table 1. Sequential cardiac function and bosentan therapy monitoring. Chronological documentation of cardiac parameters and therapeutic adjustments, including pulmonary pressures, ventricular measurements, and bosentan dosing strategy.

Day	Event	Ejection fraction (%)	Right ventricular diameter (mm)	PASP (mmHg)	Bosentan (mg)
1	Admission and initial assessment	64	28	36	–
2	Deterioration and change in support	61	36	45	–
6	Initiation of invasive mechanical ventilation	59	38	70	–
7	Continued invasive mechanical ventilation	59	38	55	125
9	Weaning from invasive mechanical ventilation	62	37	54	125
10 to 15	After extubation	61	35	40	125
20	Further improvement	62	36	40	62.5
26	Discontinuation of bosentan	61	36	45	62.5
30	Initiation of pulmonary rehabilitation	69	34	38	–
51	Discharge from hospital	67	31	28	–

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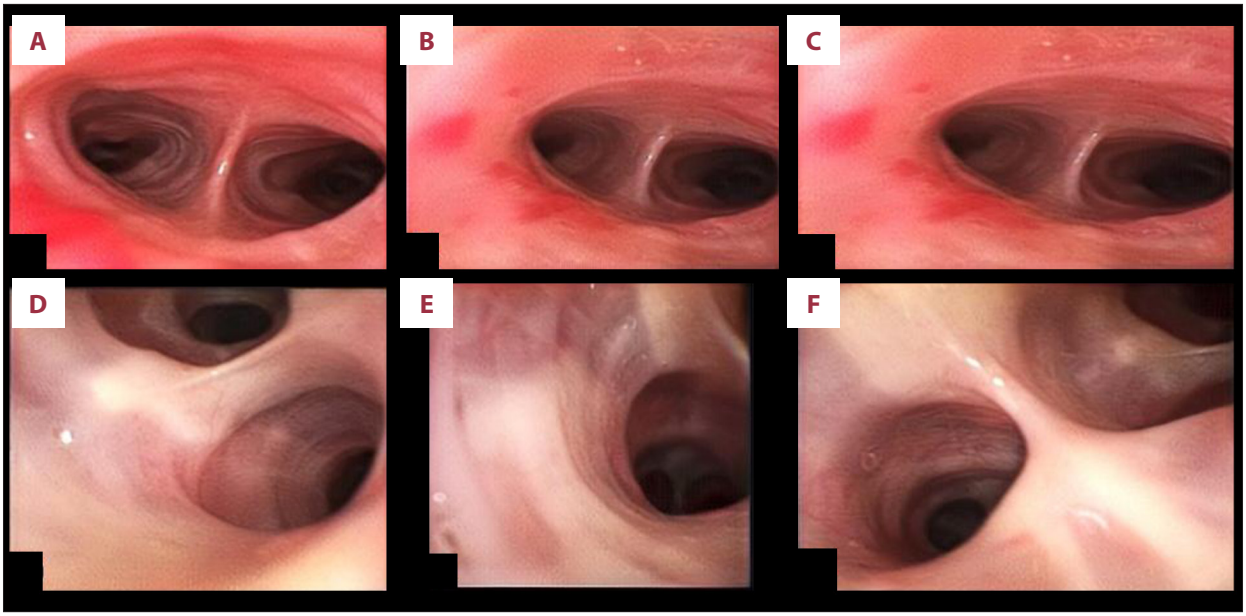

Figure 2. Bronchoscopic evaluation during active COVID-19 and fungal co-infection. Sequential bronchoscopic findings revealing: (**A**) inflammatory changes with hemorrhagic foci in bronchial mucosa; (**B**) Mucosal fold hypertrophy with characteristic fungal plaques; (**C**) active purulent secretions in segmental airways; (**D**) confirmed *Candida krusei* colonization presenting as whitish plaques; (**E**) Mucosal edema with epithelial disruption; and (**F**) enhanced vascular patterns reflecting inflammatory response.

Subsequent echocardiographic evaluation on day 7 revealed preserved left ventricular ejection fraction (61%) with elevated pulmonary artery pressure (45 mmHg; **Table 1**). Radiological evidence suggested nosocomial pneumonia, prompting initiation of broad-spectrum antimicrobial therapy (carbapenem with linezolid). Bronchoscopic evaluation (**Figure 2**) identified *C. krusei* infection, resistant to fluconazole but susceptible to caspofungin and voriconazole, leading to implementation of dual antifungal therapy for 14 days. Genomic analysis identified SARS-CoV-2 clade 22B (Omicron), lineage BA.5.2.

Echocardiographic confirmation of secondary pulmonary arterial hypertension (PAH) on day 9 prompted initiation of bosentan therapy. Following 5 days of mechanical ventilation support, successful extubation was achieved on day 15. Post-extubation echocardiography demonstrated stable cardiac function (ejection fraction 61%, right ventricular diameter 36 mm, pulmonary artery pressure 45 mmHg). SARS-CoV-2 RT-PCR negativity was documented on day 17.

Clinical improvement, particularly in pulmonary hypertension parameters, was observed by day 20. Bosentan therapy (125 mg twice daily) was maintained until day 26, when clinical stability permitted its discontinuation. Pulmonary rehabilitation was initiated on day 30, facilitating ICU discharge on day 34. Cardiac function showed significant improvement by day 39 (ejection fraction 69%, right ventricular diameter 32 mm, pulmonary artery pressure 35 mmHg).

The patient was discharged after 51 days of hospitalization. Two-month follow-up evaluation revealed persistent restrictive ventilatory defect and small airway dysfunction, confirmed by spirometry and nitrogen washout technique. Diffusion testing demonstrated impaired gas exchange, while cardiopulmonary exercise testing identified reduced exercise capacity and persistent cardiopulmonary dysfunction (**Table 2**). **Figure 3** illustrates the temporal relationship between SARS-CoV-2 infection, the development of severe pulmonary hypertension, and the therapeutic interventions leading to clinical recovery.

Discussion

This case presents 3 significant clinical challenges in COVID-19 management: secondary pulmonary hypertension following coronavirus-induced acute respiratory distress syndrome, opportunistic *C. krusei* infection, and post-COVID sequelae in a vaccinated individual [10].

The development of pulmonary hypertension in COVID-19 involves complex pathophysiological mechanisms. SARS-CoV-2 infection triggers endothelial dysfunction, vascular permeability alterations, and microvascular thrombosis, leading to increased pulmonary vascular resistance [11]. In the present case, bosentan administration demonstrated clinical improvement in secondary PAH, potentially through its dual antagonism of endothelin A and B receptors [12]. While this therapeutic response suggests potential clinical applications, controlled studies

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remain necessary to establish treatment efficacy in COVID-19-related PAH and identify optimal patient selection criteria.

The clinical course in this patient contributes to emerging evidence supporting drug repurposing strategies in COVID-19 complications [13]. Notably, the development of coronavirus-induced acute respiratory distress syndrome despite completed CoronaVac vaccination series highlights the ongoing challenge of variant escape, particularly with Omicron lineages [14]. This observation reinforces the necessity for continuous vaccine effectiveness monitoring and strategic booster protocols in high-risk populations [15].

The identification of *C. krusei* infection represents a critical therapeutic challenge in the present case. Although *C. krusei* relatively uncommon, this opportunistic pathogen carries substantial mortality risk in immunocompromised hosts [16]. The patient's COVID-19-induced immunological alterations, characterized by lymphopenia and cytokine dysregulation, likely created favorable conditions for fungal proliferation [17]. The successful clinical response to targeted antifungal therapy, guided by susceptibility testing, demonstrates the critical role of precision medicine approaches in managing COVID-19-associated opportunistic infections [18].

Follow-up evaluation at 2 months revealed persistent cardiopulmonary dysfunction characteristic of post-COVID-19 syndrome [19]. The documented restrictive pattern primarily reflected reduced lung volumes rather than air trapping or ventilatory heterogeneity. Exercise capacity limitations manifested through decreased aerobic capacity and ventilatory inefficiency, without primary ventilatory or cardiovascular limitations [20]. These findings align with established patterns of post-COVID pulmonary sequelae, potentially resulting from direct viral injury, sustained immunological responses, or persistent microvascular dysfunction [21].

Table 2. Comprehensive pulmonary function analysis. Detailed assessment including spirometric parameters demonstrating restrictive pattern; body plethysmography confirming volume restrictions; diffusion capacity indicating gas exchange impairment; and cardiopulmonary exercise testing revealing exercise limitations and ventilatory inefficiency.

Parameter	Unit	Pred.	Pre	%Pred	Post (L)	Post (%)	%Change
Spirometry							
Forced vital capacity (FVC)	L	3.80	2.85	75%	3.00	79%	5%
Forced expiratory volume in 1 second (FEV1)	L	3.10	2.45	79%	2.65	85%	8%
FEV1/FVC ratio	%	82	86	105%	88	107%	2%
Forced expiratory flow 25-75%	L/s	3.00	2.10	70%	2.40	80%	14%
Peak expiratory flow	L/s	7.50	6.80	91%	7.10	95%	4%
Body plethysmography (nitrogen washout)							
Total lung capacity (TLC)	L	5.60	4.50	80%	4.70	84%	4%
Vital capacity (VC)	L	3.90	3.00	77%	3.15	81%	5%
Residual volume (RV)	L	1.85	1.80	97%	1.75	95%	-3%
RV/TLC ratio	%	33	40	121%	37	112%	-7%
Functional residual capacity	L	2.80	2.55	91%	2.60	93%	2%
Expiratory reserve volume	L	1.30	0.90	69%	0.95	73%	6%
Inspiratory capacity	L	2.60	2.10	81%	2.20	85%	5%

Parameter	Unit	Pred.	Pre	%Pred	Post	%Change
Diffusion						
Diffusing capacity for carbon monoxide (DLCO)	mL/min/mmHg	30.0	21.0	70%	22.5	7%
Dlco adjusted for alveolar volume (DLCO/VA)	mL/min/mmHg/L	4.80	4.00	83%	4.20	5%
Alveolar volume	L	5.65	5.25	93%	5.35	2%
Transfer coefficient	mL/min/mmHg/L	5.30	4.00	75%	4.20	5%

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Table 2 continued. Comprehensive pulmonary function analysis. Detailed assessment including spirometric parameters demonstrating restrictive pattern; body plethysmography confirming volume restrictions; diffusion capacity indicating gas exchange impairment; and cardiopulmonary exercise testing revealing exercise limitations and ventilatory inefficiency.

Parameter	Unit	Pred.	Max	%Pred	on AT	%Max	On RCP
Cardiopulmonary exercise testing							
Load	W	125	111	89	73	66	65
Heart rate	1/min	147	130	88	120	92	125
Oxygen uptake (VO2)	ml/min	1540	635	41	565	89	630
Oxygen pulse (VO2/H)	ml/b	10.5	4.88	46	4.71	97	5.04
Carbon dioxide output (VCO2)	ml/min	2002	494	25	494	100	477
Respiratory exchange ratio		1.00	0.81	81	0.78	96	0.76
Minute ventilation (VE)	l/min	66.1	24.0	36	20.5	85	23.0
Breathing frequency	1/min	16.5	20.8	126	16.5	79	18.5
Systolic blood pressure	mmHg	160	143	89	120	84	120
Diastolic blood pressure	mmHg	80	60	75	80	133	80
Breathing reserve	%	70	72.1	103	76.2	106	73.3
Ventilatory equivalent for oxygen (VE/VO2)		30.0	34.9	116	31.8	91	31.7
Ventilatory equivalent for carbon dioxide (VE/VCO2)		30.0	47.8	159	44.1	92	44.5
Heart rate reserve	1/min	17	17	100	27	159	22

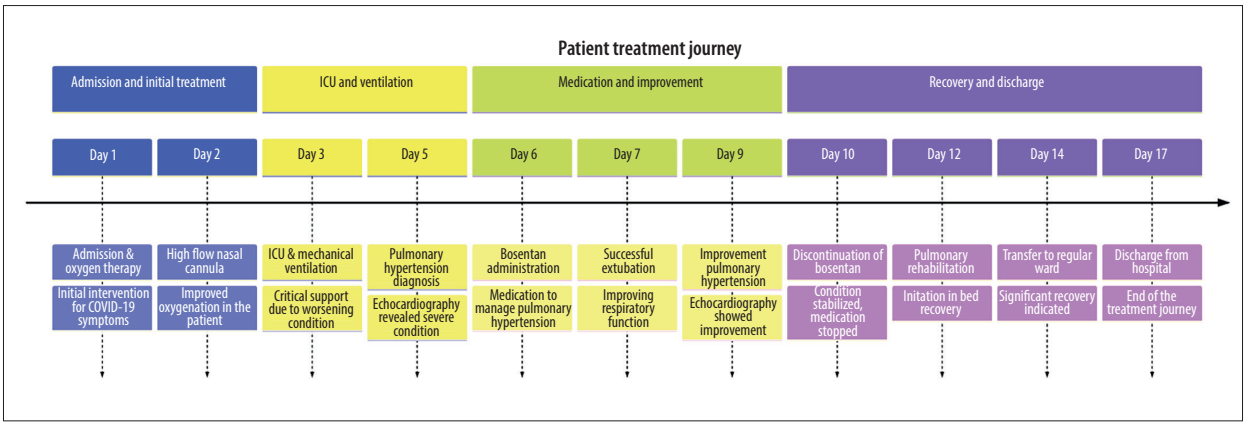

Figure 3. Temporal progression of clinical course and therapeutic interventions. Chronological documentation of disease progression, therapeutic interventions, and clinical responses in severe COVID-19 complicated by pulmonary hypertension and *Candida krusei* infection, highlighting key treatment milestones including bosentan initiation and antifungal therapy.

The underlying pathophysiology of post-COVID-19 syndrome encompasses ongoing inflammation, microvascular injury, and endothelial dysfunction, contributing to pulmonary fibrosis, vascular remodeling, and cardiac dysfunction [22]. In the present case, the patient's advanced age and pre-existing hypertension likely increased the susceptibility to prolonged symptoms and organ dysfunction, consistent with observed patterns in elderly populations with comorbidities [23].

This case exemplifies several critical aspects of complex COVID-19 management: the necessity for individualized therapeutic approaches, vigilant monitoring for opportunistic infections, comprehensive follow-up protocols for post-COVID sequelae, and strategic integration of novel therapeutic strategies with established treatment protocols. The therapeutic potential of targeting pulmonary endothelial dysfunction in COVID-19-related pulmonary hypertension is further supported by

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findings from Vives et al, who reported on a 36-year-old woman with COVID-19-induced acute pulmonary hypertension successfully treated with inhaled nitric oxide. In their case, oxygenation improved markedly, with the PaO2/FiO2 ratio increasing from 100 to 191 mmHg within the first 24 h, and further to 321 mmHg by day 7 of treatment at 15 ppm. This clinical improvement, along with the normalization of right ventricular function, resolution of tricuspid regurgitation, and sustained hemodynamic stability even after inhaled nitric oxide discontinuation, complements our experience with bosentan as an effective strategy for managing COVID-19-associated pulmonary complications. These findings underscore the need for robust, controlled trials to validate these therapeutic approaches, aiming to establish standardized protocols for broader patient application and benefit [24,25].

Conclusions

In conclusion, this case illustrates the multifaceted challenges in managing severe COVID-19 complicated by secondary pulmonary hypertension and opportunistic fungal infection. The implemented therapeutic strategy, combining bosentan for pulmonary hypertension management with targeted antifungal therapy, offers insights for future investigation. These clinical observations underscore the necessity for adaptive, evidence-based approaches in managing evolving viral challenges and their associated complications.

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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