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Paradoxical Role of Glucocorticoids in Severe *Pneumocystis jirovecii* Pneumonia Among Renal Transplant Recipients: Case Series

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Case series
Patients: —
Final Diagnosis: Acute respiratory distress syndrome (ARDS) • Pneumocystis jirovecii pneumonia
Symptoms: Dry cough • fever • progressive dyspnea
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Unusual or unexpected effect of treatment

Background: Severe *Pneumocystis jirovecii* pneumonia (PJP) in renal transplant recipients (RTRs) can rapidly progress to acute respiratory distress syndrome (ARDS) and is associated with high mortality. Glucocorticoids (GCs) play a paradoxical role, constituting a risk factor for infection and a trigger for immune reconstitution inflammatory syndrome upon withdrawal; they may also serve as a therapeutic agent for lung injury. We evaluated the efficacy of a standardized triple-therapy regimen designed to address this paradox.

Case Reports: We analyzed 7 RTRs admitted to the intensive care unit (ICU) with severe PJP-ARDS between June 2023 and September 2024. The cohort had a median age of 49 years; all patients had prior chronic low-dose GC maintenance therapy without PJP prophylaxis. All diagnoses were confirmed by metagenomic next-generation sequencing. After the onset of severe PJP-ARDS, all immunosuppressive agents were discontinued; patients were treated with trimethoprim-sulfamethoxazole and caspofungin. Early adjunctive intravenous methylprednisolone was administered to all patients, including 4 who received treatment upon ICU admission. The median starting dose was 80 mg/day (range, 40-120 mg/day), with a median treatment duration of 11 days (range, 5-17 days) and median cumulative dose of 580 mg (range, 200-840 mg). Following this triple-therapy regimen, the median duration of mechanical ventilation was 14 days, and the survival rate was 100% (7/7); no severe secondary infections or uncontrolled hyperglycemia occurred.

Conclusions: Despite constituting a predisposing factor for PJP, early adjunctive GC administration—combined with robust anti-*Pneumocystis* therapy—may be a safe and promising strategy for managing severe PJP-ARDS in RTRs.

Keywords: Glucocorticoids • *Pneumocystis carinii* • Pneumonia, Pneumocystis • Transplant Recipients

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952853

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Introduction

Renal transplantation remains the most effective treatment for end-stage renal disease. However, the long-term immunosuppressive regimens required after transplantation, particularly those containing glucocorticoids (GCs), greatly increase the risk of opportunistic infections. In particular, *Pneumocystis jirovecii* pneumonia (PJP) is a serious fungal infection among renal transplant recipients (RTRs) that substantially increases the risks of allograft dysfunction and mortality [1]. Although the overall incidence of PJP in solid organ transplant recipients ranges from 0.3% to 2.6% [2], the mortality rate of severe cases progressing to acute respiratory distress syndrome (ARDS) often exceeds 50% despite standard antimicrobial therapy [3].

GCs play a complex and paradoxical role in the pathophysiology and management of PJP, acting simultaneously as a risk factor, a potential trigger of clinical deterioration, and a therapeutic agent. First, as a cornerstone of maintenance immunosuppression, chronic GC administration increases PJP risk in a dose-dependent manner [4]. The concomitant use of other maintenance immunosuppressants, such as calcineurin inhibitors and mycophenolate derivatives, acts synergistically with GCs to profoundly suppress T-cell-mediated immunity. Such overall immunosuppression is a key driver of susceptibility to PJP in this population. Second, clinical observations indicate that many patients develop overt respiratory failure during GC tapering or after discontinuation of pulse therapy [5], suggesting that changes in immunosuppression can trigger disease progression through immune reconstitution inflammatory syndrome (IRIS). Third, adjunctive GC therapy is a standard component of care for human-immunodeficiency-virus (HIV)-associated PJP to reduce the risk of respiratory failure [6,7]. However, its efficacy and safety in non-HIV RTRs remain controversial and inadequately studied.

Here, we aimed to describe the clinical characteristics of RTRs with severe PJP-ARDS and to evaluate the efficacy and safety of a standardized combination treatment protocol comprising trimethoprim-sulfamethoxazole (TMP-SMX), caspofungin, and early adjunctive GC administration.

Case Reports

Patient Selection and Study Design

This report describes the clinical course of 7 RTRs admitted to the Department of Critical Care Medicine at The First Affiliated Hospital of Sun Yat-sen University (Nansha Campus) between June 2023 and September 2024. This case series included adult patients who developed severe pneumonia complicated by ARDS after kidney transplantation while receiving maintenance immunosuppression. In all cases, the diagnosis of PJP was confirmed by metagenomic next-generation sequencing (mNGS) of respiratory or blood samples, ensuring diagnostic consistency [8]. Severe PJP was defined by clinically significant hypoxemia, indicated by an alveolar-arterial oxygen gradient of at least 45 mm Hg or an arterial partial pressure of oxygen (PaO_2) below 60 mm Hg on room air [9].

Demographics and Clinical Characteristics

The cohort consisted of 6 men and 1 woman, with a median age of 49.0 years (range, 33-62 years). Infection timing considerably varied, occurring at a median of 5.0 years after transplantation; however, 2 patients developed infection within the first year. Six patients had underlying hypertension, and 1 had diabetes mellitus. At symptom onset, all patients were receiving a standard triple-drug immunosuppressive regimen consisting of GCs, calcineurin inhibitors, and mycophenolate derivatives, with a median prednisone-equivalent dose of 10 mg/day (**Table 1**). Importantly, no patients were receiving PJP chemoprophylaxis. One patient's clinical course was particularly notable: PJP-ARDS developed immediately after a course of high-dose pulse methylprednisolone (500 mg/day for 3 days) and plasma exchange for acute graft rejection.

The clinical presentation was acute and relatively uniform across the series. All patients presented with fever, frequently accompanied by progressive dyspnea (71.4%) and dry cough (42.9%). Clinical deterioration was rapid, comprising progression from initial respiratory symptoms to respiratory failure requiring intensive care unit (ICU) admission within a median of 6 days (**Table 1**).

Diagnosis and Severity Assessment

Upon admission to the ICU, all patients were critically ill. Clinical assessment revealed high disease severity scores, with a median Acute Physiology and Chronic Health Evaluation II (APACHE II) score of 19 and a median Pneumonia Severity Index (PSI) score of 128, placing most patients in high-risk classes IV or V. Radiological evaluation by chest computed tomography consistently demonstrated hallmark features of severe PJP, including bilateral diffuse ground-glass opacities and patchy consolidations.

Laboratory investigations highlighted profound immune depletion and systemic inflammation. Severe lymphopenia was observed (median, 0.3×10^9/L), accompanied by substantially elevated inflammatory markers, including C-reactive protein (median, 117.5 mg/L) and lactate dehydrogenase (median, 442 U/L). Serum 1,3-β-D-glucan levels were also elevated (median, 143.5 pg/mL). Definitive microbiological confirmation was achieved by mNGS in all cases, with *P. jirovecii* detected in bronchoalveolar lavage fluid from 6 patients and peripheral

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Table 1. Demographic and baseline characteristics of patients with severe PJP after renal transplantation.

Characteristics	All patients (N = 7)
Age (years)	49 (33-62)
Sex	
Male	6 (85.7%)
Female	1 (14.3%)
Primary renal diseases	
Glomerulonephritis	4 (57.1%)
Hypertensive nephropathy	2 (28.6%)
Diabetic nephropathy	1 (14.3%)
Current comorbidities	
Hypertension	6 (85.7%)
Diabetes mellitus	1 (14.3%)
Maintenance immunosuppressive regimen	
GC + tacrolimus + mycophenolate sodium	6 (85.7%)
GC + tacrolimus + mycophenolate mofetil	1 (14.3%)
Time from transplantation to onset (years)	5.0 (0.6-26.0)
Symptoms	
Fever	7 (100%)
Progressive dyspnea	5 (71.4%)
Dry cough	3 (42.9%)
Diagnosis	
Confirmed by BALF mNGS	6 (85.7%)
Confirmed by peripheral blood mNGS	1 (14.3%)
Time from respiratory symptom onset to RF (days)	6 (2-19)
Time from respiratory symptom onset to PJP diagnosis (days)	10 (3-25)
PSI score	128 (101-152)
APACHE II score	19 (13-30)
SOFA score	8 (3-14)

Data are presented as median (range) or n (%). Abbreviations: APACHE II, Acute Physiology and Chronic Health Evaluation II; BALF, bronchoalveolar lavage fluid; GC, glucocorticoid; mNGS, metagenomic next-generation sequencing; PJP, *Pneumocystis jirovecii* pneumonia; PSI, Pneumonia Severity Index; RF, respiratory failure; SOFA, Sequential Organ Failure Assessment.

blood from 1 patient. Concurrent cytomegalovirus (CMV) viremia was identified in 4 of the 7 patients (Table 2).

Therapeutic Management

After the diagnosis of severe PJP-ARDS, all patients were treated using a standardized triple-therapy protocol. First, to reduce immunosuppression, all antimetabolites and calcineurin inhibitors were discontinued. Second, a comprehensive anti-*Pneumocystis* regimen was initiated. TMP-SMX was administered at a therapeutic dosage (TMP 15-20 mg/kg/day) for a median of 22 days; it was combined with caspofungin (50 mg/day after a loading dose) for a median of 15 days. Third—central to our strategy for managing ARDS—early adjunctive GC therapy

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Table 2. Laboratory findings at intensive care unit admission and imaging characteristics.

Laboratory tests	All patients (N = 7)
Leukocyte count (× 10^9/L)	10.5 (3.2-15.0)
Lymphocyte count (× 10^9/L)	0.3 (0.1-1.0)
C-reactive protein (mg/L)	117.5 (31.1-207.8)
Procalcitonin (ng/mL)	0.6 (0.2-1.5)
Albumin (g/L)	27 (22-34)
Lactate dehydrogenase (U/L)	442 (202-620)
Serum creatinine (μmol/L)	192 (89-572)
eGFR (mL/min/1.73 m^2)	26.0 (7.9-69.2)
BDG (pg/mL)	143.5 (37.5-899.9)
GM (μg/L)	0.2 (0.1-0.3)
PJ read count	3718 (230-28753)
pH	7.4 (7.1-7.5)
$PaCO_2$ (mm Hg)	49.5 (33.0-62.0)
PaO_2 (mm Hg)	55 (37-95)
PaO_2/FiO_2 (mm Hg)	80 (37-190)
CMV co-infection	4 (57.1%)
Computed tomography findings	
Diffuse ground-grass opacities	7 (100%)
Air bronchogram	1 (14.3%)
Pleural effusion	1 (14.3%)

Data are presented as median (range) or n (%). Abbreviations: BDG, 1,3-β-D-glucan; CMV, cytomegalovirus; eGFR, estimated glomerular filtration rate; FiO_2, fraction of inspired oxygen; GM, galactomannan; $PaCO_2$, partial pressure of arterial carbon dioxide; PaO_2, partial pressure of arterial oxygen; PJ read count, metagenomic next-generation sequencing read count for *Pneumocystis jirovecii*.

was administered. Intravenous methylprednisolone was initiated upon ICU admission in most patients, with a median starting dose of 80 mg/day. The dose was tapered over 1 to 2 weeks according to respiratory improvement, resulting in a median cumulative dose of 580 mg over 11 days. Regarding viral co-infections, the 4 patients with concurrent CMV viremia received dose-adjusted intravenous ganciclovir. Regular monitoring of plasma CMV DNA levels confirmed the effectiveness of antiviral therapy. Despite concurrent treatment with adjunctive high-dose GCs, no clinically significant worsening of CMV viremia or systemic viral dissemination was observed, indicating the safety of this approach.

Supportive care was intensive. Six patients required invasive mechanical ventilation for a median of 14 days due to severe hypoxemia, and 5 had a PaO_2/fraction of inspired oxygen (FiO_2) ratio below 100 mm Hg. Vasopressors were required for hemodynamic support in 6 patients, and 3 patients underwent continuous renal replacement therapy (**Table 3**).

Outcomes and Follow-up

Clinical outcomes following this regimen were highly favorable. The cohort achieved a 100% survival rate; 6 patients recovered sufficiently to be discharged home, and 1 was transferred to a local facility for rehabilitation. Median lengths of stay were 18 days in the ICU and 40 days in the hospital (**Table 3**). Microbiological response was confirmed by follow-up mNGS, which demonstrated complete clearance of *P. jirovecii* in 6 patients and a substantial reduction in fungal burden in the remaining patient. Regarding safety, the high-dose steroid regimen resulted in transient hyperglycemia requiring insulin therapy in 4 patients (median peak glucose, 13.9 mmol/L); these episodes were readily managed. Importantly, no cases of gastrointestinal hemorrhage, secondary superinfection, or severe electrolyte disturbance were observed.

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Table 3. Treatment and clinical outcomes of patients with severe PJP after renal transplantation.

Treatment regimen	All patients (N = 7)
Maintenance glucocorticoid therapy	
Methylprednisolone	3 (42.9%)
Prednisone	4 (57.1%)
Maintenance prednisone-equivalent dose	
5 mg/day	3 (42.9%)
10 mg/day	4 (57.1%)
Duration of TMP-SMX therapy (days)	22 (10-32)
Duration of caspofungin therapy (days)	15 (10-19)
Time from ICU admission to IV corticosteroid initiation (days)	0 (0-4)
IV corticosteroid type	
Methylprednisolone	7 (100%)
Initial dose	
40 mg/day	3 (42.9%)
80 mg/day	3 (42.9%)
120 mg/day	1 (14.3%)
Duration of methylprednisolone therapy (days)	11 (5-17)
Total methylprednisolone dose (mg)	580 (200-840)
Corticosteroid-related adverse effects	
Maximum blood glucose (mmol/L)	13.9 (10.0-22.1)
Insulin therapy required	4 (57.1%)
Maximum serum sodium (mmol/L)	141 (136-148)
Gastrointestinal bleeding	0
Secondary infection	0
Duration of mechanical ventilation (days)	14 (0-25)
Length of ICU stay (days)	18 (11-46)
Length of hospital stay (days)	40 (11-82)
Outcome	
Recovery	6 (85.7%)
Transfer after clinical improvement	1 (14.3%)

Data are presented as median (range) or n (%). Abbreviations: ICU, intensive care unit; IV, intravenous; PJP, *Pneumocystis jirovecii* pneumonia; TMP-SMX, trimethoprim-sulfamethoxazole.

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Discussion

This case series highlights the paradoxical role of GCs in RTRs with PJP, demonstrating that whereas chronic exposure increases susceptibility to infection and withdrawal may precipitate clinical deterioration, early adjunctive administration can be therapeutically beneficial. We successfully managed 7 cases of severe PJP-ARDS in RTRs using a standardized triple-therapy regimen that comprised TMP-SMX, caspofungin, and adjunctive GCs, achieving a 100% survival rate. These outcomes differ from the high mortality typically associated with severe non-HIV PJP [10], suggesting that a treatment strategy with explicit emphasis on the "GC paradox" can substantially improve prognosis. Furthermore, recent studies indicate that solid organ transplant recipients may represent a distinct clinical subgroup with a more favorable prognosis relative to other non-HIV PJP populations [11]. Therefore, understanding the dual role of GCs as both a contributor to infection risk and a modulator of inflammatory lung injury is critical for managing severe cases.

Chronic GC exposure is a major predisposing factor for PJP in RTRs. In the present series, all patients developed PJP while receiving low-dose GC maintenance therapy without PJP chemoprophylaxis. Although current guidelines recommend initiating prophylaxis when the prednisone-equivalent dose exceeds 20 mg/day for at least 4 weeks [4,12,13], the median GC dose in our cohort was below this threshold. Importantly, susceptibility should be considered in the context of the overall immunosuppressive burden rather than GC dose alone. The concomitant use of calcineurin inhibitors and mycophenolate derivatives acts synergistically with GCs to profoundly suppress T-cell-mediated immunity. Mechanistically, GCs increase vulnerability through several complementary pathways, including dose-dependent suppression of $CD4^+$ and $CD8^+$ T lymphocytes [14], downregulation of B-cell effector function in lung tissue [15], and expansion of regulatory T cells accompanied by inhibition of Th1 and Th2 lymphocyte activity [16]. Collectively, these effects create a profoundly immunosuppressed state that increases susceptibility to PJP and is reflected by the severe lymphopenia observed in our cohort.

In patients with non-HIV PJP, GC tapering or discontinuation after pulse therapy frequently precipitates clinical deterioration [5]. This observation suggests that, in immunocompromised hosts with subclinical PJP infection, abrupt GC withdrawal disrupts the pathophysiological equilibrium of "high pathogen burden vs low tissue inflammation" [17,18]. Such a dynamic shift resembles IRIS. Animal studies have shown that GC withdrawal leads to rapid rebound activation of alveolar macrophages [19] and accelerated pulmonary infiltration by $CD8^+$ T cells [20], triggering increased production of proinflammatory cytokines (e.g., tumor necrosis factor-α and interleukin-6) [21] that compromise alveolar-capillary integrity. We acknowledge that, in the absence of serial inflammatory marker measurements or cytokine profiling, the present series lacks biological confirmation of these immune dynamic changes. Therefore, classifying such deterioration as IRIS remains speculative. Although the clinical course is consistent with an IRIS-like mechanism, further investigation is warranted to determine whether these specific pathways underlie clinical worsening in patients with non-HIV PJP.

Regarding therapeutic intervention, our protocol for early adjunctive intravenous methylprednisolone was primarily based on established guidelines for HIV-associated PJP, which recommend initiating GC therapy within 72 hours in patients with severe hypoxemia [6,7]. However, the use of adjunctive GCs in non-HIV PJP populations remains controversial. Although early retrospective studies failed to demonstrate prognostic benefit [22-24], more recent evidence highlights the importance of distinguishing among specific patient subgroups. For example, a recent study indicated that solid organ transplant recipients may represent a distinct clinical population displaying a more favorable baseline prognosis relative to other patients with non-HIV PJP [11]. Conversely, a recent multicenter study showed that higher cumulative doses of adjunctive corticosteroids in cases of non-HIV PJP were independently associated with increased 90-day mortality [25]. After integrating these findings with our results, we propose that the favorable outcomes observed in our cohort are attributable to avoidance of excessive cumulative immunosuppression. By utilizing a targeted, moderate-dose, short-course GC regimen tailored to this solid organ transplant population, we sought to mitigate alveolar injury and modulate excessive inflammatory responses [26]. Additionally, because more than 50% of our cohort had concurrent CMV infection, there was concern that adjunctive GC therapy might exacerbate viral replication. Importantly, concurrent administration of dose-adjusted ganciclovir effectively controlled CMV viremia, suggesting that short-course adjunctive GC therapy can be safely administered without compromising viral control.

The inclusion of caspofungin in our regimen targets the unique biological characteristics of *P. jirovecii*. Whereas TMP-SMX inhibits trophozoite replication, caspofungin synergistically disrupts cyst wall integrity through inhibition of β-1,3-D-glucan synthase [27,28]. Because cystic forms are a major source of proinflammatory β-1,3-D-glucan, their rapid elimination by caspofungin may complement the systemic anti-inflammatory effects of corticosteroids, collectively mitigating the excessive inflammatory response associated with severe ARDS.

Several limitations of this study should be acknowledged. The retrospective design, small sample size, and lack of a control group inherently limit the generalizability of our findings and preclude definitive conclusions regarding the independent

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efficacy of each component of the triple-therapy regimen. Furthermore, given that serial cytokine profiling and longitudinal inflammatory marker measurements were not performed, our discussion of an IRIS-like mechanism remains a speculative clinical hypothesis rather than a biologically confirmed phenomenon. Nevertheless, the uniform application of the treatment protocol and the 100% survival rate achieved in this typically high-mortality population provide a compelling clinical signal. Future prospective multicenter studies are needed to validate our findings and to define the optimal dosing and duration of adjunctive corticosteroid therapy in solid organ transplant recipients.

Conclusions

Chronic low-dose GC therapy increases PJP risk; altered GC exposure may contribute to disease progression through an IRIS-like mechanism. However, definitive anti-*Pneumocystis* therapy with TMP-SMX and caspofungin, combined with early adjunctive GC administration, might be a promising strategy for managing severe PJP-ARDS in RTRs.

Department and Institution Where Work Was Done

The First Affiliated Hospital, Sun Yat-sen University, Guangzhou, Guangdong, PR China.

Patient Consent

The study protocol was approved by the Hospital Ethics Committee of The First Affiliated Hospital, Sun Yat-sen University (Approval No. [2024]370). Due to the retrospective nature of the study and the use of de-identified clinical data, the requirement for informed consent was waived by the Ethics Committee.

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