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CASE REPORT

# Diagnostic Challenges of Six-Pathogen Detected by mNGS in an Immunocompromised ICU Patient with Severe Community-Acquired Pneumonia-Induced Sepsis: A Case Report and Literature Review

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**Introduction:** Severe community-acquired pneumonia (SCAP) in immunocompromised patients is often caused by rare atypical pathogens, which are difficult to detect using conventional microbiological tests (CMTs) and can progress to sepsis in severe cases. Metagenomic next-generation sequencing (mNGS), an emerging pathogen detection technique, enables rapid identification of mixed infections and provides valuable guidance for clinical treatment decisions. SCAP-induced sepsis caused by a six-pathogen co-infection has not been previously reported, but interpretation remains a challenge.

**Case Presentation:** This report describes a case of SCAP-induced sepsis detected six pathogens by mNGS in a patient with IgA nephropathy who developed immunosuppression following long-term treatment with rituximab and corticosteroids. Bronchoalveolar lavage fluid (BALF) mNGS detected six pathogens, including *Pneumocystis jirovecii, Klebsiella pneumoniae*, Primate bocaparvovirus 1, Cytomegalovirus, *Elizabethkingia anophelis*, and *Candida albicans*. The patient was admitted to the intensive care unit (ICU) and received a combination of meropenem, trimethoprim-sulfamethoxazole, ganciclovir, piperacillin-tazobactam, and caspofungin. Following appropriate treatment, the patient recovered and was successfully discharged.

**Conclusion:** mNGS offers significant advantages for the diagnosis and identification of mixed infections in immunocompromised patients with SCAP-induced sepsis. It enables clinicians to initiate timely and targeted antimicrobial therapy, which facilitates early recovery, reduces the overuse of broad-spectrum antibiotics, and ultimately improves patient prognosis. Nevertheless, its interpretation requires caution, as distinguishing true pathogens from colonizers or contaminants still relies on clinical correlation and complementary diagnostic methods.

**Keywords:** severe community-acquired pneumonia, immunocompromised patient, mNGS, sepsis, case report

## Introduction

The proportion of immunocompromised individuals has risen steadily, now comprising nearly one-third of intensive care unit (ICU) admissions.[1] Severe community-acquired pneumonia (SCAP) remains a major cause of ICU admission and is associated with high mortality (30–50%).[2,3] The outcomes are particularly poor in immunocompromised patients.[4] The high mortality rates are primarily caused by the atypical pathogens or co-infection and the delayed initiation of targeted antimicrobial treatment.[1,4,5] At least one-third of SCAP patients present to the hospital with severe sepsis.[6] Early pathogen identification is essential for timely targeted therapy. This is the cornerstone of improving outcomes and reducing mortality in these patients.[7,8]

Conventional microbiological tests (CMTs) are widely used for SCAP diagnosis but are slow and often miss atypical pathogens or co-infections.[1,9] Metagenomic next-generation sequencing (mNGS) has gained prominence in infectious disease diagnostics due to its high throughput and rapid turnaround.[10–12] For SCAP patients, the pathogen positivity rate for mNGS

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was significantly higher than that for CMTs. It also enables, for the first time, an evaluation of whether mNGS-guided antimicrobial therapy improves the prognosis of SCAP in immunocompromised patients.[10,13,14]

We report a rare case of simultaneous co-infection with six pathogens, including *Pneumocystis jirovecii, Klebsiella pneumoniae*, Primate bocaparvovirus 1, Cytomegalovirus (CMV), *Elizabethkingia anophelis*, and *Candida albicans*, identified through bronchoalveolar lavage fluid (BALF) mNGS in an immunocompromised patient with SCAP-induced sepsis in the ICU. The patient was successfully treated, with a favorable clinical outcome. Reports of such extensive co-infections involving six pathogens in this population remain exceedingly rare.

## Case Presentation

A 61-year-old elderly female presented to the emergency department on March 15, 2025, with a one-week history of fever, a three-day history of cough with sputum production, and chest tightness. She had previously visited a local hospital, where she was treated with cefuroxime and azithromycin, along with antipyretic and expectorant therapies. However, her respiratory symptoms showed no significant improvement. Therefore, she came to our hospital later that evening for further evaluation and treatment.

Upon assessment in the emergency department, she exhibited shortness of breath and marked chest tightness. Her vital signs were as follows: temperature 37.6°C, blood pressure 114/67 mmHg, and oxygen saturation 92%. Chest computed tomography (CT) revealed "bilateral pulmonary infiltrates, possibly interstitial pneumonia; extensive inflammation in both lungs; and pleural effusion" (Figure 1A). Laboratory findings showed a hemoglobin level of 97 g/L, a white blood cell (WBC) count of $11.0 \times 10^9$/L, and a neutrophil percentage of 86.5%. Despite receiving treatment with meropenem (1.0 g ivgtt), supplemental oxygen, and corticosteroids for anti-inflammatory purposes in the emergency department, the patient's symptoms did not improve, and her oxygen saturation continued to decline. Consequently, she was intubated and placed on mechanical ventilation and was subsequently admitted to the ICU for further treatment and management.

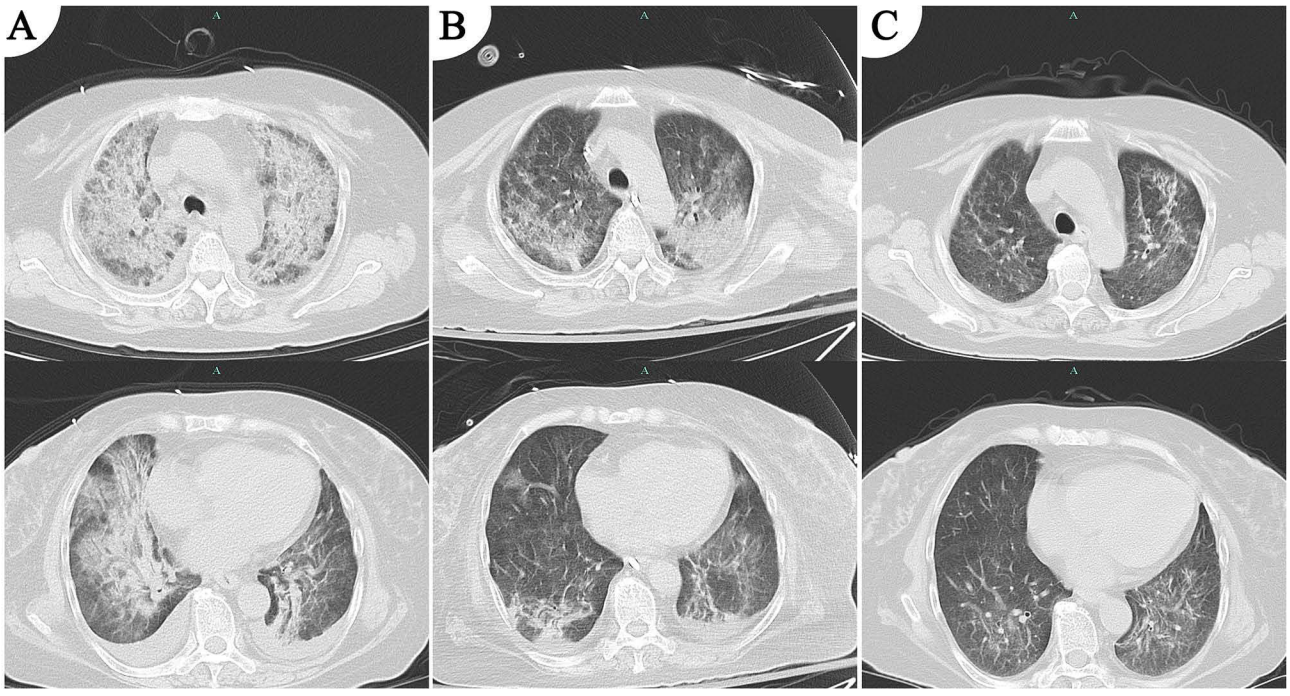


**Figure 1** Changes in pulmonary lesions on CT imaging. (**A**) (Day 1) The initial CT scan indicated extensive inflammation in both lungs, with a possible diagnosis of interstitial pneumonia; pleural effusion was also present. (**B**) (Day 7) The follow-up CT scan shows a reduction in inflammation in both lungs compared to before, with a reduction in pleural effusion (**C**) (Day 11) The CT scan after targeted antibiotic and relevant treatments showed a significant reduction in inflammation in both lungs, with pleural effusion was nearly resolved.

The patient had a history of IgA nephropathy and had been receiving long-term treatment with rituximab and corticosteroids, placing her in an immunocompromised state. In the ICU (Day 1), she continued to be treated with meropenem (1.0 g ivgtt), mechanical ventilation, and fluid resuscitation. However, on Day 2, after one day of treatment, her blood pressure continued to decline, metabolic acidosis showed no significant improvement, and serum lactate levels continued to rise (Table 1 and Figure 2). After consultation, the senior physicians diagnosed her with sepsis. Given the patient's immunosuppressed condition, antibiotic selection required careful consideration. We performed bronchoscopy to collect BALF samples, which were submitted for mNGS. CMTs, including blood culture, sputum culture, BALF culture, and polymerase chain reaction (PCR), were also performed simultaneously.

On Day 3, the patient's condition continued to deteriorate, with persistent hypotension (NE: 1.3 μg/kg·min), along with rising body temperature, interleukin-6 (IL-6) and C-reactive protein (CRP) levels (Table 1 and Figure 2). At 7:00 a.m., the mNGS results were returned (Figure 3A), revealing a co-infection with six pathogens: *Pneumocystis jirovecii* (71.62%, 3566 reads), *Klebsiella pneumoniae* (14.16%, 705 reads), Primate bocaparvovirus 1 (1.21%, 60 reads), CMV (0.36%, 18 reads), *Elizabethkingia anophelis* (0.42%, 21 reads), and *Candida albicans*. Given the complexity of the findings, a multidisciplinary case discussion was held. Based on clinical judgment, and considering the patient's immunosuppressed state and high risk for opportunistic infections, infection with the other five pathogens was confirmed, while *Candida albicans* was considered likely to be either a contaminant or a colonizer. Consequently, meropenem was discontinued, and targeted combination therapy was initiated with ganciclovir (0.25 g q12h ivgtt), piperacillin-tazobactam (4.5 g q8h ivgtt), and trimethoprim-sulfamethoxazole (3# q6h nasal feeding) to cover the identified pathogens.

On Day 5, after two days of targeted treatment, the patient's condition began to improve, with gradual normalization of CRP, IL-6 levels, and blood pressure. Additionally, *Candida albicans* was isolated by culture, and upon confirmation of *Candida albicans* infection, antifungal therapy with caspofungin (50 mg qd ivgtt) was initiated. On Day 7, follow-up CT scans revealed a reduction in pulmonary inflammation and pleural effusion (Figure 1B). The patient's $PaO_2/FiO_2$ ratio

**Table 1** The Timeline of Laboratory and Clinical Data of the Patient

| Tests | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 | Day 10 | Day 11 | Day 12 | Day 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Hemoglobin, g/L | 80 | 77 | 76 | 78 | 77 | 82 | 81 | 85 | 84 | 86 | 90 | 95 | 102 |
| WBC, $\times10^9$/L | 11.3 | 6.7 | 9.3 | 8.5 | 6.6 | 9.9 | 8.8 | 6.8 | 8.1 | 6.5 | 5.2 | 3.9 | 2.2 |
| Neutrophil% | 94.9 | 92.4 | 91.1 | 87.5 | 93.8 | 93.7 | 92.5 | 94.5 | 89.7 | 86.1 | 86.6 | 78.5 | 70.2 |
| Lymphocyte% | 3.6 | 6.1 | 7.1 | 10.5 | 4.5 | 5 | 5.7 | 3.8 | 8.4 | 9.8 | 9.9 | 10.6 | 13.5 |
| Platelets, $\times10^9$/L | 346 | 330 | 268 | 275 | 287 | 246 | 276 | 262 | 285 | 369 | 370 | 410 | 461 |
| Cr, μmmol/L | 58.3 | 50.3 | 60.2 | 59.1 | 61.3 | 75.5 | 70.5 | 83.7 | 77.7 | 72.8 | 83.9 | 89.3 | 98 |
| CRP, mg/L | 122.98 | 51.79 | 24.18 | 66.24 | 34.19 | 53.27 | 38.74 | 14.22 | 30.75 | 34.01 | 11.72 | 2.74 | 0.28 |
| PCT, ng/L | 0.12 | 0.22 | 0.16 | 0.07 | 0.09 | 0.13 | 0.09 | 0.12 | 0.09 | 0.10 | 0.09 | 0.06 | 0.04 |
| Bilirubin, μmol/L | 8.5 | 8.9 | 6.6 | 3.0 | 2.7 | 4.6 | 3.4 | 4.7 | 4.3 | 4 | 3.9 | 3.2 | 2.6 |
| ALT, U/L | 11.2 | 11.9 | 13.6 | 14.4 | 18.8 | 17.3 | 15.2 | 16.8 | 14.1 | 19.2 | 17.6 | 20.5 | 18.9 |
| AST, U/L | 61.4 | 63.6 | 53.1 | 47.4 | 47.2 | 41.7 | 30.1 | 43.7 | 39.4 | 39.6 | 31.5 | 18.6 | 17.8 |
| IL-6, pg/mL | 114.87 | 3.14 | 162.46 | 27.89 | 11.09 | 18.4 | 23.41 | 78.14 | 90.49 | 40.82 | 24.3 | 16.48 | 10.62 |
| Glucose, mmol/L | 11.3 | 10.5 | 7.3 | 13.3 | 10.6 | 11.1 | 11.3 | 9.4 | 9 | 8.9 | 8.4 | 7.8 | 6.9 |
| **Arterial blood gas** | | | | | | | | | | | | | |
| PH | 7.25 | 7.36 | 7.44 | 7.49 | 7.47 | 7.45 | 7.47 | 7.49 | 7.43 | 7.46 | 7.51 | 7.46 | 7.28 |
| $PO_2$, mmHg | 68.1 | 92.8 | 94.1 | 93.3 | 92.9 | 95.6 | 98.1 | 96.5 | 99.7 | 97.2 | 98.5 | 97.8 | 99.5 |
| $PCO_2$, mmHg | 50.3 | 36.8 | 36.7 | 30.9 | 35.1 | 34 | 39.1 | 38.1 | 44.9 | 35.1 | 27.1 | 24.3 | 22.5 |
| $HCO_3^-$, mmol/L | 21.9 | 19.7 | 20.2 | 23.7 | 27.8 | 30.5 | 29.1 | 27.9 | 25.4 | 22.5 | 19.7 | 20.3 | 18.9 |
| LAC, mmol/L | 2.9 | 3.4 | 3.3 | 3.5 | 3.1 | 2.7 | 2.5 | 2.1 | 1.3 | 1.7 | 1.5 | 1.4 | 1 |
| **Oxygen support** | | | | | | | | | | | | | |
| PEEP, $cmH_2O$ | 12 | 12 | 12 | 12 | 12 | 12 | 10 | 8 | 6 | / | / | / | / |
| $FiO_2$, % | 60 | 45 | 45 | 45 | 45 | 45 | 40 | 40 | 40 | 38 | 35 | 35 | / |
| HFOT, L/min | / | / | / | / | / | / | / | / | / | 30 | 30 | 30 | / |
| NE, μg (kg·min) | / | 0.675 | 0.675 | 0.337 | / | / | / | / | / | / | / | / | / |

**Abbreviations**: WBC, white blood count; Cr, creatinine; CRP, c-reactive protein; PCT, procalcitonin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LAC, lactic acid; PEEP, positive end-expiratory pressure; HFOT, high-flow oxygen therapy; NE, norepinephrine.


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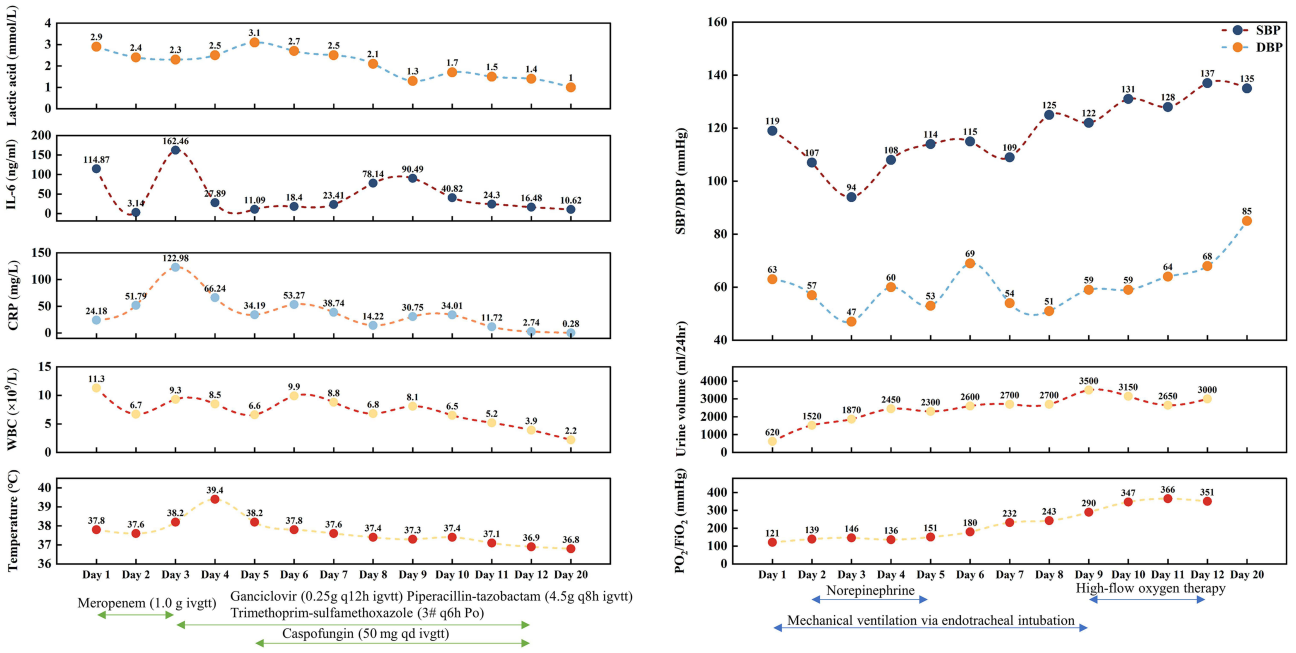


**Figure 2** Changes in patient's clinical parameters during ICU hospitalization.
**Abbreviations**: WBC, white blood count; IL-6, Interleukin-6; CRP, c-reactive protein; SBP, systolic blood pressure; DBP, diastolic blood pressure.

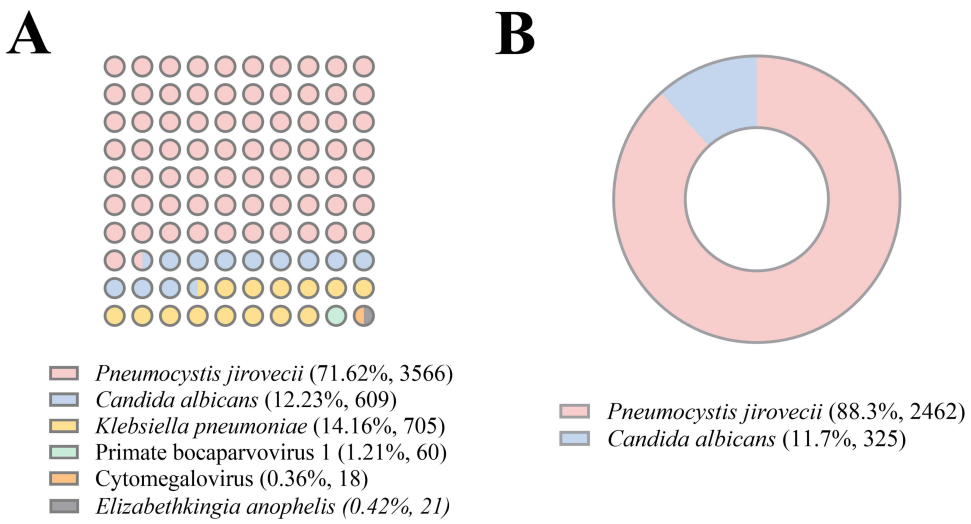


**Figure 3** The distribution of pathogens identified by mNGS. (**A**) mNGS result on Day 3 (**B**) mNGS result on Day 7.

and urine volume continued to improve (Table 1 and Figure 2). Repeat mNGS results at this point indicated the presence of only *Pneumocystis jirovecii* and *Candida albicans* (Figure 3B), suggesting that the patient was responding well to the antimicrobial therapy, which was therefore continued. On Day 11, repeat CT scan demonstrated a marked reduction in bilateral pulmonary inflammation with near-complete resolution of pleural effusion (Figure 1C). Although levels of CRP and IL-6 fluctuated during this period (Table 1 and Figure 2), they eventually improved, along with other clinical indicators such as lactic acid, body temperature, and urine output. On Day 12, the patient was transferred to a general ward. On Day 20, patient was discharged in good condition.

## Discussion

To our knowledge, this is the first report of SCAP-induced sepsis caused by co-infection with six pathogens in an immunocompromised patient.

Immunocompromised patients in the ICU are particularly susceptible to infections caused by specific pathogens, multi-drug-resistant organisms, and opportunistic organisms.[15,16] In previous studies, infections with *Streptococcus pneumoniae*, respiratory viruses, *Haemophilus influenzae*, and atypical pathogens have been frequently identified in patients with SCAP.[4] However, with the recent advancement of mNGS, it has emerged as a valuable tool for identifying infectious pathogens in immunocompromised patients. Recent studies have shown that immunocompromised patients with SCAP exhibit a distinct spectrum of pathogens compared to immunocompetent patients.[5,10,17] In immunocompromised patients with SCAP, in addition to common pathogens, the most frequently identified respiratory pathogens include CMV, *Pneumocystis jirovecii*, Epstein-Barr virus and *Aspergillus species*. These pathogens are more likely to lead to sepsis and may be associated with an increased risk of mortality in this population.[18–20] Therefore, early identification of pathogens and timely administration of targeted antimicrobial therapy are crucial for improving outcomes in this population.[1,21]

CMTs have various limitations. For example, standard cultures often require prolonged turnaround times, exhibit low positivity rates, and may fail to detect fastidious or atypical organisms.[13] Moreover, many patients have already received antibiotic therapy prior to ICU admission, which can further reduce the diagnostic yield of CMTs.

The patient had received antibiotic therapy prior to hospital admission, resulting in negative CMTs findings. Although mNGS identified six pathogens, previous studies have shown that mNGS may also detect commensal or colonizing organisms.[22] Therefore, we interpreted the results with caution. After multidisciplinary expert discussion, all specialists agreed on the presence of infections caused by pathogens other than *Candida albicans*, as it is a common colonizer frequently detected by mNGS.[23,24] However, considering the patient's immunocompromised status, we hypothesized that an invasive Candida infection could not be ruled out. This suspicion was later confirmed by positive culture results. Following targeted antimicrobial therapy, the patient eventually recovered and was discharged from the hospital. This further underscores the importance of mNGS in guiding treatment and improving outcomes in immunocompromised patients.

Compared with published cases involving up to five concurrent pathogens,[25] our case represents an even more complex infectious scenario, highlighting both the diagnostic challenges and the unique contribution of mNGS in detecting unexpected co-pathogens. Nevertheless, mNGS also has important limitations, including high cost, potential false positives, delayed turnaround time in certain settings, and challenges in data interpretation. These barriers currently restrict its widespread adoption in routine clinical practice, particularly in resource-limited environments. In addition, as this is a single-case report, statistical analysis could not be performed, which represents another limitation of the study.

## Abbreviations

ICU, Intensive care unit; mNGS, Metagenomic next-generation sequencing; SCAP, Severe community-acquired pneumonia; BALF, Bronchoalveolar lavage fluid; CMTs, Conventional microbiological tests; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PCR, Polymerase chain reaction; PCT, Procalcitonin; CRP, C-reactive protein; PLT, Platelet; WBC, White blood cell; CMV, Cytomegalovirus; CT, Computed tomography; IL-6, Interleukin-6.

## Data Sharing Statement

The raw sequence data for this study have been uploaded and deposited in NCBI under BioProject no. PRJNA1258890.

## Ethics Statement

This case report was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Quzhou People's Hospital (2025-50). Informed consent was obtained from the patient for publication of this report and any accompanying images.

## Consent for Publication

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

## 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 that they have no competing interests in this work.

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