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CASE SERIES

Polymyxin-Associated Neurotoxicity: A Case of Guillain-Barré Syndrome Temporally Linked to Colistin Methanesulfonate and Respiratory Paralysis with Polymyxin B

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Abstract: Polymyxins are critical for multidrug-resistant Gram-negative infections, yet severe neurotoxicity remains underrecognized, within Guillain-Barré Syndrome (GBS) has not been previously reported in association with polymyxin E. We report two cases illustrating a temporal association between polymyxin therapy and severe neurological manifestations. The first is a case of polymyxin B-induced respiratory paralysis in a patient with bloodstream infection. The second represents the first reported case of GBS associated with polymyxin E (Colistin Methanesulfonate) in a patient with pneumonia; the diagnosis was confirmed electrophysiologically, and the patient responded to immunotherapy. These findings suggest a potential link between polymyxin therapy and severe, atypical neurotoxicity. Clinicians should be vigilant for symptoms ranging from acute paresthesia to GBS, as early recognition and drug discontinuation are crucial to preventing severe outcomes.

Keywords: polymyxin, neurotoxicity, Guillain-Barré syndrome, colistin methanesulfonate, multidrug-resistant infections

Introduction

Polymyxins including polymyxin B and Colistin Methanesulfonate (CMS) are a class of polypeptide antibiotics that were developed in the 1940s but fell out of favor due to concerns over nephrotoxicity and neurotoxicity.[1] Their clinical application has recently revived in response to the global rise in multidrug-resistant (MDR) pathogens such as *Acinetobacter baumannii, Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*.[2] This resurgence is primarily driven by the increasing prevalence of carbapenem-resistant Gram-negative bacteria, for which polymyxins have become a critical last-line treatment option. Additionally, advances in dosage optimization and toxicity management, including better handling of fluid and electrolyte imbalances, have further supported their reintroduction, as newer studies report significantly lower rates of nephrotoxicity and neurotoxicity compared to older data.[3] However, the renewed use of polymyxins has also brought attention to their adverse effects, including dizziness, paresthesia, neuromuscular blockade, and, in rare cases, respiratory failure.[4–6]

Polymyxin-induced neurotoxicity is typically attributed to presynaptic inhibition of acetylcholine release.[7] In contrast, immune-mediated neuropathies such as Guillain–Barré syndrome (GBS) represent a distinct pathological entity. GBS is characterized by acute inflammatory demyelination or axonal injury, often triggered by preceding infections or, rarely, drug exposure. While prior reports have described "GBS-like" symptoms during polymyxin therapy, these typically lacked definitive electrophysiological or cerebrospinal fluid findings required for GBS diagnosis, and none have been linked to CMS.

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A hypothetical biological rationale linking polymyxins to immune-mediated neuropathy may involve drug-induced neuronal injury via oxidative stress and mitochondrial dysfunction and would lead to exposure of neural antigens and subsequent autoimmune activation.[8,9] This mechanism aligns with the delayed onset and immunotherapy-responsive course observed in classic GBS, distinguishing it from direct pharmacotoxic effects.

However, whether polymyxin therapy can trigger true immune-mediated GBS remains unclear, and no confirmed cases have been reported to date. Herein, we present two illustrative cases: one of acute respiratory paralysis following polymyxin B administration, consistent with direct neurotoxicity, and the first reported case of GBS temporally associated with CMS, fulfilling diagnostic criteria and responding to immunotherapy. These cases aim to highlight the diagnostic challenge in distinguishing polymyxin-induced neurotoxicity from immune-mediated neuropathy and to propose a mechanistic framework for future investigation.

Case Presentation

Case 1 (Timeline Summarized in Figure 1): Polymyxin B-Associated Neurotoxicity

A male in his 30s was admitted to Ruijin Hospital affiliated to Shanghai Jiao Tong University on March 20, 2019, following surgery for severe acute pancreatitis. Data for this case report were retrieved from the patient's electronic health records and the hospital's pharmacy surveillance system. His postoperative course was complicated by a catheter-related bloodstream infection. Cultures from the central venous catheter grew extensively drug-resistant *Klebsiella pneumoniae* (XDR-KP), susceptible only to polymyxin B.

Consequently, intravenous polymyxin B therapy was initiated on March 31, 2019. The patient received a loading dose of 25,000 units/kg (based on actual body weight) infused over 2 hours, followed by a maintenance dose of 13,000 units/kg every 12 hours. The patient had normal renal function (serum creatinine within normal range) at the time of initiation.

Approximately 2 hours after the first infusion, the patient reported perioral and fingertip tingling, which rapidly progressed to numbness in all four limbs and constipation. Neurological examination revealed symmetrical reduction in light touch and pinprick sensation in a stocking-glove distribution, with preserved muscle strength and deep tendon reflexes prior to respiratory event. Despite these symptoms, a second infusion of polymyxin B was administered the next day (April 1). Within one hour, the patient developed acute respiratory distress, with oxygen saturation falling from 98% to 86%. Emergency oxygen supplementation stabilized him, and all further polymyxin B were immediately halted.

Given the clear temporal link between polymyxin B infusion and the rapid onset of characteristic symptoms (paresthesia progressing to respiratory compromise), along with the exclusion of other causes (eg, metabolic disturbances, concurrent neurotoxic drugs), a diagnosis of acute polymyxin B-induced neurotoxicity was made. Therapeutic drug monitoring was not performed, as the decision to discontinue polymyxin B was made immediately based on the

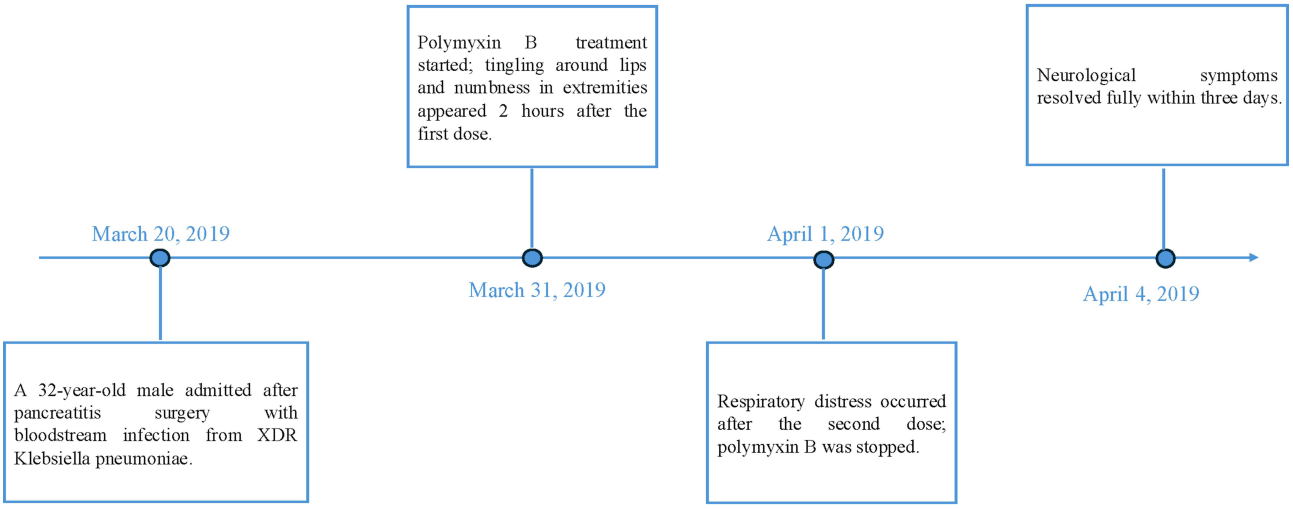

Figure 1 Timeline of acute neurotoxicity following polymyxin B administration in Case 1.

acute clinical presentation in a patient with normal renal function. Management followed the standard supportive care for drug-induced neuromuscular tocivity.

All neurological symptoms, including paresthesia and respiratory distress, resolved completely within three days of drug discontinuation (by April 4, 2019). This case illustrates the potential of polymyxin B to cause severe but reversible neurotoxic reactions, including paresthesia and respiratory paralysis.

Case 2 (Clinical Course Detailed in Figure 2): CMS-Associated Guillain-Barré Syndrome

A male in his 60s presented to Anhui Province Hospital on June 11, 2024, with acute abdominal pain radiating to the lower back. Laboratory tests confirmed acute pancreatitis, with elevated amylase (1,069 U/L) and lipase (2,743 U/L). Despite treatment with ceftriaxone-sulbactam, the condition worsened, necessitating escalation to imipenem-cilastatin and hemoperfusion. The patient was transferred to Ruijin Hospital affiliated to Shanghai Jiao Tong University on June 25, 2024. Cultures subsequently identified carbapenem-resistant *Acinetobacter baumannii* and multidrug-resistant *Pseudomonas aeruginosa*, both susceptible only to polymyxin E (colistin).

At Ruijin Hospital, empirical antibiotic therapy with meropenem and vancomycin was initiated. Microbial cultures from the hemodialysis catheter identified Candida albicans and carbapenem-resistant *Acinetobacter baumannii* (sensitive only to eravacycline and polymyxin E). Sputum cultures revealed multidrug-resistant Pseudomonas aeruginosa (sensitive only to polymyxin E). Based on these results, the treatment was adjusted on July 1, 2024, to include cefoperazone-sulbactam (3 g every 8 hours), caspofungin (50 mg daily), intravenous colistimethate sodium (CMS, 150 mg BID), and nebulized CMS (75 mg BID).

Following the initiation of polymyxin E therapy, the patient experienced dizziness and difficulty sleeping, particularly at night. Initial treatment with clonazepam (2 mg nightly) provided no relief. The regimen was revised to include betahistine mesylate (6 mg TID) and quetiapine fumarate (25 mg nightly), which modestly improved symptoms. However, by July 8, the dizziness persisted, prompting therapeutic drug monitoring. Blood samples were drawn at steady-state, just prior to the next scheduled dose (through concentration). Using high-performance liquid chromatography-tandem mass spectrometry (HPLC-MS/MS), serum levels of CMS-E1 and CMS-E2 were elevated at 8.31 mg/L and 2.11 mg/L, respectively (total: 10.42 mg/L). This total concentration exceeded the suggested upper limit of the therapeutic range (eg, 2/5 mg/L), indicating potential accumulation. Polymyxin E was discontinued on July 10, while cefoperazone-sulbactam and caspofungin therapy continued. The patient reported that dizziness and sleep quality had significantly improved by July 11, which allowed discontinuation of betahistine mesylate. By July 14, blood cultures were negative, and the patient was transferred to a general ward on July 25. He was discharged on August 3, with normal muscle strength and tone, and no residual sensory deficits.

On August 13, during a routine follow-up, the patient reported progressive limb weakness and difficulty walking. Physical examination revealed symmetric, ascending muscle weakness (Medical Research Council grade 3 in all limbs). Deep tendon reflexes were universally absent. No cranial nerve deficits or sensory level was detected. A mild degree of

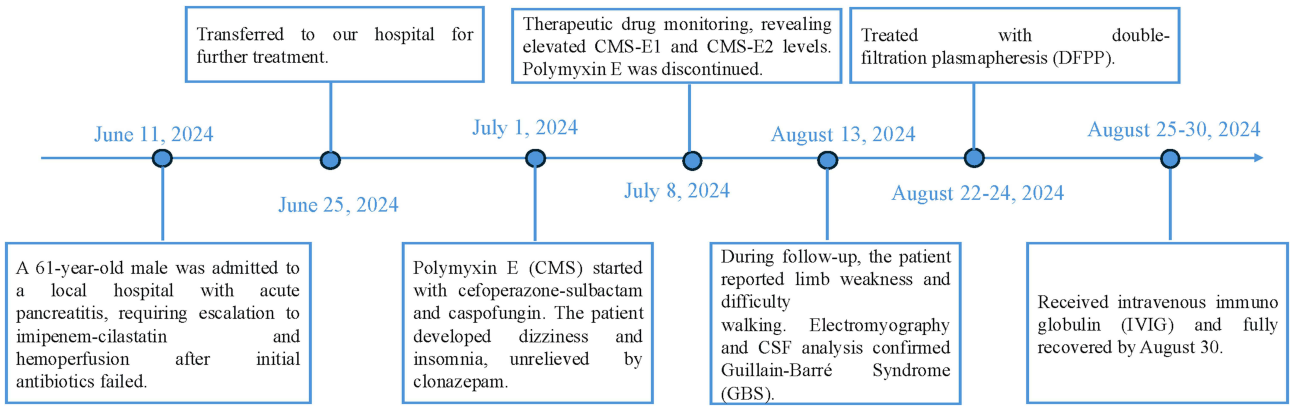

Figure 2 Clinical course of Guillain-Barré syndrome following polymyxin E (CMS) therapy in Case 2.

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sensory ataxia was observed during finger-nose testing. Nerve conduction studies and electromyography (NCS/EMG) were performed, revealing findings consistent with acute demyelinating polyneuropathy, including prolonged distal latencies, reduced conduction velocities, and partial conduction blocks. Cerebrospinal fluid (CSF) analysis performed via lumbar puncture showed markedly elevated protein levels (3305.63 mg/L) with a normal cell count (<5 mononuclear cells/μL), demonstrating albuminocytologic dissociation; Pandy's test was positive. CSF was also sent for bacterial and fungal cultures, which were sterile, and autoimmune encephalitis antibody panel, which was negative. A diagnosis of GBS was made according to the Brighton criteria after a multidisciplinary consultation.[10] To our knowledge, this represents the first reported case of GBS associated with polymyxin E. Following current GBS management guidelines, the patient underwent double-filtration plasmapheresis (August 22 to 24, 2024) followed by a five-day course of intravenous immunoglobulin (IVIG). Significant improvement in muscle strength was noted, with the left upper limb regaining grade 4+ strength, the right upper limb grade 4 strength, and the lower limbs grade 3 strength. By August 30, the patient had fully recovered muscle strength (grade 5) and was discharged with only mild residual paresthesia.

Discussion

This study series describes two distinct and severe forms of neurotoxicity associated with polymyxin antibiotics: an acute, reversible respiratory paralysis included by polymyxin B, and to our knowledge, the first reported case of GBS linked to polymyxin E (colistin methanesulfonate, CMS). These findings expand and recognized spectrum of polymyxin neurotoxicity and highlight critical diagnostic and management challenges.

In critically ill patients, the differential diagnosis for acute neuromuscular weakness is broad and includes critical illness polyneuropathy/myopathy (CIP/CIM),[11,12] metabolic derangements (eg, severe electrolyte disturbances), and other drug-induced neuropathies. In our second case, the diagnosis of GBS was favored over CIP/CIM based on the clear albuminocytologic dissociation in CSF (typically normal in CIP/CIM), the demyelinating pattern on NCS/EMG (often axonal in CIP), and the temporal profile—symptoms progressed after transferring out of the intensive care setting and significant clinical improvement followed immunotherapy, which is not typical for CIP/CIM. Furthermore, extensive laboratory workup ruled out significant electrolyte imbalances (eg, hypokalemia, hyponatremia) or endocrine disorders at the onset of weakness. Although the patient had a recent severe infection, which itself can trigger GBS,[13] the close temporal sequence following CMS exposure (with symptom onset weeks after drug initiation and a latent period after cessation) and the lack of other identified preceding infections in the 4 weeks prior to weakness make CMS a highly plausible trigger in this context.

Previous reports have documented neurotoxicity mimicking GBS. For instance, Camargo et al described colistin neurotoxicity in a cystic fibrosis patients with features resembling GBS, but diagnostic workup ultimately excluded it, with perioral paresthesia being a distinguishing clue.[14] Similarly, Ramezanzade et al documented GBS-like symptoms that diagnostic testing attributed to colistin neurotoxicity.[15] In another report, Nigam et al highlighted a postoperative patient developing hallucinations and neuromuscular symptoms that resolved promptly after colistin cessation, consistent with direct toxicity rather than immune-mediated pathology.[4] Wadia and Tran further emphasized severe colistin-induced respiratory failure and neuromuscular blockade in their case.[5] The case of polymyxin B-induced respiratory paralysis aligns with prior reports of neuromuscular dysfunction associated with polymyxin therapy. Studies have documented symptoms such as hypercapnic respiratory failure, paresthesia, and numbness affecting the face, hands, and head, often resolving rapidly upon drug discontinuation and dose adjustment.[6,16,17] Importantly, neurotoxicity occurs in approximately 3% of patients receiving polymyxin therapy, with renal impairment identified as a significant risk factor due to reduced clearance and subsequent drug accumulation.[18] The CMS-associated case, however, provides definitive evidence of true GBS: it presented with a latent period after drug cessation, featured clear albuminocytologic dissociation in CSF, demonstrated demyelinating polyneuropathy on EMG, and required immunotherapy for recovery. This temporal and pathophysiological profile distinguishes it from prior reports of "GBS-like" colistin toxicity and establishes a novel association. While our cases demonstrate a compelling temporal association between polymyxin therapy and severe neurological syndromes, a definitive causal relationship, particularly for the CMS-associated GBS, cannot be established from observational case reports alone. The diagnosis in Case 2 fulfills Brighton criteria for GBS, and the sequence of events (onset weeks after CMS initiation, with a latent period post-cessation) is suggestive. However, the association is

proposed based on biological plausibility and the exclusion of alternative triggers in the immediate 4-week pre-weakness period, rather than proven causation.

The contrast in clinical presentation between our two cases likely reflects distinct underlying mechanisms. The acute respiratory paralysis following polymyxin B (Case 1) is characteristic of direct, pharmacologically driven neurotoxicity. This is primarily attributed to a presynaptic neuromuscular blockade inhibiting acetylcholine release, explaining its rapid onset (within hours) and rapid reversibility upon drug cessation.[19] In contrast, the clinical course in Case 2 aligns more closely with a possible immune-mediated process. The delayed onset of symmetrical ascending weakness, occurring weeks after CMS initiation and following a latent period after drug discontinuation, coupled with the classic CSF finding of albuminocytologic dissociation and the clear response to immunotherapy, collectively support a diagnosis of true GBS rather than direct toxicity. A plausible, albeit unproven, mechanistic link is that CMS, through drug-induced neuronal stress (eg, via oxidative stress or mitochondrial dysfunction,[20,21] might expose neural antigens, thereby triggering an autoimmune response akin to post-infectious GBS. This immune-driven, delayed process fundamentally differs from the immediate pharmacotoxic effect seen with polymyxin B.

The overlapping symptoms of GBS and polymyxin-induced neurotoxicity, including paresthesia, muscle weakness, and hyporeflexia, often complicate differentiation.[8] However, key differences in their progression and diagnostic markers can aid in their distinction. GBS typically presents with progressive, ascending weakness and areflexia, with CSF analysis revealing albuminocytologic dissociation. Electromyography EMG findings of demyelination or axonal damage further support the diagnosis.[22] In contrast, polymyxin neurotoxicity manifests rapidly after drug initiation and is often characterized by perioral tingling and dizziness.[14,23] Symptoms resolve quickly with drug discontinuation, distinguishing it from prolonged recovery of GBS. Among GBS subtypes, Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is most common, presenting with demyelination and slow recovery, while Acute Motor Axonal Neuropathy (AMAN) can mimic drug-induced neurotoxicity due to motor-dominant symptoms. Early CSF analysis and EMG are critical in distinguishing these conditions. In polymyxin toxicity, serum drug levels help confirm neurotoxic etiology, particularly in patients with renal impairment.[24] Management hinges on early differentiation. GBS often requires immunotherapy, whereas polymyxin neurotoxicity is reversible with prompt drug cessation.[3,25] Recognizing the rapid symptom onset and reversibility of polymyxin-induced effects avoids unnecessary interventions, ensuring timely and appropriate treatment tailored to the underlying cause.

The distinct profiles of these two neurotoxicities, summarized in Table 1, necessitate different clinical approaches. First, vigilance is paramount; clinicians should monitor patients receiving polymyxins for neurological symptoms, from early paresthesia to severe weakness. Second, prompt diagnostic differentiation is critical. Acute onset (within hours/days) of symptoms, especially with perioral tingling, suggests direct pharmacotoxicity, which is typically reversible upon drug cessation. In contrast, a subacute or delayed onset (days to weeks after initiation or even following discontinuation)

Table 1 Differentiating Polymyxin-Induced Direct Neurotoxicity from Polymyxin-Associated Guillain-Barré Syndrome

Feature	Polymyxin Direct Neurotoxicity	Polymyxin-Associated GBS
Onset	Acute (hours to days after dose)	Delayed (days to weeks post-initiation or after discontinuation)
Key Symptoms	Perioral/distal paresthesia, dizziness, respiratory muscle paralysis	Progressive, symmetric limb weakness and sensory deficits
Proposed Mechanism	Neuromuscular blockade (presynaptic)	Immune-mediated (autoimmune)
Diagnostic Clues	1. Clear temporal link to infusion 2. Rapid improvement after cessation 3. Elevated drug levels (TDM)	1. CSF albuminocytologic dissociation 2. EMG: demyelinating features 3. Meets GBS diagnostic criteria
Primary Management	1. Immediate drug cessation 2. Supportive care	1. Drug cessation 2. Immunotherapy (IVIG/plasmapheresis)
Prognosis	Rapid recovery after discontinuation	Slower recovery; may require immunotherapy and rehabilitation

Abbreviations: GBS, Guillain-Barré Syndrome; CSF, Cerebrospinal fluid; IVIG, intravenous immunoglobulin.

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of ascending weakness should raise suspicion for immune-mediated processes like GBS, warranting immediate cerebrospinal fluid analysis and electrophysiological studies. Third, management must be tailored accordingly. While direct neurotoxicity primarily requires drug withdrawal and supportive care, GBS necessitates rapid initiation of immunotherapy (eg, intravenous immunoglobulin or plasmapheresis) in consultation with neurology. Finally, preventive strategies, including therapeutic drug monitoring and strict dose adjustment in renal impairment, are essential to mitigate the risk of drug accumulation and subsequent toxicity.

This report has several limitations inherent to its design as a descriptive case series. First, establishing a definitive causal relationship between polymyxin therapy and the neurological events, particularly GBS, remains challenging. This is inherent to the observational nature of case reports and the potential for unmeasured confounding (eg, concurrent critical illness, metabolic disturbances, or subclinical infections). Specifically for case 2, the extended interval between CMS cessation and GBS onset, while consistent with an immune-mediated process, also introduces greater uncertainty regarding causality comparted to the immediate reaction in Case 1. Second, the sample size is limited to two cases, which precludes generalization of the findings. Third, while therapeutic drug monitoring was performed in Case 2, it was not available in Case 1, and target concentration ranges for neurotoxicity are not well-defined. Despite these limitations, our cases provide a detailed clinical narrative that highlights a previously unreported potential severe adverse effect and underscores the importance of clinical vigilance.

Conclusion

This report describes the first documented case of Guillain-Barré Syndrome temporally linked to CMS therapy, alongside a classic case of polymyxin B-induced acute respiratory paralysis. These observations highlight a potential, though yet unproven, association and expand the recognized spectrum of severe neurological adverse events that may occur during polymyxin therapy. Clinicians should maintain a high index of vigilance, recognize potential symptoms early, and be prepared to consider a broad differential diagnosis that includes both direct neurotoxicity and immune-mediated processes like GBS. Therapeutic drug monitoring and dose adjustment based on renal function are key risk-mitigation strategies. Prompt drug discontinuation and neurology consultation for an appropriate diagnostic workup and intervention are essential upon suspicion of serious neurotoxicity. Given the observational nature of this report and the potential for confounding, further systematic studies and pharmacovigilance efforts are warranted to clarify the potential causal role of polymyxins, particularly CMS, in triggering immune-mediated neuropathies.

Abbreviations

CMS, Colistin Methanesulfonate; GBS, Guillain-Barré Syndrome; MDR, Multidrug-Resistant; XDR-KP, Extensively Drug-Resistant *Klebsiella pneumoniae*; CSF, Cerebrospinal Fluid; EMG, Electromyography; DFPP, Double-Filtration Plasmapheresis; IVIG, Intravenous Immunoglobulin.

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the institutional policy of Ruijin Hospital, Shanghai Jiaotong University School of Medicine, regarding the publication of anonymized case reports. Formal ethics committee approval was not required as per this policy, given that the report involves no experimental intervention, and all patient data have been fully anonymized. Written informed consent for publication was obtained from the patients.

Consent for Publication

Written informed consent was obtained from the patients for the publication of clinical data included in this case report.

Author Contributions

Dr. Jiayang Huang is the corresponding author and the guarantor for this case report. Dr. Juan He, Dr. Lu Li, and Ms. Xiaolan Bian, and Dr. Jiayang Huang contributed equally for editing the case presentation and the discussion. Mr. Ke Zhang and Mr. Wangsheng Li contributed equally for the graphic editing and clinical data collection. Dr. Li Ma is the physician in charge of both patients in the case report. 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

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