

# Refractory Purulent Meningitis Caused by Extended-Spectrum Beta-Lactamase-Producing *Escherichia coli* Complicated by Supraspinal Neurogenic Bladder in an Infant

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## Case Report

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# Abstract

Purulent meningitis is a severe central nervous system (CNS) infection in early infancy associated with high morbidity and long-term neurological sequelae. While neurogenic bladder secondary to purulent meningitis has been reported in adults, this complication is rarely described in pediatric survivors. We present a 2-month-old male infant with refractory meningitis caused by extended-spectrum beta-lactamase-producing *Escherichia coli* (ESBL-EC), complicated by subdural effusions, septic shock, and subsequent supraspinal-origin neurogenic bladder. Despite 21 days of intravenous meropenem therapy, the patient had persistently abnormal cerebrospinal fluid (CSF) parameters and recurrent fever, requiring intermittent dexamethasone and neurosurgical subdural drainage. Serial neuroimaging revealed bilateral frontotemporal cortical softening and hemorrhage. At 4-month follow-up, urological evaluation demonstrated reduced bladder compliance, detrusor overactivity, elevated post-void residual volume, and grade-III left vesicoureteral reflux. Normal spinal magnetic resonance imaging (MRI) localized injury to supraspinal micturition centers. Clean intermittent catheterization and antibiotic prophylaxis were initiated. Nevertheless, recurrent febrile urinary tract infections (UTIs) and progressive growth deceleration persisted up to 12 months of age. This case demonstrates that refractory infantile purulent meningitis with cortical brain injury may trigger neurogenic bladder. Early recognition, multidisciplinary teamwork, and long-term urological surveillance are critical for affected children. Further research is required to optimize antibiotic and corticosteroid regimens for complicated pediatric CNS infections.

## Introduction

Purulent meningitis is a life-threatening CNS infection in infants, carrying substantial risks of acute complications and permanent neurodevelopmental impairment. A Chinese multicenter surveillance study<sup>[1]</sup> reported that 84.0% of childhood purulent meningitis cases occurred in infants, with neonates accounting for 36.5%. Globally, an estimated up to 190 000 neonates die of meningitis every year<sup>[2]</sup>. Furthermore, 20%-50% of surviving children may develop long-term neurological sequelae<sup>[3]</sup>. A recent Chinese multicenter survey<sup>[4]</sup> reported the case-fatality rate of neonatal pyogenic meningitis is 9.1%, and the incidence of neurological complications is 5.9%. Refractory meningitis is defined by sustained clinical and laboratory instability despite appropriate antimicrobial therapy and confers poor prognosis<sup>[5]</sup>.

Neurogenic bladder refers to voiding dysfunction resulting from lesions along micturition-related neural pathways. In children, spinal dysraphism represents the predominant etiology<sup>[6]</sup>, whereas acquired supraspinal causes remain under-recognized. Multiple adult case reports have documented neurogenic bladder following purulent meningitis<sup>[7, 8]</sup>, yet comparable pediatric cases have not been previously published. Herein, we report an infant who developed neurogenic bladder after severe refractory ESBL-producing *E. coli* meningitis. This report aims to raise clinical awareness of this rarely-reported urological sequela and stress the importance of multidisciplinary long-term follow-up for high-risk meningitis survivors.

# Case Description

## Patient Information

The male infant was born at 38 + 5 weeks gestation, with a birth weight of 2.7 kg and Apgar scores of 10/10. His past medical history included neonatal ABO hemolytic jaundice and a prior episode of ESBL-producing *E. coli* meningitis at 1 month of age. He completed 21-day meropenem treatment with partial clinical improvement; however, CSF abnormalities persisted and brain MRI showed bilateral frontal and left-temporal cortical gliosis. He was readmitted at 2 months of age with a 3-hour history of fever.

## Discussion

To our knowledge, this is the first reported infant case of neurogenic bladder secondary to purulent meningitis. Several key clinical take-home messages merit emphasis.

First, this patient fulfilled diagnostic criteria for refractory purulent meningitis, despite 21 days of adequate-dose meropenem, CSF abnormalities and clinical instability persisted<sup>[9, 10]</sup>. Potential underlying mechanisms include impaired blood-brain-barrier integrity and elevated intracranial pressure that reduces antibiotic brain penetration<sup>[11, 12]</sup>. Although intraventricular or intrathecal antibiotics are recommended for multidrug-resistant CNS infections<sup>[13–15]</sup>, safety data for these approaches in young infants remain limited and off-label<sup>[16]</sup>. Given gradual clinical improvement, our multidisciplinary team opted to continue systemic intravenous antimicrobial therapy.

Second, corticosteroid use in pediatric purulent meningitis remains controversial<sup>[17]</sup>. With written parental informed consent, intermittent dexamethasone was given during episodes of septic shock and progressive subdural effusion. We observed fever resolution and improved nystagmus without obvious steroid-related adverse events. Nevertheless, optimal timing, dosage, and duration of corticosteroids for complicated infant meningitis should be individualized, considering potential risks to neurodevelopment.

Third, the neurogenic bladder in this case most likely originated from supraspinal cortical injury. Brain imaging identified bilateral frontotemporal cortical softening and hemorrhagic lesions, which may damage cortical micturition centers and their descending inhibitory pathways to the pontine micturition center, resulting in detrusor overactivity and reduced bladder compliance<sup>[18]</sup>. Normal spinal MRI further supports a cerebral, rather than spinal, etiology. This observation expands the known long-term sequelae of infant purulent meningitis beyond cognitive and motor deficits to include urological dysfunction<sup>[19, 20]</sup>.

Fourth, neurogenic bladder management in infancy poses substantial clinical challenges. Even with clean intermittent catheterization and prophylactic antibiotics, our patient suffered recurrent febrile UTIs and growth faltering, highlighting substantial impacts on quality of life<sup>[21]</sup>. Long-term renal function surveillance is mandatory, as high-grade vesicoureteral reflux may lead to reflux nephropathy and chronic kidney damage<sup>[22]</sup>.

Limitations: As a single-case report, definitive causal links between meningitis and neurogenic bladder cannot be established. Multiple overlapping therapeutic interventions were applied; accordingly, clinical improvement cannot be attributed to any single agent.

## Conclusion

Severe refractory infantile purulent meningitis complicated by bilateral cortical brain injury can result in supraspinal-origin neurogenic bladder. Clinicians should maintain high suspicion for urological sequelae among meningitis survivors with confirmed cortical lesions. Multidisciplinary collaboration between pediatric infectious disease, neurology, and urology specialists is essential to optimize long-term outcomes<sup>[23, 24]</sup>. Further studies are needed to refine antibiotic delivery strategies and corticosteroid regimens for this vulnerable infant population.

## Declarations

## Author Contributions

FC and ML collected clinical data and drafted the manuscript. ZC,DM, ML, WJ, NS, and CC participated in clinical case management. WA performed radiological interpretation. JY conceptualized this case report and critically revised the manuscript. All authors have read and approved the final version.

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## Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

## Ethical Statement

Written informed consent was obtained from the patient's legal guardians for publication of this case report and de-identified imaging data. No personally identifiable patient information has been disclosed.

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