

Successful treatment of pyogenic ventriculitis caused by extensively drug-resistant *Acinetobacter baumannii* with multi-route tigecycline: A case report

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Abstract

BACKGROUND

Pyogenic ventriculitis caused by extensively drug-resistant *Acinetobacter baumannii* (*A. baumannii*) is one of the most severe complications associated with craniotomy. However, limited therapeutic options exist for the treatment of *A. baumannii* ventriculitis due to the poor penetration rate of most antibiotics through the blood-brain barrier.

CASE SUMMARY

A 68-year-old male patient with severe traumatic brain injury developed pyogenic ventriculitis on postoperative day 24 caused by extensively drug-resistant *A. baumannii* susceptible to tigecycline only. Successful treatment was accomplished through multi-route administration of tigecycline, including intravenous combined with continuous ventricular irrigation plus intraventricular administration. The pus was cleared on the 3rd day post-irrigation, and cerebrospinal fluid cultures were negative after 12 d.

CONCLUSION

Our findings suggest that multi-route administration of tigecycline can be a therapeutic option against pyogenic ventriculitis caused by extensively drug-resistant *A. baumannii*.

Key Words: Pyogenic ventriculitis; *Acinetobacter baumannii*; Extensively drug-resistant; Tigecycline; Ventricular irrigation; Case report

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Core Tip: Pyogenic Ventriculitis caused by extensively drug-resistant (XDR) *Acinetobacter baumannii* (*A. baumannii*) is one of the most severe complications associated with craniotomy. We present herein a rare case of pyogenic ventriculitis caused by XDR *A. baumannii*. Successful treatment was accomplished through multi-route administration of tigecycline, including intravenous combined with continuous ventricular irrigation plus intraventricular administration. This case suggests that multi-route administration of tigecycline can be a therapeutic option against pyogenic ventriculitis caused by XDR *A. baumannii*.

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INTRODUCTION

Ventricular infection caused by *Acinetobacter baumannii* (*A. baumannii*) accounts for 3.6% to 11.2% of all cases of hospital-acquired ventriculitis^[1]. In recent years, strains of extensively drug-resistant (XDR) *A. baumannii* have emerged due to the overuse of antibiotics^[2]. Meningitis and ventriculitis caused by XDR strains are associated with a dismal prognosis, with mortality rates of up to 71%^[3-5].

Tigecycline, a new type of glycylcycline, is one of the few newly developed antimicrobial agents that are active against Gram-negative bacteria^[2]. *In vitro* experiments^[6-8] have shown that tigecycline is effective against *A. baumannii*, with strong antibacterial effects on XDR *A. baumannii*. Previous case reports and case series have provided information on the efficacy and safety of tigecycline in the treatment of ventriculitis or meningitis^[5,9-11]. In these cases, tigecycline was commonly administered *via* intravenous (IV) with or without intraventricular (IVT) route. Additionally, intraoperative intraventricular lavage for pyogenic ventriculitis has also been reported^[12]. However, the efficacy and safety profile of multi-route administration of tigecycline, including IV plus continuous ventricular irrigation (CVI)/IVT, remain relatively uncertain.

Here, we present the first case of successful treatment of pyogenic ventriculitis caused by XDR *A. baumannii* with multi-route tigecycline.

CASE PRESENTATION

Chief complaints

A 68-year-old male patient was admitted to our hospital due to a severe car accident and was diagnosed as having a severe traumatic brain injury.

History of present illness

The patient was admitted to our hospital due to a severe car accident 3 h ago. The clinical neurological examination revealed a deep coma. The diagnosis was a severe traumatic brain injury.

History of past illness

The patient did not have a specific history of past illness.

Personal and family history

The patient did not have a specific personal and family history.

Physical examination

When admitted to the hospital, the patient's temperature was 37.2 °C, heart rate was 98 bpm, respiratory rate was 18 breaths per minute, blood pressure was 138/78 mmHg, and oxygen saturation in room air was 98%. The clinical neurological

examination revealed a deep coma, with a Glasgow coma scale score of 3/15.

Laboratory examinations

On hospital day 18, laboratory tests showed 105 mg/L of C-reactive protein (CRP), and the white blood cell count (WBC) was $10.37 \times 10^9/L$. Additionally, his renal and liver function tests were normal. The cerebral spinal fluid (CSF) obtained through lumbar puncture showed increased WBC, while the glucose level was 0.8 mmol/L, and the total protein was 4.49 g/L. Three sets of sputum, blood, and CSF cultures were obtained, but no bacterial growth was detected in any of the three samples. On hospital day 21, analysis of the sputum and CSF cultures again demonstrated the absence of any bacterial growth. On hospital day 26, the patient's CSF culture tested positive for *A. baumannii* that was susceptible only to tigecycline, though susceptibility to polymyxin was not tested (Table 1). The same strain of XDR *A. baumannii* was persistently isolated from the CSF until hospital day 35. The results of blood and CSF analysis are shown in Table 2.

Imaging examinations

On hospital day 18, head computed tomography (CT) showed normal postoperative changes after the operations (Figure 1A). On hospital day 24, head CT indicated ventricular empyema (Figure 1B). On hospital day 29, no ventricular pus was detected on the head CT.

FINAL DIAGNOSIS

Pyogenic ventriculitis caused by XDR *A. baumannii*.

TREATMENT

The patient underwent bilateral decompressive craniectomy and evacuation of cerebellar hematoma simultaneously after admission. Then the patient was admitted to the intensive care unit following the operation. In order to prevent the development of an infection, 2 g of sulperazone was administered every 8 h (q8h) and ceased on hospital day 5.

On hospital day 7, the patient was transferred to the general ward. The body temperature of the patient remained normal until hospital day 18, when it increased to 39.1 °C. Laboratory tests showed 105 mg/L of CRP, and the WBC was $10.37 \times 10^9/L$. Additionally, his renal and liver function tests were normal. The CSF obtained through lumbar puncture showed increased WBC, while the glucose level was 0.8 mmol/L, and the total protein was 4.49 g/L. He was started empirically on 1 g meropenem q8h and 1 g vancomycin q12h. Three sets of sputum, blood, and CSF cultures were obtained, but no bacterial growth was detected in any of the three samples. In the following 6 d, the patient presented with remittent fever (peak at 38.8 °C). On hospital day 21, analysis of the sputum and CSF cultures again demonstrated the absence of any bacterial growth. On hospital day 24, the patient's fever increased to 40.1 °C, which was associated with meningeal signs and altered mental status. Head CT scans indicated ventricular empyema (Figure 1B). And emergency treatment included lumbar cistern drainage, right frontal external ventricular drainage (EVD), and left occipital EVD. The drainage tube used was VentriClear of Medtronic, which consists of translucent silicone elastomer impregnated with the antimicrobial agents. The pus was copiously irrigated with normal saline (NS) from both the frontal and occipital EVD. Meropenem and vancomycin continued to be administered.

On hospital day 26, the patient's CSF culture tested positive for *A. baumannii* that was susceptible only to tigecycline, though susceptibility to polymyxin was not tested (Table 1). Therefore, with the permission of the family and written consent, antimicrobial therapy was changed to IV tigecycline (starting at 100 mg and then 50 mg q12h) combined with CVI tigecycline. The CVI tigecycline was performed as follows: A 4 mg dose in 50 mL of NS, which was controlled with a syringe pump, was administered at a rate of 12.5 mL per hour at a frequency of q6h. The CVI tigecycline was administered *via* the right frontal EVD, during which the left occipital EVD was drained at a similar rate to balance the intracranial pressure. During continuous ventricular irrigation and EVD handling, to maintain asepsis, we disinfected the incision and drainage tube interface of the patient every other day. The lumbar cistern

Table 1 Antimicrobial susceptibility testing for *Acinetobacter baumannii* in cerebrospinal fluid

Antibiotic	Susceptibility	MIC (mg/L)
Amikacin	R	> 32
Ceftriaxone	I	> 32
Cefotaxime	R	> 32
Ceftazidime	R	> 16
Cefepime	R	> 16
Tetracycline	R	> 8
Levofloxacin	R	> 4
Selectrin	R	> 2
Piperacillin	R	> 64
Tigecycline	S	
Gentamicin	R	> 8
Ciprofloxacin	R	> 2
Tobramycin	R	> 8
Meropenem	R	> 8
Ticarillin	R	> 64

R: Resistance; I: Intermediary; S: Sensitivity; MIC: Minimum inhibitory concentration.

Table 2 Clinical course and cerebrospinal fluid examination

Hospital stay	Highest body temperature (°C)	Blood WBC (× 10 ⁹ /L)	Blood CRP (mg/L)	CSF glucose (mmol/L)	CSF protein (g/L)	CSF WBC (× 10 ⁶ /L)	Culture of CSF
Day 18	39.1	10.37	104	0.8	4.49	8800	N
Day 24	38.5	9.85	71	0.8	5.75	14000	MDRAB
Day 27	37.6	6.79	65	1.8	2.58	1200	MDRAB
Day 31	37.9	7.02	43	2.9	2.57	590	MDRAB
Day 32	37.2	7.71		2.6	2.25	160	MDRAB
Day 33	37.1	8.08		3.2	2.22	240	MDRAB
Day 36	37.4	8.32	14	3.4	3.88	40	N
Day 41	37.0	8.21		3.5	2.64	68	N
Day 42	37.2	6.51		3	2.13	90	N
Day 43	37.1	5.76	6	3.6	2.32	64	N

MDRAB: Multi-drug resistant *Acinetobacter baumannii*; N: Negative; CSF: Cerebrospinal fluid; WBC: White blood cell; CRP: C reactive protein.

drainage was removed on hospital day 27 as no CSF was discharged.

On hospital day 29, the CSF drained from the left occipital EVD was clear grossly, and no ventricular pus was detected on the head CT. However, the same strain of *A. baumannii* was again isolated from the CSF. Then, the right frontal EVD was removed, and the antimicrobial therapy was changed to IV tigecycline (50 mg q12h) combined with IVT tigecycline. The IVT tigecycline was performed as follows: A 2 mg dose in 4 mL of NS was injected through the left occipital EVD in 2 min at a frequency of q8h. After each dose, the left occipital EVD was closed for 2 h to prevent early antibiotic outflow. The same strain of XDR *A. baumannii* was persistently isolated from the CSF until hospital day 35. The patient still presented with remittent fever, but the peak decreased to 38.4 °C. The results of blood and CSF analysis are shown in Table 2. Antibiotic therapy remained unchanged.

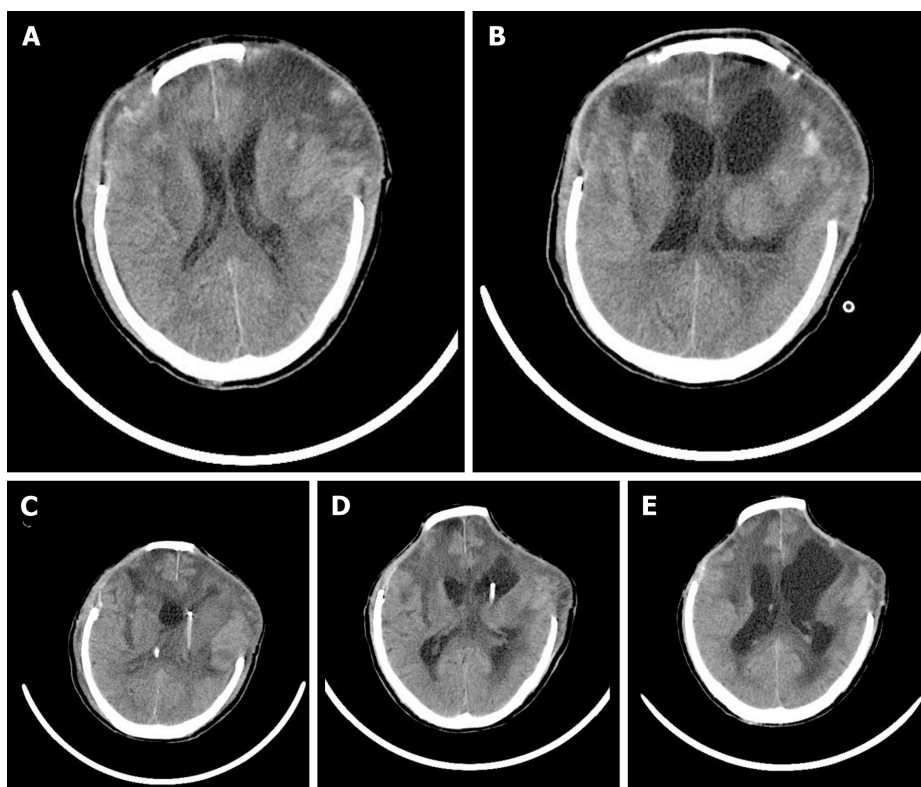


Figure 1 Computed tomography images of the patient. A: No ventricle pus was detected by head computed tomography (CT) on day 18; B: Head CT image obtained on day 24 showing ventricle pus; C: Head CT image after bilateral ventricular drainage; D: Head CT image obtained on day 30 showing that the ventricle pus had disappeared; E: Head CT image after removing the left ventricular drainage tube.

On hospital day 36, the patient's CSF culture tested negative for the first time. The IV plus IVT tigecycline was continued until hospital day 43, on which day the CSF culture was negative for the third time. Then, the occipital EVD was removed, and the patient was transferred to another hospital for rehabilitation treatment.

The timeline and antibiotics usage of the patient are shown in [Figure 2](#).

OUTCOME AND FOLLOW-UP

Three months after the car accident, the patient received a ventriculoperitoneal shunt due to hydrocephalus.

DISCUSSION

There are three categories of antimicrobial-resistant drugs, multidrug-resistant (MDR), XDR, and pandrug-resistant. XDR is defined as non-sensitivity to all penicillins and cephalosporins (including inhibitor combinations), fluoroquinolones, aminoglycosides, and carbapenems. We report the first case of pyogenic ventriculitis caused by XDR *A. baumannii* that was successfully treated by multi-route administration of tigecycline, including IV combined with CVI plus IVT.

A. baumannii is an opportunistic nosocomial pathogen that is listed by the Infectious Disease Society of America as one of the six most common MDR microorganisms in hospitals worldwide^[13]. Resistance to *A. baumannii* has been steadily increasing since the 1970s, when most strains were sensitive to commonly used antibiotics. By 2007, the proportion of MDR isolates was as high as 70%^[14]. *A. baumannii* has developed resistance to the vast majority of available antibiotics due to its ability to adapt rapidly to the environment and employ various resistance mechanisms, including the upregulation of efflux pumps, lactamases, and cell wall channels^[4,15-17]. Moreover, few antibiotics are available to treat XDR *A. baumannii* ventriculitis, as achieving effective concentrations within CSF *via* traditional administration is challenging^[3,18,19]. Hence, the treatment of MDR/XDR *A. baumannii* ventriculitis, especially pyogenic ventriculitis,

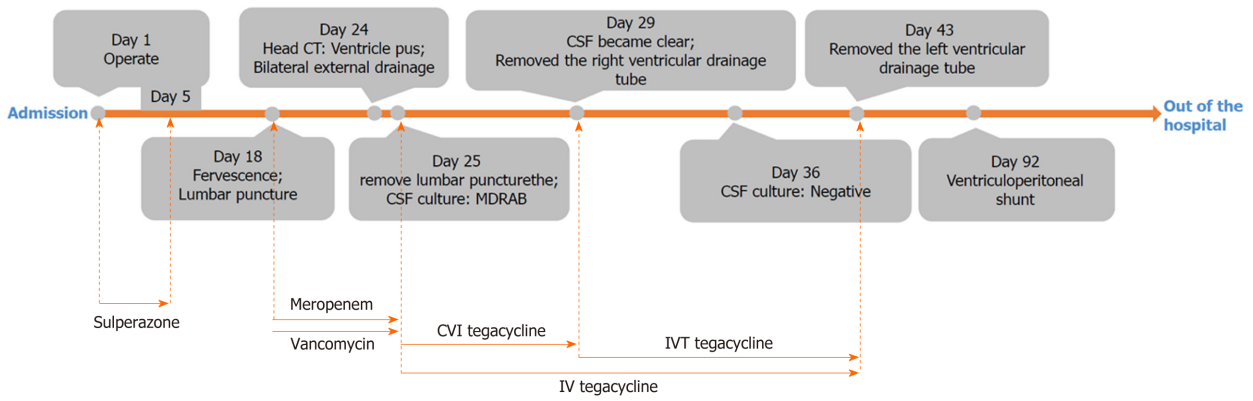


Figure 2 The timeline and antibiotics usage of the patient. IVT: Intraventricular; CVI: Continuous ventricular irrigation; CSF: Cerebral spinal fluid.

remains a challenge for neurosurgeons. It has been reported that the average CSF concentration of tigecycline is equal to 7.9% of the serum concentration^[5]. Hence, tigecycline is not commonly recommended as the first-line antibiotic for *A. baumannii* ventriculitis. In this case, the strain isolated from CSF was found to be sensitive to only tigecycline. Thus, this limited our choice of antibiotics.

Tigecycline was the first clinically available glycylicycline antibiotic that was designed to overcome MDR. The clinical use of IV tigecycline was approved in the United States in 2005, in the European Union in 2006, and in China in 2012. The first successful use of IVT tigecycline for the treatment of MDR *A. baumannii* meningitis was reported in 2016^[5]. In 2017, the first case treated with continuous saline lavage through an intraventricular drainage tube combined with intraventricular meropenem in the prone position was reported^[20]. In 2018, a case report and literature review reported the successful treatment of MDR *A. baumannii* ventriculitis with continuous ventricular lavage of tigecycline. In the case reported by Long *et al*^[21], tigecycline was administered through occipital EVD, and continued drainage was performed through a contralateral occipital EVD. In this case, in order to ensure the effective CSF concentration of tigecycline, we combined IV with CVI and IVT administration. This strategy has not been previously reported in the literature but was necessary due to the life-threatening infection and CT evidence of suppurations in the ventricles. We obtained satisfactory results as the CSF was clear on day 5 of irrigation, and CSF cultures were negative on day 12. The dose of tigecycline in the ventricle was 8 mg/d and then 6 mg/d. Previous studies have shown that the dose of tigecycline in the ventricle was 4-20 mg/d^[5,21-23]. These cases have demonstrated the safety and efficacy of this strategy.

Tigecycline is also used to treat other central nervous system infections. Soto-Hernández *et al*^[23] and Wu *et al*^[24] reported a case of intracranial infection with multidrug-resistant *Klebsiella* treated with tigecycline. And Sahin *et al*^[25] reported a child with enterococcal ventriculitis treated with tigecycline. These cases demonstrate the efficacy and safety of multi-route tigecycline in the treatment of central nervous system infections.

Tigecycline shows low toxicity, particularly for neurological adverse events. As with most other antibiotics, adverse reactions of the digestive tract are common^[26]. Additionally, no reports of adverse events following the intrathecal injection of tigecycline during the treatment of ventriculitis were noted. However, adverse reactions still cannot be ignored and can include headache, numbness, muscle spasms, weakness, *etc*^[27,28]. Controlling the rate of intrathecal administration can effectively prevent the occurrence of such complications. In this case, the speed of administration was controlled with a micropump. However, due to the lack of research on such treatment, large scale clinical studies are required to evaluate the safety of the intraventricular lavage and intraventricular tigecycline.

CONCLUSION

In summary, we report the efficacy and safety of multi-route tigecycline for pyogenic ventriculitis caused by XDR *A. baumannii*. Our findings suggest that multi-route administration of tigecycline can be a therapeutic option against pyogenic ventriculitis

caused by XDR *A. baumannii*. However, there is a lack of research on this topic, and therefore, larger samples and prospective randomized controlled trials are required to evaluate the safety of the intraventricular lavage and intraventricular administration of tigecycline.

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