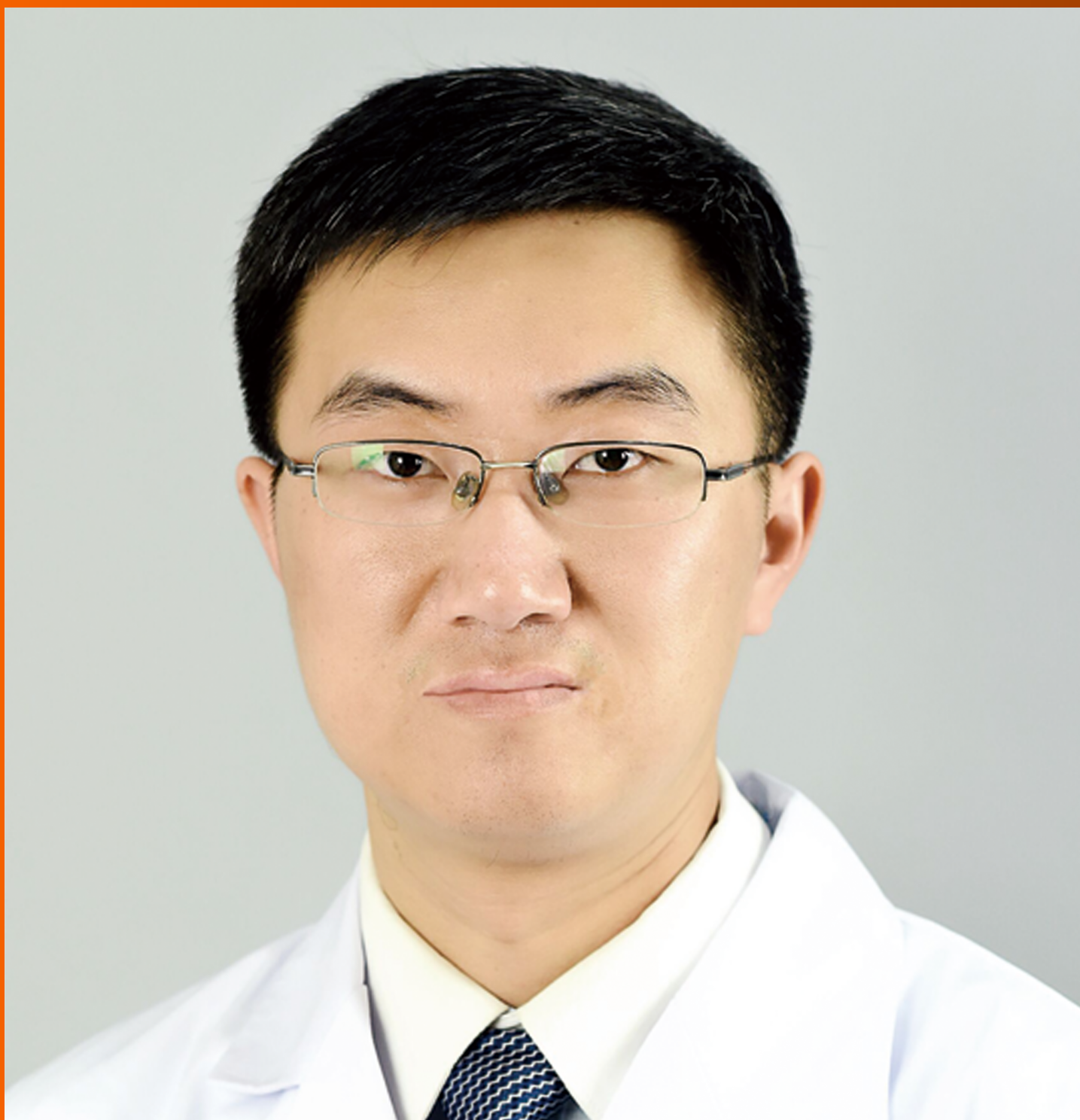


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# Rhombencephalitis caused by *Listeria monocytogenes* with hydrocephalus and intracranial hemorrhage: A case report and review of the literature

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## Abstract

### BACKGROUND

*Listeria monocytogenes* (*L. monocytogenes*), a Gram-positive facultatively intracellular bacterium, is the causative agent of human listeriosis. *Listeria* infection is usually found in immunocompromised patients, including elderly people, pregnant women, and newborns, whereas it is rare in healthy people. *L. monocytogenes* may cause meningitis, meningoencephalitis, and some very rare and severe complications, such as hydrocephalus and intracranial hemorrhage, which cause high mortality and morbidity worldwide. Up to now, reports on hydrocephalus and intracranial hemorrhage due to *L. monocytogenes* are few.

### CASE SUMMARY

We herein report a case of rhombencephalitis caused by *L. monocytogenes* in a 29-year-old man. He was admitted to the hospital with a 2-d history of headache and fever. He consumed unpasteurized cooked beef two days before appearance. His medical history included type 2 diabetes mellitus, and contaminated beef intake 2 d before onset. Cerebrospinal fluid analysis revealed Gram-positive rod infection, and blood culture was positive for *L. monocytogenes*. Magnetic resonance imaging findings suggested rhombencephalitis and hydrocephalus. Treatment was started empirically and then modified according to the blood culture results. Repeated CT images were suggestive of intracranial hemorrhage. Although the patient underwent aggressive external ventricular drainage, he died of a continuing deterioration of intracranial conditions.

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## CONCLUSION

Hydrocephalus, intracranial hemorrhage, and inappropriate antimicrobial treatment are the determinations of unfavorable outcomes.

**Key words:** Rhombencephalitis; *Listeria monocytogenes*; Central nervous system infections; Hydrocephalus; Intracranial hemorrhage; Case report

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**Core tip:** *Listeria monocytogenes* (*L. monocytogenes*) infection occurs predominantly in immunocompromised subjects. Various manifestations of listeriosis have been reported previously, but hydrocephalus and intracranial hemorrhage due to *Listeria* are rare. Hydrocephalus, intracranial hemorrhage, and inappropriate antimicrobial treatment are determinants of unfavorable outcomes. A pertinent literature review might contribute to improving our understanding of the pathogenesis and treatment of this disease.

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## INTRODUCTION

*Listeria monocytogenes* (*L. monocytogenes*) is one of the very few bacteria that can infect neurons to produce a serious and often fatal disease, with a mortality of 20%-50%<sup>[1-4]</sup>. *L. monocytogenes* infection occurs predominantly in the following populations: elderly people, pregnant women, newborns, and immunodeficient patients; patients with chronic liver disease, malignant hemopathies, and diabetes; patients on chronic hemodialysis; and, less frequently, healthy individuals<sup>[5,6]</sup>. The main routes of transmission are confirmed to be through the consumption of contaminated food and *via* vertical transmission from mother to child<sup>[7]</sup>. Penetration of the intestinal, blood-brain, blood-choroid, and fetoplacental barriers is one of the most important virulence factors of *L. monocytogenes*<sup>[8]</sup>. Therefore, the manifestations of listeriosis are varied, such as gastroenteritis, septicemia, meningitis, and other conditions.

Neurolisteriosis, a central nervous system (CNS) infection caused by *L. monocytogenes*, represents 5%-10% of listeriosis cases and is less common in the world, especially rhombencephalitis<sup>[9-11]</sup>. Hydrocephalus and intracranial hemorrhage are rare complications of listeriosis, occurring in 10%-15% and 3% of neurolisteriosis cases, respectively<sup>[12,13]</sup>. In this paper, we present a young patient with *L. monocytogenes* rhombencephalitis who presented with persistent alteration of consciousness, hydrocephalus, and intracranial hemorrhage. This case is rare due to the occurrence of hydrocephalus and intracranial hemorrhage. Cases published between 1985 and 2018 that are related to *Listeria* hydrocephalus are reviewed in Tables 1 and 2.

## CASE PRESENTATION

### Chief complaints

A 29-year-old Chinese man was admitted to the hospital with a 2-d history of intermittent fevers of up to 39 °C, and forehead headache without nausea.

### History of present illness

Two days prior to onset, he had consumed unpasteurized cooked beef that was stored in the refrigerator for a few days.

### History of past illness

His medical history included type 2 diabetes mellitus, which was poorly controlled, fatty liver, smoking, and drinking.

### Personal and family history

**Table 1** Characteristics of four cases of neonatal listeriosis with hydrocephalus published between 1989 and 2018

Ref.	Gestation/gender	CT/sonography on admission	Time to diagnosis of hydrocephalus	Other complications	Intervention	Outcome
Svare <i>et al</i> <sup>[22]</sup> , 1991	NB, 32 W/M	Not done	6 wk	Epilepsy, intraventricular hemorrhage	VPD	Moderately retarded with reduced muscular tone at 3 mo
Madlinger <i>et al</i> <sup>[25]</sup> , 1998	NB, 34 W/F	Sonography, normal	9 wk	None	VAD, VDP	Recovery
Chan <i>et al</i> <sup>[26]</sup> , 2007	NB, 31 W/M	Not done	10 d	Subtle seizure	VPD	Significant improvement
Laciar <i>et al</i> <sup>[27]</sup> , 2011	NB, 37 W/F	Not done	3 d	None	EVD	NA

NB: Newborn; W: Weeks; M: Male; F: Female; EVD: External ventricular drain; VPD: Ventriculo-peritoneal drain; VAD: Ventriculo-atrial drain; NA: Not available.

He denied a family history of hypertension and stroke.

### **Physical examination upon admission**

The physical examination was unremarkable, except for nuchal rigidity.

### **Laboratory examinations**

The blood laboratory findings showed that glucose, C-reactive protein, and erythrocyte sedimentation rate were high, while white blood cells (WBCs), red blood cells, hemoglobin, urea, creatinine, serum minerals, and autoimmune antibodies were normal. The first lumbar puncture on admission revealed a turbid cerebrospinal fluid (CSF) with 2090 leukocytes/mm<sup>3</sup> (30% neutrophils, 70% monocytes), 233.85 mg/dL protein, 1.4 mmol/L glucose (serum glucose 9 mmol/L), and pressure > 33 cmH<sub>2</sub>O. CSF Gram stain showed Gram-positive rods and was negative for fungi and acid-fast bacilli (Table 3). On the 8<sup>th</sup> day, the blood cultures yielded *L. monocytogenes*, which was susceptible to ampicillin, erythromycin, meropenem, and penicillin but resistant to sulfamethoxazole. CSF and urine cultures were negative. Repeated CSF examination on the 14<sup>th</sup> and 28<sup>th</sup> day showed a greater decrease in WBCs and protein (Table 3).

### **Imaging examinations**

The initial brain CT was unremarkable, and chest CT showed bilateral bronchopneumonia. On the 4<sup>th</sup> day of admission, magnetic resonance imaging (MRI) of the brain showed an abnormally high T2 flow attenuated inversion recovery (FLAIR) signal in the right pons and prominent temporal horns with enlargement of the ventricles (Figure 1). On the 14<sup>th</sup> day, brain CT showed hemorrhage of the right pons and hydrocephalus (bilateral lateral ventricular and the third ventricle hydrocephalus) (Figure 2). The 3<sup>rd</sup> cerebral CT was performed on the day after extraventricular drainage, revealed significant dilatation of fourth ventricle, and no remission in lateral ventricles (Figure 3). The 4<sup>th</sup> brain CT on the 29<sup>th</sup> day showed rehaemorrhage of the lateral ventricle and a larger ventricular system (Figure 4).

## **FINAL DIAGNOSIS**

The patient was finally diagnosed with *Listeria* rhombencephalitis, hydrocephalus, and intracranial hemorrhage.

## **TREATMENT**

Although empiric antibiotic therapy for bacterial meningitis (Ceftriaxone 2 g, every 12 h for 2 d, followed by meropenem 1 g, every 8 h for 2 d) and all other supportive symptomatic treatments were administered after performing blood cultures, the patient developed new symptoms with fever, sinus tachycardia, tachypnea, confusion [Glasgow Coma Scale (GCS) score 12/15], bilateral horizontal nystagmus, bilateral abducens nerve palsy, dysarthria, and weakness of all four limbs. He was transferred to the intensive care unit (ICU) on the 5<sup>th</sup> day. On the 8<sup>th</sup> day, he went into coma (GCS score 5/15), and was intubated and ventilated without autonomous respiration. According to blood cultures, new antibiotic therapy with ampicillin, etimicin and

**Table 2** Characteristics of 18 cases of non-perinatal listeriosis with hydrocephalus published between 1989 and 2018

Ref.	Age/gender	Immune-competent	CT on admission	Time to diagnosis of hydrocephalus	Other complications	Intervention	Outcome
Ulloa-Gutierrez <i>et al</i> <sup>[6]</sup> , 2004	10 Y/M	Yes	Not done	8 d	None	VPD	Recovery
Ulloa-Gutierrez <i>et al</i> <sup>[6]</sup> , 2004	3½ Y/M	Yes	Normal	5 d	None	VPD	Died
Ulloa-Gutierrez <i>et al</i> <sup>[6]</sup> , 2004	6½ Y/M	Yes	Not done	5 d	None	VPD	Died
Kasanmoentalib <i>et al</i> <sup>[12]</sup> , 2010	57 Y/M	Yes	Not done	5 d	Tracheoesophageal fistula	EVD	Severe cognitive slowness
Ito <i>et al</i> <sup>[13]</sup> , 2007	62 Y/M	No	Normal	14 d	Ventriculitis	EVD	Improvement, remained confused and disoriented
McCaffrey <i>et al</i> <sup>[20]</sup> , 2012	57 Y/M	No	Yes, hydrocephalus	1 d	Ventriculitis	EVD	NA
Dhiwakar <i>et al</i> <sup>[21]</sup> , 2007	40 Y/F	No	Not done	2 mo	Seizures, ventriculitis, basal arachnoiditis, cerebellar tonsillar herniation	VPD, VAD	Near-complete recovery
Chan <i>et al</i> <sup>[26]</sup> , 2001	42 Y/M	Yes	Yes, hydrocephalus	4 d	Subdural collection, extensive; cerebritis and ventriculitis	EVD	Died
Lee <i>et al</i> <sup>[28]</sup> , 2010	7 Y/F	Yes	Not done	10 d	None	EVD, VPD	Recovery
Platnaris <i>et al</i> <sup>[29]</sup> , 2009	7 M/M	Yes	Normal	10 d	Seizures	EVD	Normal development having achieved skills according to his age at 22 mo of age
Papandreou <i>et al</i> <sup>[30]</sup> , 2015	3 Y/F	Yes	Normal	8 d	Cerebellar tonsillar herniation, ventriculitis, and AIDP	EVD, VPD	Incomplete recovery
Gaini <i>et al</i> <sup>[31]</sup> , 2015	74 Y/M	Yes	Normal	6 d	Brain abscess	EVD	Severe sequelae, died 1 yr later
Ruggieri <i>et al</i> <sup>[32]</sup> , 2014	27 Y/F	Yes	Yes, hindbrain multifocal lesions	9 d	None	EVD	Only a motor deficit of the right arm remained
Cunha <i>et al</i> <sup>[33]</sup> , 2004	50 Y/M	Yes	Yes, hydrocephalus	1 d	None	No	Died 10 d after admission
Frat <i>et al</i> <sup>[34]</sup> , 2001	72 Y/F	Yes	Normal	12 d	Seizures	VPD	Recovery after 5 mo of rehabilitative care
Raps <i>et al</i> <sup>[35]</sup> , 1989	47 Y/F	No	Not done	Several weeks	Cervical cord compression	EVD, VPD	No significant deficit 6 mo later
Yang <i>et al</i> <sup>[36]</sup> , 2006	42 Y/M	No	Normal	9 d	Seizures	ORI	Recovery
Rana <i>et al</i> <sup>[37]</sup> , 2014	75 Y/M	No	Not done	5 d	None	VPD	Gradual recovery

M: Male; Y: Years; M: Months; F: Female; EVD: External ventricular drain; VPD: Ventriculo-peritoneal drain; VAD: Ventriculo-atrial drain; ORI: Ommaya reservoir implantation; AIDP: Acute inflammatory demyelinating polyneuropathy; NA: Not available.

meropenem was administered. On the 12<sup>th</sup> day, etimicin was discontinued as he became afebrile. We performed an extraventricular drainage to relieve hydrocephalus on the 22<sup>nd</sup> day (Figure 3). On the 29<sup>th</sup> day, because of rehaemorrhagia of the lateral ventricle, his condition rapidly deteriorated (GCS score 3/15), with anisocoria (left pupil 4 mm and right pupil 2 mm).

**Table 3 Cerebrospinal fluid analysis across disease duration**

CSF test	On the 2 <sup>nd</sup> d	On the 14 <sup>th</sup> d	On the 28 <sup>th</sup> d <sup>1</sup>
Color	Turbid	Turbid	Mildly turbid
Pressure(cm H <sub>2</sub> O)	> 33	12.5	NA
Erythrocyte count (/mm <sup>3</sup> )	0	13198	3313
WBC count (/mm <sup>3</sup> )	2090	782	85
WBC distribution (L/N)	70/30	3/97	17/68
Protein (mg/dL)	233.85	441	119
CSF glucose (mmol/L)	1.40	5.42	5.60
Plasma glucose (mmol/L)	9.00	11.05	10.0
Gram stain	Gram-positive rods	Normal	Normal

<sup>1</sup>CSF from brain ventricular draining. WBC: White blood cell; CSF: Cerebrospinal fluid; L: Lymphocytes; N: Neutrophils; NA: No data available.

## OUTCOME AND FOLLOW-UP

The patient died on the 31<sup>st</sup> day. Autopsy could not be performed.

## DISCUSSION

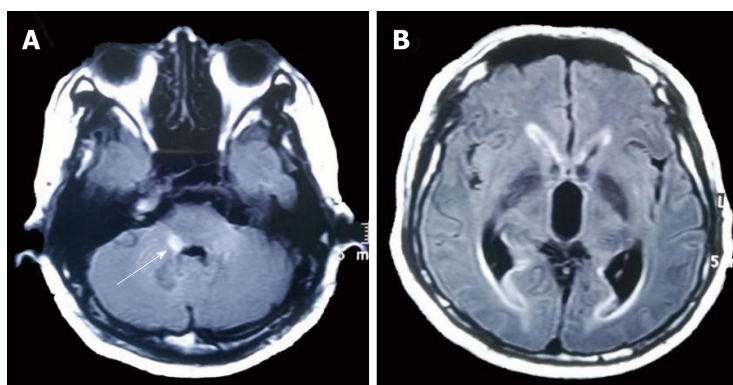
Although *L. monocytogenes* has been reported to be the third most common cause of community-acquired bacterial meningitis, following pneumococcal and meningococcal meningitis in adults, its occurrence is relatively rare, accounting for only 5% of encephalitis cases in metropolitan France<sup>[14]</sup>. *Listeria* has an important impact on public health, with high hospitalization and mortality rates despite antibiotic treatment<sup>[15]</sup>. As listeriosis is not incorporated into the national monitoring system for cases, epidemiological data on *Listeria* are scarce in China<sup>[7,16]</sup>. In a study published in 2013, Feng *et al*<sup>[16]</sup> reviewed 147 cases of listeriosis in China from 1964 to 2010, with neurolisteriosis accounting for 31% of cases. The overall case-fatality rate was 26%, highest among neonatal cases (46%) and lowest among pregnant cases (4%)<sup>[16]</sup>. In a study conducted by Wang *et al*<sup>[7]</sup>, 38 cases of listeriosis, including 5 neonatal, 8 maternal, and 25 nonmaternal cases, were reviewed in China between 1999 and 2011, and the case-fatality rates for neonatal, maternal, and nonmaternal cases were 20%, 0%, and 26%, respectively<sup>[7]</sup>.

CSF and blood cultures are the most specific for diagnosis. Early diagnosis of neurolisteriosis is difficult not only because the presentation of CSF is similar to the manifestations of other bacterial encephalitis and meningitis (pleocytosis, hyperproteinorrachia, and hypoglycorrachia) but also because approximately 50% of CSF Gram stains are negative<sup>[17]</sup>. Jubelt *et al*<sup>[18]</sup> reported that approximately three-quarters of patients have CSF pleocytosis, with approximately equal percentages of mononuclear and polymorphonuclear cells. In our case, there was an initial predominance of lymphocytic cells, which then turned to mononuclear cell predominance; this change might be related to pathological processes and the application of antibiotics. *Listeria* is usually revealed first on blood cultures, which are positive in 62% of encephalitis cases<sup>[19]</sup>. Therefore, early before antibiotic administration, repeated blood and CSF cultures are necessary and helpful for early and differential diagnoses.

*L. monocytogenes* infection most frequently presents as acute bacterial meningitis, less commonly as meningoencephalitis, and least commonly as rhombencephalitis, accounting for approximately 10% of neurolisteriosis cases<sup>[12,13]</sup>. Although the exact mechanism of rhombencephalitis remains poorly understood, *L. monocytogenes* has a well-known predilection for the brainstem. Karlsson *et al*<sup>[9]</sup> reviewed 120 patients with *Listeria* rhombencephalitis and suggested that *L. monocytogenes* enters the cerebellopontine angle through the trigeminal nerve in a subset of patients, invading the brainstem *via* the sensory trigeminal nuclei. As MRI is superior to CT in detecting subtentorial abnormal lesions, it has become more helpful for diagnosing rhombencephalitis, which has a high signal on T2-FLAIR sequences.

*L. monocytogenes* complications, such as acute hydrocephalus, hemorrhage, brain abscess, spine abscess, cerebritis, and ventriculitis, can develop, and the mortality associated with these complications is significantly high. Hydrocephalus is most common in tuberculous encephalitis but rare in listeriosis, with an approximate 3%





**Figure 1** Axial brain T2-FLAIR magnetic resonance imaging shows a hyperintense lesion of the right pons (A, white arrow), and prominent temporal horns with enlargement of ventricles (B) on the 4<sup>th</sup> d of administration.

incidence of *L. monocytogenes* meningoencephalitis in adults<sup>[13]</sup>. The exact mechanism of hydrocephalus remains unclear. The development of meningitis-associated hydrocephalus may be due to several mechanisms, such as a high level of CSF protein, impaired CSF absorption due to the obliteration of the subarachnoid space by meningeal exudates, and/or blockade of the CSF pathway by leptomeningeal inflammation<sup>[20]</sup>.

Retrospective analysis of hydrocephalus due to listeriosis is scarce at present, and most of the literature consists of case reports. The time to onset of hydrocephalus varies greatly, ranging from 1 d to 9 wk<sup>[20,21]</sup>. Ventricular drainage may not be an effective way to relieve hydrocephalus and improve survival<sup>[12,14]</sup>. A study from the Netherlands reviewed 26 hydrocephalus cases in 577 bacterial meningitis patients (4.5%), including four cases of *L. monocytogenes* (15%), all of whom underwent placement of an external ventricular drain catheter<sup>[14]</sup>. None of these patients improved clinically after catheter placement, and all had poor outcomes for hydrocephalus, with three deaths (75%) and one case of serious sequela (25%), thus indicating that patients with hydrocephalus were at a high risk for unfavorable outcomes and that hydrocephalus was an independent risk factor for death<sup>[14]</sup>. In our case, the patient underwent ventricular drainage, but a continuous improvement in cognitive function was not obvious.

Another rare complication of *Listeria* meningitis is intracranial hemorrhage, which is also one of the determinants of unfavorable outcomes<sup>[2]</sup>. Most reported cases of intracranial hemorrhage occur in infants and young children, while the condition is quite rare in adults. Svarea *et al*<sup>[22]</sup> reported a case of maternal listeriosis resulting in preterm delivery and intraventricular hemorrhage, which was diagnosed by an ultrasound scan. In a prospective study of 860 episodes with bacterial meningitis in the Netherlands, 24 (2.79%) were diagnosed with intracranial hemorrhage, with *S. pneumoniae* accounting for 67% and *L. monocytogenes* accounting for 4%<sup>[2]</sup>. The underlying pathophysiology of intraventricular hemorrhage in *L. monocytogenes* infection is still unknown and may be related to dysregulation of both the coagulation and fibrinolytic pathways and to vascular endothelial cell swelling and activation<sup>[2]</sup>.

An empirical therapy for bacterial meningitis, generally third-generation cephalosporins, is always applied at an early stage when bacterial meningitis is suspected. However, this treatment option does not cover *L. monocytogenes*. Former publications have demonstrated that inappropriate empirical antibiotic therapy leads to unfavorable outcomes<sup>[23]</sup>. Therefore, it is very important to adjust the appropriate antibiotic therapy as soon as possible once *Listeria* is highly suspected or confirmed.

*Listeria* is known to be difficult to treat, not only because *L. monocytogenes* has an intracellular life cycle but also because only a few antibiotics demonstrate activity against *Listeria*<sup>[24]</sup>. Due to the lack of multicenter clinical controlled studies, the optimal antibiotic regimen and duration for neurolisteriosis have not been definitively defined. However, amoxicillin, ampicillin, and penicillin G are generally considered effective regimens in the treatment of listeriosis<sup>[24]</sup>. The addition of aminoglycosides (such as gentamicin) could be considered a treatment regimen for *L. monocytogenes* meningitis, but its use remains controversial due to the occurrence of kidney damage<sup>[24]</sup>. The drugs should be applied at high doses, and the duration of this treatment should be extended to 21 d or longer, until complete eradication, to prevent relapse<sup>[24]</sup>. Furthermore, cotrimoxazole, rifampin, meropenem, linezolid, tetracyclines, and moxifloxacin should also be considered active against *Listeria*<sup>[23]</sup>. In our patient, the combination of ampicillin, etimicin, and meropenem was used for *Listeria*, and it

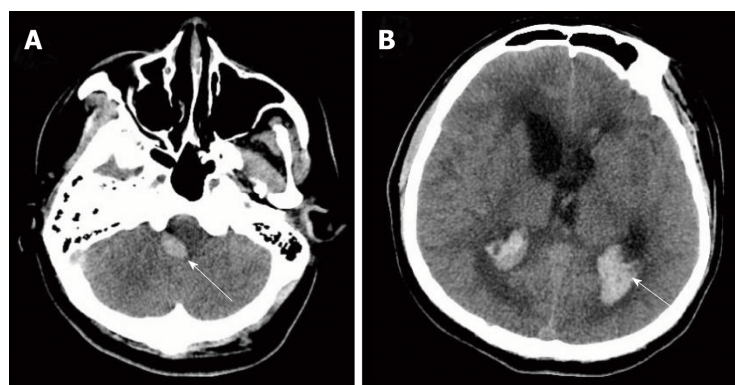


Figure 2 Axial brain computed tomography shows hemorrhage of the right pons (A, white arrow), and gross hydrocephalus and hemorrhage (B, white arrow) on the 14<sup>th</sup> d of administration.

was proven effective by repeated CSF examinations (Table 3).

## CONCLUSION

We report a case of acute hydrocephalus and intracranial hemorrhage due to complications from *L. monocytogenes* rhombencephalitis. The pathogenesis of complications has been reviewed. *L. monocytogenes* may be prone to entering the brainstem through the trigeminal nerve; hydrocephalus may be close with a high level of CSF protein and impaired CSF absorption and circulation; the occurrence of intracranial hemorrhage may be related to dysregulation of both the coagulation and fibrinolytic pathways and to vascular endothelial cell swelling and activation. Hydrocephalus, intracranial hemorrhage, and inappropriate antimicrobial treatment are the determinations of unfavorable outcomes.

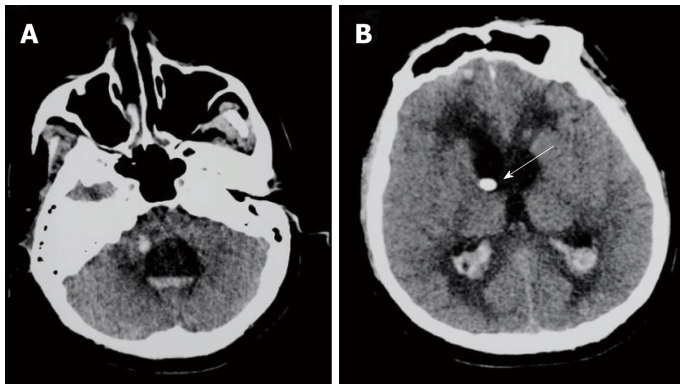


Figure 3 Axial brain computed tomography shows no improvement of hydrocephalus in the lateral ventricle on the 22<sup>nd</sup> d of administration (A and B). The ventriculostomy tube is also shown (B, white arrow).

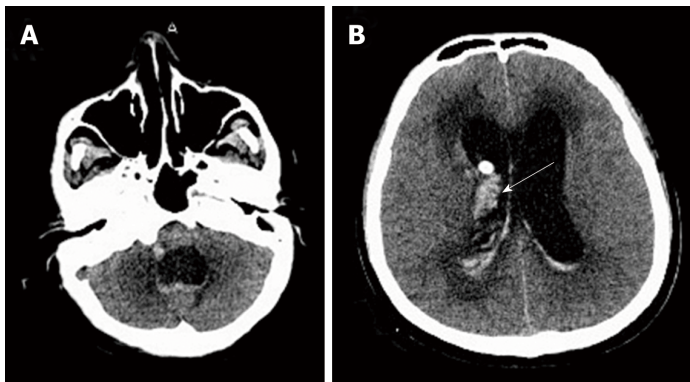


Figure 4 Axial brain computed tomography shows rehaemorrhage of the lateral ventricle and a larger ventricular system (A and B) on the 29<sup>th</sup> d of administration.

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