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

Liang JJ *et al*. *Listeria* rhombencephalitis, hydrocephalus, and intracranial hemorrhage

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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.

***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* 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%[1-4]. *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[5,6]. The main routes of transmission are confirmed to be through the consumption of contaminated food and *via* vertical transmission from mother to child[7]. Penetration of the intestinal, blood–brain, blood–choroid, and fetoplacental barriers is one of the most important virulence factors of *L*. *monocytogenes*[8]. 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[9-11]. Hydrocephalus and intracranial hemorrhage are rare complications of listeriosis*,* occurring in 10%-15% and 3% of neurolisteriosis cases, respectively[12,13]. 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***

He denied a family history of hypertension and stroke.

***Physical examination upon admission***

On admission, the physical examination was unremarkable, except for nuchal rigidity. 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 initial brain CT was unremarkable, and chest CT showed bilateral bronchopneumonia. Lumbar puncture revealed a turbid cerebrospinal fluid (CSF) with 2090 leukocytes/mm3 (30% neutrophils, 70% monocytes), 233.85 mg/dL protein, 1.4 mmol/L glucose (serum glucose 9 mmol/L), and pressure > 33 cmH2O. CSF Gram stain showed Gram-positive rods and was negative for fungi and acid-fast bacilli (Table 3).

***Laboratory examinations***

Empiric anti-inflammatory therapy for bacterial meningitis (Ceftriaxone 2 g, every 12 h for 2 d, followed by meropenem 1 g, every 8 h for 2) and all other supportive symptomatic treatments were administered after performing blood cultures. On the 4th 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). The patient was transferred to the intensive care unit (ICU) on the 5th day for further treatment because of new symptoms and poor clinical improvement.

***Imaging examinations***

The physical examination on ICU admission revealed a temperature of 38 °C, sinus tachycardia (heart rate > 150 bpm), tachypnea (respiratory rate > 30 bpm), confusion [Glasgow Coma Scale (GCS) score 12/15], bilateral horizontal nystagmus, bilateral abducens nerve palsy, dysarthria, weakness of all four limbs, and nuchal rigidity. Ampicillin combined with etimicin was administered with the diagnosis of bacterial meningitis and rhombencephalitis. On the 7th day, the patient’s mental status rapidly deteriorated to coma (GCS score 5/15), and he was intubated and ventilated without autonomous respiration. On the 8th day, the blood cultures yielded *L*. *monocytogenes*, which was susceptible to ampicillin, erythrocin, meropenem, and penicillin but resistant to sulfamethoxazole. CSF and urine cultures were negative. Meropenem was added to the combined treatment according to his blood cultures. On the 12th day, etimicin was discontinued as he became afebrile. On the 14th day, brain CT and lumbar puncture were performed again. CT showed hemorrhage of the right pons and hydrocephalus (bilateral lateral ventricular and the third ventricle hydrocephalus) (Figure 2). The lumbar puncture had an initial pressure of 12.5 cmH2O and revealed 13198 erythrocytes/mm3, 782 leukocytes/mm3 (3% neutrophils and 97% monocytes), 44.1 mg/dL protein, 5.42 mmol/L glucose (serum glucose 11.05 mmol/L), and a negative Gram stain (Table 3).

**FINAL DIAGNOSIS**

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

**TREATMENT**

The 3rd cerebral CT on the 22nd day revealed significant dilatation of all brain ventricles, and an extraventricular drainage was performed to relieve hydrocephalus (Figure 3). Repeated CSF examination from drainage on the 28th day showed a greater decrease in WBCs and protein (Table 3). On the 29th day, his condition rapidly deteriorated, with anisocoria (left pupil 4 mm and right pupil 2 mm). The 4th brain CT on the 29th day showed rehaemorrhagia of the lateral ventricle and a larger ventricular system (Figure 4).

**OUTCOME AND FOLLOW-UP**

The patient died on the 31st 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[14]. *Listeria* has an important impact on public health, with high hospitalization and mortality rates despite antibiotic treatment[15]. As listeriosis is not incorporated into the national monitoring system for cases, epidemiological data on *Listeria* are scarce in China[7,16]. In a study published in 2013, Feng *et al*[16] 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%)[16]. In a study conducted by Wang *et al*[7], 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[7].

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 hypoglycorrhachia) but also because approximately 50% of CSF Gram stains are negative[17]. Jubelt *et al*[18] 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[19]. 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[12,13]. Although the exact mechanism of rhombencephalitis remains poorly understood, *L. monocytogenes* has a well-known predilection for the brainstem. Karlsson *et al*[9] 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% incidence of *L. monocytogenes* meningoencephalitis in adults[13]. 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[20].

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[20,21]. Ventricular drainage may not be an effective way to relieve hydrocephalus and improve survival[12,14]. 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[14]. 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[14]. 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[2]. Most reported cases of intracranial hemorrhage occur in infants and young children, while the condition is quite rare in adults. Svarea *et al*[22] 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%[2]. 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[2].

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[23]. 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*[24]. 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[24]. 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[24]. 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[24]. Furthermore, cotrimoxazole, rifampin, meropenem, linezolid, tetracyclines, and moxifloxacin should also be considered active against *Listeria*[23]. In our patient, the combination of ampicillin, etimicin, and meropenem was used for *Listeria*, and it 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.

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**Figure 1 Axial brian 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 4th d of administration.**



**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 14th d of administration.**



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



**Figure 4 Axial brain computed tomography shows rehaemorrhagia of the lateral ventricle and a larger ventricular system (A and B) on the 29th d of administration.**

**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 *et al*[22],1991 | NB, 32 W/M | Not done  | 6 wk | Epilepsy, intraventricular hemorrhage  | VPD | Moderately retarded with reduced muscular tone at 3 mo |
| Madlinger *et al*[25], 1998 | NB, 34 W/F | Sonography, normal | 9 wk | None  | VAD, VDP  | Recovery |
| Chan *et al*[26], 2007  | NB, 31 W/M | Not done | 10 d | Subtle seizure  | VPD | Significant improvement  |
| Laciar *et al*[27],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.

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

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

|  |  |  |  |
| --- | --- | --- | --- |
| **CSF test** | **On the 2nd d**  | **On the 14th d**  | **On the 28th d1** |
| Color | Turbid  | Turbid  | Mildly turbid |
| Pressure(cm H2O) | >33 | 12.5 | NA |
| Erythrocyte count (/mm 3) | 0 | 13198  | 3313 |
| WBC count (/mm 3) | 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 |

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