Meningo-rhombencephalitis caused by *Listeria monocytogenes* in a healthy adult patient with a history of fresh dairy consumption: a case report

Meningo-rombencefalite causada por *Listeria monocytogenes* em um paciente adulto saudável com histórico de consumo de laticínios frescos: relato de caso

Régis Lambrecht Andrade[1], **Florisberto Lambrecht**[2]

[1]Medical student – Catholic University of Pelotas (UCPel), Pelotas, RS, Brazil;
[2]Neurosurgeon – Charity Hospital of Canguçu, Canguçu, RS, Brazil.

ABSTRACT

Introduction: *Listeria monocytogenes* is an opportunistic foodborne bacterium that can affect the central nervous system not only in immunosuppressed patients, but also in healthy individuals. For diagnostic suspicion, a complete medical history is essential, including not only the presence of diseases and the use of immunosuppressive drugs, but also dietary habits.
Objective: To report an atypical case of meningo-rhombencephalitis caused by *Listeria monocytogenes* in a healthy, non-immunosuppressed, adult patient with a history of fresh dairy consumption.
Methods: Descriptive, retrospective, observational and analytical case report study using information obtained from the patient and physical medical records.
Case description: Male patient, 43 years old, farmer, healthy, who ingested unpasteurized milk and raw cheese, presented with initial symptoms of bacterial meningitis and, despite empirical antibiotic therapy, he developed signs of rhombencephalitis and obstructive hydrocephalus, requiring external ventricular drainage (EVD). With the identification of *Listeria monocytogenes* in the cerebrospinal fluid (CSF), it was possible to change the antibiotic to ampicillin, and the patient began to show favorable progress, becoming asymptomatic.
Conclusion: Although rare in non-immunosuppressed patients and lacking a typical presentation, neurolisteriosis should be included in the differential diagnosis of patients with central nervous system infection, especially those involved in the production and/or consumption of dairy products. Cultural tests are essential for identifying the bacterium, and early use of specific antibiotics is crucial for reducing morbidity and mortality rates associated with this disease.

Keywords: *Listeria monocytogenes*, laticínios, listeriose, encefalite, hidrocefalia, imunocompetência, ampicillina.

RESUMO

Introdução: Listeria monocytogenes é uma bactéria oportunista de transmissão alimentar que pode acometer o sistema nervoso central não apenas em pacientes imunossuprimidos, mas também em indivíduos saudáveis. Para a suspeita diagnóstica, uma história clínica completa é essencial, incluindo não apenas a presença de doenças e o uso de medicamentos imunossupressores, mas também os hábitos alimentares.
Objetivo: Relatar um caso atípico de meningo-romboencefalite causada por Listeria monocytogenes em um paciente adulto, saudável, não imunossuprimido, com histórico de consumo de laticínios frescos.
Métodos: Estudo descritivo, retrospectivo, observacional e analítico do tipo relato de caso, utilizando informações obtidas do paciente e de prontuários médicos físicos.
Descrição do caso: Paciente do sexo masculino, 43 anos, agricultor, previamente saudável, que ingeriu leite não pasteurizado e queijo cru, apresentou sintomas iniciais de meningite bacteriana e, apesar da antibioticoterapia empírica, evoluiu com sinais de romboencefalite e hidrocefalia obstrutiva, necessitando de drenagem ventricular externa (DVE). Com a identificação de *Listeria monocytogenes* no líquido cefalorraquidiano (LCR), foi possível substituir o antibiótico por ampicilina, e o paciente passou a apresentar evolução favorável, tornando-se assintomático.
Conclusão: Embora rara em pacientes não imunossuprimidos e sem apresentação clínica típica, a neurolisteriose deve ser incluída no diagnóstico diferencial de pacientes com infecção do sistema nervoso central, especialmente daqueles envolvidos na produção e/ou no consumo de produtos lácteos. Os testes microbiológicos são essenciais para a identificação da bactéria, e o uso precoce de antibióticos específicos é crucial para reduzir as taxas de morbidade e mortalidade associadas a essa doença.

Palavras-chave: *Listeria monocytogenes*, laticínios, listeriose, encefalite, hidrocefalia, imunocompetência, ampicillina.

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Corresponding authors:
1. Régis Lambrecht Andrade, regis.andrade@sou.ucpel.edu.br
2. Florisberto Lambrecht, drflorisneuro@hotmail.com

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INTRODUCTION

Listeria monocytogenes is a bacterium that can be transmitted to humans through the ingestion of contaminated food and, less frequently, vertically[1].

Human listeriosis most commonly manifests as self-limiting gastroenteritis, but occasionally it can be invasive with systemic dissemination, potentially affecting the central nervous system (CNS)[1]. It is a cosmopolitan disease that mainly affects newborns, the elderly, pregnant women and immunosuppressed patients, rarely affecting healthy individuals[2,3]. Although the diagnosis of neurolisteriosis may be challenging, prompt initiation of appropriate therapy is crucial for improving both survival and functional outcomes[4].

This report describes the case of a healthy adult with the habit of consuming unpasteurized milk and raw cheese, who presented meningo-rhombencephalitis caused by *L. monocytogenes*, complicated by obstructive hydrocephalus. With the use of ampicillin and an external ventricular drain, he progressed favorably and became asymptomatic, which is a rare outcome.

CASE REPORT

A 43-year-old man, farmer, healthy, presented to the hospital experiencing a diffuse headache and nausea, progressing to vomiting, fever (up to 40.2 °C) and difficulty moving his neck and walking. He reported the symptoms started after strenuous labor activity under hot weather conditions 5 days earlier. Upon admission to the hospital, on day 5 of the illness, he was drowsy, alert, isochoric, with neck stiffness, no cranial nerve abnormalities, preserved strength, reflexes, no sensitivity loss and mild gait ataxia. Laboratory tests showed hemoglobin at 13.2 g/dL, 19,700 white blood cells (WBC)/mm^3, 4% bands, 68% segmented cells, 11% lymphocytes, 124,000 platelets/mm^3, urea at 46 mg/dL, and creatinine at 1.2 mg/dL. A head computed tomography (CT) (Figure 1A) revealed no abnormalities. Cerebrospinal fluid (CSF) obtained by lumbar puncture was xanthochromic and slightly turbid, with mild pleocytosis (20 WBC/mm^3), 90% polymorphonuclear and 10% mononuclear cells, 320 red blood cells/mm^3, glucose 5 mg/dL and protein 199 mg/dL (Table 1). Treatment was started with ceftriaxone 2 g IV, every 12 hours, and dexamethasone 4 mg IV, every 6 hours, in addition to symptomatic medications.

On day 7, he presented diplopia due to left sixth cranial nerve palsy, and gait ataxia worsened.

On day 8, he presented decreased sensory function, generalized tonic-clonic seizures and nystagmus. Cranial magnetic resonance imaging (MRI) (Figure 1B) showed obstructive hydrocephalus, with dilation of the lateral ventricles and mesencephalic duct, with ependymal transudation, signal alteration in the midbrain (rhombencephalitis), and perineural edema near the optic nerves. A right frontal external ventricular drain (EVD) was placed. Cerebrospinal fluid (CSF) obtained at that time (Table 1) was xanthochromic and turbid, with increased pleocytosis (69 WBC/mm^3), 87% mononuclear cells, 3% eosinophils, 10% polymorphonuclear cells, glucose 40 mg/dL and protein 118 mg/dL, indicating progression of pleocytosis despite 3 days of ceftriaxone therapy. The culture results for the first CSF sample (Table 1), obtained on the same day, revealed the presence of *Listeria monocytogenes*; the polymerase chain reaction (PCR) test of the CSF was negative for other pathogens such as *Neisseria meningitidis, Streptococcus pneumoniae or Haemophilus influenzae*. A review of the patient's history revealed he had consumed raw homemade cheese made from cow's milk 15 days earlier and that he also had a habit of drinking fresh milk. He had not experienced any abdominal pain or diarrhea. Ceftriaxone was discontinued, and ampicillin 2 g IV every 4 hours was started. HIV and VDRL were negative. The patient was transferred to the intensive care unit (ICU).

On day 10, he was fully awake, speaking a few simple sentences, recognizing family members, with isochoric and photoreactive pupils, still exhibiting strabismus due to left sixth nerve palsy, and presenting with hyponatremia (127 mEq/L).

On day 12 of disease progression, a CT scan was repeated (Figure 1C), which showed the EVD catheter positioned in the anterior horn of the right lateral ventricle and a reduction in hydrocephalus. The CSF (Table 1) showed decreased pleocytosis (20 WBC/mm^3), 10% mononuclear and 90% polymorphonuclear cells, glucose 57 mg/dL and protein 15 mg/dL.

On day 15, the nasogastric (NG) tube was removed, and the patient resumed oral feeding.

On day 20, the EVD system was closed; the patient's intracranial pressure (ICP) remained at 12 cmH_2O. Another CT scan was performed (Figure 1D), which showed ventricles with normal morphology and near-normal dimensions. The last CSF sample was collected (Table 1) by the EVD on day 14 of ampicillin treatment. The fluid was already clear, colorless, with no cellular presence, with a glucose level of 71 mg/dL and a protein level of 21 mg/dL. The EVD was removed. The latest CT scan (Figure 1E), taken 5 days after discontinuing EVD, was normal.

The patient was discharged from the hospital after 21 days of ampicillin treatment; he was alert and oriented, with mild paresis of the left lateral rectus muscle, mild nystagmus in the horizontal gaze, mild neck stiffness, and gait ataxia, but he was able to walk without assistance and did not require a ventriculoperitoneal (VP) shunt. The blood test results were as follows: Ht: 39.5%; Hb: 13.6 g/dL; white blood cells: 6,310 cells/mm^3; bands: 2%; segmented cells: 57%; lymphocytes: 23%; platelets; 301,000/mm^3; urea: 23 mg/dl; creatinine: 0.8 mg/dL; Na^+: 137 mEq/dL; K^+: 4.1 mEq/dL; glucose: 78 mg/dL; ESR: 6 mm; and CRP: less than 6 mg/dL. The patient was instructed to continue phenobarbital at a dose of 100 mg nightly.

Two months later, the patient was completely asymptomatic, with no strabismus, and working as usual on his farm.

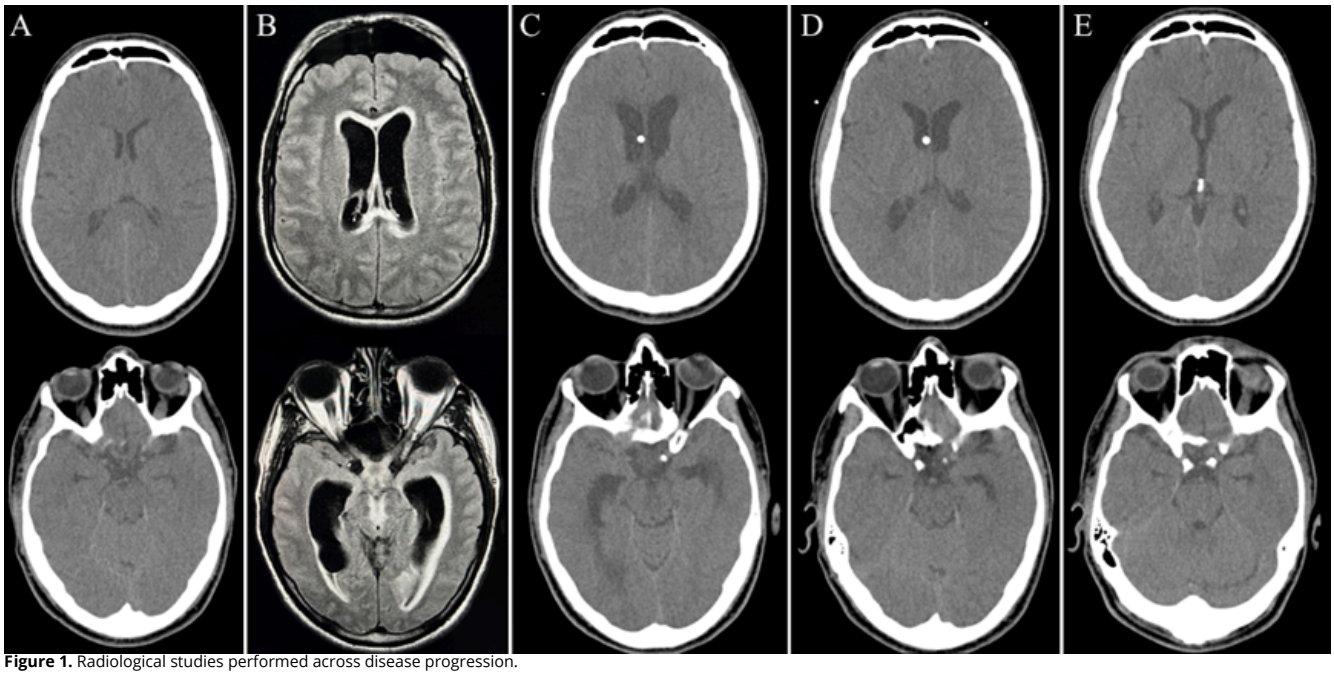

Figure 1. Radiological studies performed across disease progression.
Figure 1A. Initial head CT scan with normal findings.
Figure 1B. Brain MRI, with enlarged lateral ventricles and mesencephalic duct suggestive of acute obstructive hydrocephalus, as well as a signal alteration in the midbrain (rhombencephalitis), and perineural edema near the optic nerves.
Figure 1C. Head CT scan showing EVD in the frontal horn of the right lateral ventricle, with hydrocephalus reduction.
Figure 1D. Head CT scan showing EVD in the frontal horn of the right lateral ventricle, with normal morphology and near-normal dimensions.
Figure 1E. Head CT scan 5 days after EVD removal, showing no evidence of hydrocephalus.

Table 1 – Antibiotic therapy and cerebrospinal fluid analysis across disease duration

Days from symptoms onset	5	8	12	18	22
Antibiotic day	1st ceftriaxone	1st ampicillin	4th ampicillin	10th ampicillin	14th ampicillin
CSF					
Color	Xanthochromic	Xanthochromic	Colorless	Colorless	Colorless
Appearance	Slightly turbid	Slightly turbid	Clear	Clear	Clear
RBC (/mm^3)	320	25	8	10	0
WBC (/mm^3)	20	69	20	5	0
MNC (%)	10	10	10	45	-
PMN (%)	90	90	90	55	-
Glucose (mg/dl)	5	46	57	71	71
Protein (mg/dl)	199	109	56	32	21
Culture	*L. monocytogenes*	-	-	-	-

RBC: Red blood cells; WBC: White blood cells; MNC: Mononuclear cells; PMN: Polymorphonuclear cells.

DISCUSSION

L. monocytogenes is a Gram-positive, rod-shaped, intracellular, non-spore-forming, non-encapsulated, facultative anaerobic, motile and opportunistic bacterium. It is ubiquitous and can be found in soil, water, decomposing plant matter, contaminated foods such as dairy products (cheese and milk) and meat products (both ready-to-eat and raw), fresh vegetables and food production environments[2]. This bacterium is capable of growing and surviving under adverse pH conditions, low temperatures, and high salt concentrations — barriers commonly used in the food industry to inhibit the growth of pathogenic microorganisms[14,15]. However, it is sensitive to pasteurization[2,14].

Listeriosis is a foodborne disease (FBD)[1,2,14,15]. The

main sources of listeriosis are dairy products, which are often consumed raw[14]. It is a cosmopolitan disease that primarily affects newborns, the elderly, pregnant women and immunosuppressed patients; although it is considerably rare in immunocompetent individuals, isolated cases have also been reported[3,9,14].

The incubation period for the bacillus ranges from 3 to 70 days, which may complicate identification of the infection source[14]. Listeriosis presents as a self-limiting diarrheal syndrome or, in susceptible patients, as an invasive disease, including sepsis and, in particular, CNS infections affecting various anatomical sites. CNS manifestations include meningitis, nonspecific meningoencephalitis, rhombencephalitis, myelitis, cerebritis and brain abscesses[8]. Infections outside the CNS, such as arthritis, endocarditis, hepatitis, liver abscess, biliary tract disease, peritonitis, osteomyelitis, endophthalmitis and skin infections, may rarely occur[15].

Vertical transmission[1,14,15], transplacental transmission, or transmission via the colonized birth canal may occur, potentially leading to spontaneous abortion, stillbirth, prematurity, sepsis, skin rash, liver and spleen dysfunction, and central nervous system infection[15].

The most common manifestation of neurolisteriosis, accounting for more than 90% of cases, is meningitis[1], which may present similarly to other forms of bacterial meningitis[12]. Clinical features vary widely, ranging from fever and nonspecific alterations in mental status to severe coma. It should be noted that signs of meningeal irritation occur in 40% of patients[8]. The atypical clinical presentation may delay diagnosis and, consequently, further worsen the course of the disease, especially in immunosuppressed patients[8]. The rarest forms are acute or chronic meningoencephalitis, rhombencephalitis, cerebritis, and brain or spinal cord abscesses[1].

Rhombencephalitis is a rare form of neurolisteriosis[4] that affects the brainstem, and its nonspecific early symptoms may hinder prompt diagnosis[12]. Initial features commonly include fever, headache, nausea and vomiting, whereas signs of meningeal irritation are less frequent. Subsequently, patients develop multiple cranial nerve abnormalities accompanied by cerebellar dysfunction, including ataxia. Fever may be absent in 15% of patients, which complicates diagnosis and makes the condition more suggestive of non-infectious diseases[15]. Prompt diagnosis of rhombencephalitis is critical, given the 100% mortality rate in untreated cases; however, it is often challenging[4] and delayed, relying primarily on culture results and brain MRI findings[1].

L. monocytogenes may also cause brain abscesses, accounting for 1–10% of reported cases of CNS listeriosis, and should be considered in the differential diagnosis of hematogenous brain abscess in immunocompromised patients who fail to respond to cephalosporin therapy[11].

For the diagnostic confirmation of neurolisteriosis, it is necessary to isolate the bacterium from sterile body fluids, such as blood or cerebrospinal fluid (CSF)[5,8,15]. CSF collection should not be delayed, as this could compromise targeted treatment and, consequently, the patient's prognosis[5]. In rhombencephalitis, the initial CSF profile may not show significant abnormalities, and typically more than 50% of CSF cultures are negative[3,4]. An infectious cause for the signs and symptoms observed may thus be mistakenly discarded[4]. The CSF analysis reveals changes similar to those seen in other meningeal infections: pleocytosis (with a predominance of polymorphonuclear or lymphomononuclear cells), elevated protein levels, and reduced glucose levels[1,5]; however, findings consistent with bacterial meningitis are less common in *L. monocytogenes* meningitis than in other causes of bacterial meningitis[10].

In cases of clinical suspicion, blood cultures should be obtained before initiating treatment, given their high diagnostic yield. Blood cultures are positive in 85% of cases, whereas CSF microbiological testing is positive in only one-third of cases and typically yields results later[1].

Serological findings are often unremarkable, with leukocytosis observed in only about 31% of cases[1].

Polymerase chain reaction (PCR) assays have recently shown promise as an alternative diagnostic tool in cases of high clinical suspicion in which cultures are non-diagnostic[8].

Regarding imaging evaluation, brain MRI is far superior to computed tomography (CT) for the diagnosis of rhombencephalitis, as CT scans may initially appear normal[4].

Penicillins are the treatment of choice for L. monocytogenes infection; the most recommended option is high-dose ampicillin (9 g/day) for at least 21 days, which may be extended to up to 6 weeks in cases of brain abscess. Combination therapy with ampicillin and gentamicin, intended to achieve a synergistic effect, has been described; however, results are inconsistent, possibly due to the limited penetration of gentamicin into the cerebrospinal fluid[5,8]. In patients with penicillin allergy, sulfamethoxazole/trimethoprim (SMX-TMP) is an appropriate alternative[8].

It is important to emphasize the strong association between TNF-α antagonists and invasive listeriosis, to ensure that early empirical therapy is initiated in cases of meningoencephalitis in at-risk patients, thereby preventing adverse outcomes[6].

The use of corticosteroids in patients with L. monocytogenes meningoencephalitis has yielded mixed results, and there is insufficient evidence to support their prescription[8].

In patients with rhombencephalitis, close monitoring is recommended due to the risk of respiratory arrest[12].

In cases of *L. monocytogenes* brain abscess, the optimal duration of antibiotic therapy will depend on the underlying disease, clinical severity, the number and size of the abscesses, and the appropriateness of surgical

treatment[11]. Immunocompromised patients with CNS infection should continue antibiotic treatment for at least 6–8 weeks until cultures are negative and/or brain imaging shows improvement[11]. Surgery may be considered in specific cases[1]. Reduction of immunosuppressive therapy should be considered, particularly in patients with inadequate clinical response[11].

CNS infections by *L. monocytogenes* are more likely to be associated with neurological complications such as subdural effusion, recurrent seizures, hemiparesis, cranial nerve palsy, hydrocephalus, brain abscess and purulent ventriculitis[5]. In the reported case, the patient developed left sixth nerve palsy on day 7 of illness, followed by supratentorial obstructive hydrocephalus on day 8, thus requiring drainage.

Hydrocephalus is a recognized complication of *L. monocytogenes* meningoencephalitis[3,7,13] and is associated with significant morbidity and mortality[13]. The etiology of hydrocephalus is likely multifactorial, involving elevated CSF protein levels and impaired absorption due to obliteration of the subarachnoid space by meningeal exudates or impaired CSF absorption in the arachnoid granulations caused by a severe inflammatory reaction[3,13]. After *S. pneumoniae, L. monocytogenes* is the second most common pathogen causing hydrocephalus[13]. It occurs in 10–15% of cases, being an independent risk factor for death[3]. Other prognostic factors include extreme ages, delayed initiation of treatment, immunosuppression and the presence of brain abscesses and rhombencephalitis[1].

Another rare complication is intracranial hemorrhage, which is also a factor contributing to adverse outcomes. Most reported cases occur in infants and young children, whereas the condition is rare in adults[3]. Ventriculitis is also an uncommon complication and may present with increased intracranial pressure[10].

The mortality rate among patients with neurolisteriosis is estimated at 30%, with many survivors experiencing long-term sequelae[7,8]. The mortality rate reported for rhombencephalitis is 40%, which may decrease to 24% with timely treatment. Approximately 55% of patients develop neurological sequelae, and 34% experience respiratory failure[1].

An additional consideration is brucellosis, which represents an important differential diagnosis in patients consuming fresh dairy products such as raw milk and soft cheeses, as ingestion of unpasteurized dairy products remains the most common route of transmission[16]. Brucellosis is a worldwide occupational zoonosis, typically affecting veterinarians, abattoir workers, shepherds and professionals of the dairy industry[16,17]. Although it presents with a wide spectrum of clinical manifestations, central nervous system involvement occurs in up to 10% of cases and may include meningitis, meningoencephalitis and brain abscesses, and, more rarely, meningovascular and demyelinating syndromes[16]. CNS involvement is associated with a potentially severe prognosis. When present, cranial nerve involvement most commonly affects the sixth and eighth nerves[17]. To date, no single definitive diagnostic test exists; therefore, diagnosis is made through clinical evaluation and routine laboratory tests, combined with culture, serology or polymerase chain reaction analysis of the CSF[17]. A confirmed diagnosis requires isolation of the bacterium from blood or tissue samples, with blood and bone marrow specimens considered the most sensitive[16,17]; however, reported positivity rates range from 15% to 70%[16].

In the presented case, the initial clinical history was consistent with meningitis: the first CSF sample revealed mild pleocytosis with a predominance of polymorphonuclear cells, suggesting early-stage bacterial meningitis. Significantly low glucose levels and elevated protein levels prompted initiation of ceftriaxone therapy, as the patient was a healthy adult with no history of immunosuppression. The patient's condition worsened, with the onset of gait ataxia and sixth nerve palsy, consistent with rhombenencephalitis, as well as acute hydrocephalus, thus requiring placement of an EVD. CSF obtained from the EVD also demonstrated pleocytosis, with a predominance of polymorphonuclear cells, indicating that, despite the administration of ceftriaxone, both clinical and cerebrospinal fluid parameters worsened. The first CSF culture revealed *L. monocytogenes*, prompting discontinuation of ceftriaxone and initiation of ampicillin therapy. Review of the patient's history revealed consumption of raw cheese 15 days earlier, in addition to regular intake of unpasteurized cow's milk. Brucellosis was discarded due to the absence of other clinical manifestations commonly associated with this condition, the positive CSF culture for *L. monocytogenes* and clinical improvement following ampicillin treatment. Dexamethasone was administered for the first 7 days, and although CSF analysis on day 14 normalized, ampicillin was continued for a total of 21 days. The authors consider acute hydrocephalus to have represented the greatest risk for mortality, and the favorable outcome in this case was likely due to early identification of the pathogen in the first CSF sample and prompt initiation of appropriate antibiotic therapy.

CONCLUSION

This report describes a rare case of meningo-rhombencephalitis caused by *L. monocytogenes* in a 43-year-old, otherwise healthy farmer. Early identification of the bacterium in the initial CSF culture was crucial for establishing the diagnosis, allowing prompt initiation of ampicillin therapy and resulting in an excellent clinical outcome. Despite the acute obstructive hydrocephalus complication, the timely placement of an EVD resolved the complication without the need for a subsequent ventriculoperitoneal shunt.

Although *L. monocytogenes* most frequently causes CNS infections in immunosuppressed individuals, it should

also be considered as a causative agent in otherwise healthy patients who fail to respond to conventional empirical therapy with third-generation cephalosporins, particularly those who regularly consume fresh foods — especially dairy products such as milk and cheese —, which are the primary sources of human exposure.

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