

Listeria Monocytogenes Meningitis: Two Case Reports and Literature Review

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Abstract

Introduction: *Listeria monocytogenes* is a pathogen widely distributed in nature, but its clinical manifestations are uncommon, being more relevant in populations with low immunity. This bacterium is acquired through improper food handling and mainly presents itself in the form of meningitis and septicemia. **Methods:** Retrospective survey of patients with *L. monocytogenes* meningitis at Couto Maia Hospital, a reference center for infectious diseases in the state of Bahia, between 1972 and 2009. Two reported cases will be the focus of the study. **Case series:** Case 1: male, 20 years old, previously healthy, presented with headache, fever, vomiting, and signs of meningeal irritation. Lumbar puncture, cerebrospinal fluid (CSF) study and culture, and complete blood count were performed. Culture was positive for *L. monocytogenes*. Treated with ceftriaxone, progressing to death. Case 2: female, 15 years old, diagnosed with juvenile rheumatoid arthritis, presented with headache, fever, and nausea. CSF culture positive for *L. monocytogenes*. Antibiotic therapy was conducted with changes in the course and remission of the condition. Treated with ampicillin and ceftriaxone. **Discussion:** Meningitis and sepsis are the most common manifestations caused by *L. monocytogenes*. After escaping the immune system, the pathogen reaches the bloodstream and central nervous system. The clinical presentation is similar to that of meningitis caused by other etiologies. CSF shows pleocytosis, hyperproteinorrhachia, and a tendency toward hypoglycorrhachia. The antibiotic of choice is ampicillin, and the outcome is multifactorial. **Conclusion:** *L. monocytogenes* meningitis is uncommon but should be considered in immunocompromised patients.

Keywords: listeria monocytogenes; listeria meningitis; meningitis; case report

Introduction

L. monocytogenes is described as a gram-positive, aerobic, and facultative anaerobic bacillus that can be isolated in organic fluids such as cerebrospinal fluid, blood, or amniotic fluid¹. It is a facultative intracellular bacterium, and this location is a protective factor against the host's humoral response². This bacterium is widely distributed in nature and can be found in soil, decaying vegetables, and tap water [1,3,4,26,30]. It is mainly acquired through the ingestion of undercooked foods, vegetables, milk and dairy products, fish, and chicken [2,4,5].

The clinical aspects of listeriosis are pleomorphic, with subacute, acute, and chronic presentations possible, depending on the site of infection in humans [6]. Thus, symptoms can range from flu-like symptoms to fulminant meningoencephalitis and sepsis [2,7]. Meningitis caused by *L. monocytogenes* is one of the most common manifestations of listeriosis, the other being septicemia [6,8-11]. *L. monocytogenes* meningitis is important because it mainly affects newborns, pregnant women, the

elderly, alcoholics, and immunocompromised individuals (patients with acquired human immunodeficiency syndrome, transplant recipients, diabetics, and patients with neoplasms), but it can also occur in immunocompetent individuals [2,3,5,6,8,9,12-14,26,27].

Objectives

This study aims to describe the demographic, clinical, laboratory, and therapeutic aspects of two cases of *L. monocytogenes* meningitis that occurred between 1972 and 2009 at Couto Maia Hospital (HCMaia), a referral hospital for infectious diseases in the state of Bahia.

Methods

This study is a descriptive case report that describes the demographic, clinical, laboratory, and therapeutic data of the cases studied. To constitute the sample, cases of meningitis caused by *L. monocytogenes* were collected from the records of the Medical and Statistical Archive Service and, simultaneously, from the results of cerebrospinal fluid (CSF)

cultures with growth of *L. monocytogenes* in the Biochemistry Laboratory, in the period between 1972 and 2009 at HC Maia. A literature review was conducted in Scielo, Pubmed, and Bireme, using the keywords meningitis, *listeria monocytogenes*, listeriosis, and neurolisteriosis in Portuguese, English, and Spanish, for the period between 1970 and 2025.

Case Studies

Case 1

A 20-year-old male patient, born and raised in Porto Sauípe, Bahia, sought care at a local health clinic with a 12-hour history of intense, holocranial headache with photophobia. He was prescribed dipyrone, but his condition did not improve. He developed a fever (39°C), at which point he sought another health service and was referred to HC Maia for a CSF study. On the way, he had two episodes of vomiting. Upon admission to HC Maia, he reported anorexia and asthenia. As a history, his mother reported measles, chickenpox, and mumps in childhood. He reported living in a residence without basic sanitation and electricity and working in farming and cattle raising.

Physical examination revealed torpor, hypochromia (+/4+), temperature of 36.5°C, systemic blood pressure of 130X80 mmHg, palpable tonsillar lymph nodes, and signs of meningeal irritation (neck stiffness, positive Lasegue and Kernig signs).

The cerebrospinal fluid study revealed a cloudy appearance, 6,000 cells/mm³ with a predominance of polymorphonuclear cells, glucose <20mg%, positive globulins, and proteins of 250mg%. The cerebrospinal fluid culture was positive for *L. monocytogenes*. The blood count showed hematocrit 43.7%, hemoglobin 14.3g%, leukocytes at 17,400 (with 2% rods, 72% segmented, 22% lymphocytes, 4% monocytes).

He was treated with 2 g of ceftriaxone intravenously every twelve hours and 4 mg of dexamethasone every six hours. The patient progressed with fever, episodes of vomiting, disorientation, agitation, followed by hypoactivity, progressing to torpor, culminating in death on the fourth day of hospitalization.

Case 2

Female patient, 15 years old, student, born in Ilhéus, Bahia, coming from Salvador, Bahia. She was admitted to HC Maia complaining of severe headache for twenty-four hours. This patient had a previous diagnosis of juvenile rheumatoid arthritis, with pain, edema, and heat in her wrists and ankles, and had been taking 10 mg/week of methotrexate for a year and a half. The patient reported worsening arthritis and mood swings fifteen days ago, progressing seven days later with mild headache, predominantly frontal, responsive to analgesics, but recurrent. There is a report of fever forty-eight hours prior to admission, accompanied by nausea, vomiting, and worsening headache twenty-four hours ago. Her medical history included bacterial pneumonia one year ago, amenorrhea for two months, chronic anemia treated with polymaltose ferric hydroxide complex with folic acid, and allergy to acetylsalicylic acid. She reported living in a house with basic sanitation but in poor socioeconomic conditions.

Physical examination revealed fair general condition, facial expression of acute distress, pain on palpation of chondrosternal joints, soft, painless subcutaneous nodules on both feet, deformity and difficulty in extending the fingers, claw-like position of the toes, absence of meningeal or localized signs.

A lumbar puncture was performed. The cerebrospinal fluid study showed a cloudy appearance, 3,200 cells/mm³ with a predominance of neutrophils, glucose of 20mg%, positive globulins, and proteins of 200mg%. The cerebrospinal fluid culture was positive for *L. monocytogenes*. The direct smear study was negative. The complete blood count showed hemoglobin of 8.0 g%, hematocrit of 25%, hypochromia ++, anisocytosis +, leukocyte count of 4,400 cells/mm³ (3%

rods, 80% segmented, 15% lymphocytes, 2% monocytes), erythrocyte sedimentation rate of 63 mm in the first hour, CRP positive.

The patient was initially treated with ampicillin, but the antibiotic was changed to ceftriaxone on the second day of hospitalization (HD), combined with chloramphenicol on the sixth HD, and ceftriaxone was changed back to ampicillin on the tenth HD, maintaining the combination with chloramphenicol until the nineteenth HD. Prednisone 25 mg/day was administered orally on the first and ninth to nineteenth HD, and dexamethasone was administered intravenously every six hours between the second and eighth HD. He also received indomethacin 100 mg PO/day, methotrexate 5 mg PO on Mondays and Tuesdays, and polymaltose ferric hydroxide complex with folic acid orally, one tablet every twelve hours.

She developed diffuse abdominal pain, fluctuations in consciousness, fever spikes (39-40°C), strabismus, diplopia, and signs of meningeal irritation (neck stiffness, positive Lasegue, Brudzinski, and Kernig signs), improving on the seventh day. On the nineteenth day of hospitalization, she was pain-free and lucid, and was discharged due to improvement, continuing on indomethacin, prednisone, and methotrexate, with instructions for outpatient follow-up.

Discussion

Meningitis and sepsis, as reported by several authors, are the most common manifestations of *L. monocytogenes* infection [1,6,7,15,16]. In the literature, studies show evidence of a link between these presentations and their follow-up with a previous state of immune system compromise [1,2,3,5,6,11,13-19], since the host response to this infection is primarily based on the cell-mediated response [1,7,15]. Thus, in patients with certain pathologies, such as alcoholism, diabetes, acquired human immunodeficiency syndrome, neoplasms, and collagenoses, as well as in non-pathological situations, such as neonates, pregnant women, and the elderly, infections by this agent are more common [1,2,6,7,10,11,19]. Thus, the patient described in case 2 had a condition favorable to infection by *L. monocytogenes*, since he had juvenile rheumatoid arthritis and was using methotrexate, a drug that can depress hematopoiesis and cause anemia, leukopenia, and/or thrombocytopenia, leading to a state of immunodeficiency. However, in the first case, no immunodeficiency was identified, reflecting the possibility of infection even in previously healthy patients, which has been observed in previous studies [2,3,5,6,8,9,12-14].

Regarding incidence, it is stated that *L. monocytogenes* infections were initially considered zoonoses, but currently, a higher frequency of these infections has been described in urban areas, ruling out the need for contact with animal handling areas for their acquisition¹. Thirteen serovars of the bacterium are described, classified based on their somatic and flagellar antigens, with serovars 1/2a, 1/2b, and 4b being the most pathogenic [1,8,9,10,15,26,28]. In a study of the occurrence of *L. monocytogenes* in different soil samples, serovars 1/2b and 4b were identified in samples from pastures and vegetable gardens, and serovar 4b in urban areas [4]. Although *L. monocytogenes* is widely disseminated in nature, its incidence as an infectious agent in humans is sporadic [12,20,21]. As possible risk factors for the occurrence of *L. monocytogenes* in the cases in this study, references were made to housing without basic sanitation, farm work, and cattle breeding in patient 1 and poor socioeconomic status in patient 2, but none of the cases had a record of the infecting serovar in their medical records.

After oral acquisition, *L. monocytogenes* crosses the intestinal wall, multiplying in various types of cells, such as phagocytes and Peyer's patch cells [1,7,15]. Analyzing the behavior of *L. monocytogenes*, cell invasion appears to be mediated by the bacterial protein internalin, comparable to the integrins of *Legionella*, *Bordetella pertussis*, or the invasins of *Yersinia pseudotuberculosis* [11,28]. *L. monocytogenes* also uses listeriolysin O to escape through the vacuolar membrane, reaching the cytoplasm of the host cell, where conditions are favorable for growth and

replication. However, whether or not it passes through the membrane depends on the virulence of the serovar [7,10,16,22,26,28]. After crossing the mucosal barrier of the gastrointestinal tract and reaching the bloodstream, *L. monocytogenes* has tropism for the central nervous system, suggesting that infection of this site is the most common among the clinical manifestations of listeriosis [6,9,15,16,19]. The picture of meningitis is described as similar to that present in meningitis caused by other etiological agents, presenting fever, headache, vomiting, neck stiffness, and other neurological symptoms such as irritability and sensory changes [2,4,6,7,9,23,24]. Regarding the clinical picture, both cases initially presented with severe headache, vomiting, and fever spikes. There was a difference in the presence of signs of meningeal irritation (neck stiffness, positive Kernig and Lasegue signs), which appeared at the onset of symptoms only in case 1.

The CSF study is essential for diagnostic confirmation when *L. monocytogenes* is detected through culture. The literature also describes other common findings, such as cloudy appearance, pleocytosis with a predominance of polymorphonuclear cells, hyperproteinorrachia, and normal to low glycorrachia [2,3,5,6,11,14,17,24,25,26,27]. The complete blood count usually shows leukocytosis [3,5,11,13]. There was similarity between the CSF studies of cases 1 and 2, which presented leukocytosis, with a predominance of polymorphonuclear cells, hypoglycorrachia, hyperproteinorrachia, and positive culture for *L. monocytogenes*. The blood count showed leukocytosis in case 1 and leukopenia in case 2,

Ampicillin is the antibiotic most described in the literature as the treatment of choice [6,11,15,16,18,20,22,24,25,26,29], a finding corroborated by the demonstration of sensitivities to ampicillin, chloramphenicol, erythromycin, gentamicin, and tetracycline in the antibiogram [6,22,24,25]. Ceftriaxone is also described as an option for empirical treatment, associated with ampicillin, as well as ampicillin associated with gentamicin [5,29]. Both patients in this series received adjunctive corticosteroids; the benefit of adjunctive dexamethasone in *L. monocytogenes* meningitis remains controversial, with cohort studies reporting conflicting effects on outcome. [31] Ampicillin was frequently chosen for the treatment of meningitis caused by *L. monocytogenes* in several patient samples reported in the literature, either as monotherapy [3,11,17] or in combination with chloramphenicol [25], gentamicin [3], amikacin [18], or amikacin and sulbactam [2]. The treatment chosen for cases 1 and 2 was different. The patient in case 1 was treated with ceftriaxone for three days. In case 2, treatment for juvenile rheumatoid arthritis with oral methotrexate was maintained, and antibiotic therapy was changed during follow-up: initially treated with ampicillin, but the antibiotic regimen was changed to ceftriaxone on the second HD, combined with chloramphenicol on the sixth HD, and changed again to ampicillin on the tenth HD, maintaining the combination with chloramphenicol until discharge on the nineteenth HD.

The evolution of patients with meningitis described in the literature is variable, depending on the previous immune status, pathological potential of the serovar, and bacteremia [3,27]. Complete remission of the clinical picture was reported by some authors [2,3,5,6,11,18,23,25], but there was unfavorable evolution with death in five studies analyzed [2,6,16,17,25]. The cases described here evolved with fever spikes and fluctuation in the level of consciousness, diverging in the follow-up by hypoactivity, torpor, maintenance of hyperthermia, and death in case 1, and gradual improvement, with the emergence and remission of neurological signs and symptoms (strabismus, diplopia, signs of meningeal irritation) and discharge in case 2, demonstrating the broad spectrum of disease progression.

Conclusion

Meningitis caused by *L. monocytogenes* is an uncommon infection of the central nervous system, as clearly shown in the literature and consistent with the rare occurrence of cases in a referral hospital described in this study. Despite its incidence, the clinical picture is similar to that of other

types of meningitis, with fever, headache, vomiting, and neck stiffness. Laboratory tests include complete blood count, cerebrospinal fluid (CSF) study, and culture, presenting characteristic elements. According to the literature, the treatment of choice focuses on ampicillin and ceftriaxone, which can be combined with each other or with amikacin, gentamicin, or chloramphenicol. The prognosis varies depending on the host's immunity and the load and pathogenicity of the infecting serovar. Due to the numerous descriptions in the literature of cases associated with low organic resistance and the occurrence of meningitis caused by *L. monocytogenes*, this etiology should be considered, especially in the investigation of patients with compromised immune systems, whether due to an immunodeficiency disorder or because they are neonates, pregnant women, or elderly individuals, who present with fever, headache, vomiting, and neck stiffness.

References

1. de Noordhout CM, Devleeschauwer B, Angulo FJ, Verbeke G, Haagsma J, Kirk M, et al. (2014). The global burden of listeriosis: a systematic review and meta-analysis. *Lancet Infect Dis.* 14(11):1073-1082.
2. Li D, Li H. (2023). The clinical characteristics, diagnostic methods, treatment, and outcomes of *Listeria monocytogenes* meningitis: a case series study from China. *Infect Drug Resist.* 16:6375-6383.
3. Arslan F, Meynet E, Sunbul M, Sipahi OR, Kurtaran B, Kaya S, et al. (2015). The clinical features, diagnosis, treatment, and prognosis of neuroinvasive listeriosis: a multinational study. *Eur J Clin Microbiol Infect Dis.* 34(6):1213-1221.
4. Hofer E, Póvoa MM. (1984). Research on *Listeria monocytogenes* in soils. *Memórias do Instituto Oswaldo Cruz.* 79(1):45-53.
5. Adeva-Bartolomé MT, et al. (2005). Cerebral abscesses caused by *Listeria monocytogenes*. *Journal of Neurology.* 40(4):219-221.
6. Koopmans MM, Bijlsma MW, Brouwer MC, van de Beek D, van der Ende A. (2017). *Listeria monocytogenes* meningitis in the Netherlands, 1985-2014: a nationwide surveillance study. *J Infect.* 75(1):12-19.
7. Pizarro-Cerdá J, Kühbacher A, Cossart P. (2012). Entry of *Listeria monocytogenes* in mammalian epithelial cells: an updated view. *Cold Spring Harb Perspect Med.* 2(11):a010009.
8. Moura A, Criscuolo A, Pouseele H, Maury MM, Leclercq A, Tarr C, et al. (2016). Whole genome-based population biology and epidemiological surveillance of *Listeria monocytogenes*. *Nat Microbiol.* 2:16185.
9. Maury MM, Tsai YH, Charlier C, Touchon M, Chenal-Francisque V, Leclercq A, et al. (2016). Uncovering *Listeria monocytogenes* hypervirulence by harnessing its biodiversity. *Nat Genet.* 48(3):308-313.
10. Disson O, Lecuit M. (2012). Targeting of the central nervous system by *Listeria monocytogenes*. *Virulence.* 3(2):213-221.
11. Silva LJ, et al. (1992). Listeriosis and AIDS: Case Report and Literature Review. *Journal of the Brazilian Society of Tropical Medicine.* 34(5):475-478.
12. Bijlsma MW, Brouwer MC, Kananmoolalib ES, Kloek AT, Lucas MJ, Tanck MW, et al. (2016). Community-acquired bacterial meningitis in adults in the Netherlands, 2006-14: a prospective cohort study. *Lancet Infect Dis.* 16(3):339-347.
13. Xia X, Zhang L, Zheng H, Peng X, Jiang L, Hu Y. (2024). Clinical characteristics and prognosis of pediatric *Listeria monocytogenes* meningitis based on 10-year data from a large children's hospital in China. *Microbiol Spectr.* 12(3):e03244-23.
14. Elazary R, et al. (2008). Bacterial Meningitis and Sigmoid Diverticulitis Caused by *Listeria monocytogenes*. *The Israel Medical Association Journal.* 10(7):546-547.

15. Ramaswamy V, et al. (2007). Listeria – review of epidemiology and pathogenesis. *Journal of Microbiology, Immunology and Infectology*. 40:4-13.
16. Koopmans MM, Brouwer MC, Bijlsma MW, Bovenkerk S, Keijzers W, van der Ende A, et al. (2013). Listeria monocytogenes sequence type 6 and increased rate of unfavorable outcome in meningitis: epidemiologic cohort study. *Clin Infect Dis*. 57(2):247-253.
17. Amaya-Villar R, García-Cabrera E, Sulleiro-Igual E, Fernández-Viladrich P, Fontanals-Aymerich D, Catalán-Alonso P, et al. (2010). Three-year multicenter surveillance of community-acquired Listeria monocytogenes meningitis in adults. *BMC Infect Dis*. 10:324.
18. Mo T, Wu F, Dou X, Yang J, Shu Q. (2022). A retrospective study of rare Listeria meningitis in immunocompetent children in China. *Front Neurol*. 13:827145.
19. Siegman-Igra Y, et al. (2001). Listeria monocytogenes infection in Israel and review of cases worldwide. *Emerg Infect Dis*. 3(8):305-310.
20. Hofer E. (1974). Research on the occurrence of Listeria monocytogenes in human feces. *Journal of the Brazilian Society of Tropical Medicine*. 8:109-116.
21. Chenal-Francisque V, Lopez J, Cantinelli T, Caro V, Tran C, Leclercq A, et al. (2011). Worldwide distribution of major clones of Listeria monocytogenes. *Emerg Infect Dis*. 17(6):1110-1112.
22. Thønnings S, Knudsen JD, Schønheyder HC, Søgaard M, Arpi M, Gradel KO, et al. (2016). Antibiotic treatment and mortality in patients with Listeria monocytogenes meningitis or bacteraemia. *Clin Microbiol Infect*. 22(8):725-730.
23. Catão RMR, et al. (2003). Meningitis caused by Listeria monocytogenes in Campina Grande-Paraíba, Brazil: case report. *Brazilian Journal of Clinical Analysis*. 35(2):81-83.
24. Carvalho VO, et al. (1991). Clinical and laboratory aspects of 11 patients with listeriosis – Emílio Ribas Hospital – SP. In: *Proceedings of the 4th Brazilian Congress of Infectious Diseases*. Salvador. p.44.
25. Koopmans MM, Brouwer MC, Geldhoff M, Seron MV, Houben J, van der Ende A, et al. (2014). Cerebrospinal fluid inflammatory markers in patients with Listeria monocytogenes meningitis. *BBA Clin*. 1:44-51.
26. Koopmans MM, Brouwer MC, Vázquez-Boland JA, van de Beek D. (2023). Human listeriosis. *Clin Microbiol Rev*. 36(1): e00060-19.
27. Charlier C, Perrodeau É, Leclercq A, Cazenave B, Pilmis B, Henry B, et al. (2017). Clinical features and prognostic factors of listeriosis: the MONALISA national prospective cohort study. *Lancet Infect Dis*. 17(5):510-519.
28. Radoshevich L, Cossart P. (2018). Listeria monocytogenes: towards a complete picture of its physiology and pathogenesis. *Nat Rev Microbiol*. 16(1):32-46.
29. van de Beek D, Cabellos C, Dzupova O, Esposito S, Klein M, Kloek AT, et al. (2016). ESCMID guideline: diagnosis and treatment of acute bacterial meningitis. *Clin Microbiol Infect*. 22(Suppl 3): S37-S62.
30. Disson O, Charlier C, Pérot P, Leclercq A, Nir-Paz R, Kathariou S, et al. (2025). Listeriosis. *Nat Rev Dis Primers*. 11(1):71.
31. Brouwer MC, van de Beek D. (2023). Adjunctive dexamethasone treatment in adults with Listeria monocytogenes meningitis: a prospective nationwide cohort study. *EClinicalMedicine*. 58:101922.



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