A Case Report of *Listeria monocytogenes* Abscesses Presenting as Cortically Predominant Ring-Enhancing Lesions

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Abstract

**Introduction:** *Listeria monocytogenes*, a common cause of bacterial meningitis, rarely involves the central nervous system (CNS) in the form of multiple cerebral ring-enhancing lesions. **Methods:** An 81-year-old woman with rapidly progressive decline in her mental status in the setting of multiple cortically predominant ring-enhancing lesions was transferred to our institution. A mild upper respiratory tract infection and diarrhea symptoms preceded the mental status deterioration. Her past medical history is significant for type 2 diabetes mellitus. In light of the patient’s age, the presence of hyponatremia and the history of diabetes mellitus, the empiric antimicrobial treatment was modified to include ampicillin, meropenem, vancomycin, voriconazole and pyrimethamine/sulfadiazine to prevent opportunistic infections. Intravenous dexamethasone was added due to significant perilesional vasogenic edema. **Results:** The patient presented with stupor, but neither fever nor leukocytosis. CSF results were significant only for a mildly elevated protein level. The report of a repeat brain MRI was as follows: large areas of high FLAIR signals and tubular/lobulated/ring enhancement in bifrontal regions with a smaller focus in the left anterior midbrain, indicating for underlying multicentric glioma or multicentric primary CNS lymphoma. A brain biopsy, however, revealed an early abscess formation caused by a *L. monocytogenes* infection. **Conclusion:** A high index of suspicion in patients with risk factors for this infection is key to ensure the timely initiation of appropriate empirical antibiotic therapy in the setting of cerebral ring-
enhancing lesions. Intravenous ampicillin is the treatment of choice, but meropenem represents a valid alternative.

**Introduction**

Invasive foodborne infections with the bacterium *Listeria monocytogenes* are rare occurrences, with an estimated 1,600 cases per year in the United States [1]; yet, they carry significant morbidity and mortality rates, making early recognition and timely treatment of the infection crucial for improving patient outcomes. Extreme ages (neonates and people older than 50 years of age), pregnant women, diabetic and immunocompromised patients (especially those with cell-mediated response impairment), are at higher risk for invasive listeriosis, and central nervous system (CNS) involvement can be seen in up to 55% of patients, usually in the form of meningitis or meningoencephalitis, less commonly rhomboencephalitis. More rarely, listeria brain abscesses have been reported [4], and early recognition of the nature of these lesions remains a major challenge. Indeed, the neuroradiological appearance of these abscesses is not different from that of other types of brain abscesses and may also mimic primary or metastatic brain tumors. In addition, clinical manifestations of CNS infection (e.g. fever, headache, encephalopathy, meningeal signs, focal neurological deficits, seizures and hyponatremia) are aspecific, and a link between neurological presentations and the ingestion of food contaminated by listeria may be difficult to identify, as the onset of symptoms can be as late as 1 month after pathogen exposure [2–5].

Here we describe a case of CNS infection with *L. monocytogenes* presenting as multiple ring-enhancing lesions to emphasize the challenges in recognizing this type of clinical presentation and to show the importance of maintaining a high index of suspicion in patients with risk factors for this infection in order to allow a timely initiation of the appropriate treatment in the crucial early stages of the disease.

**Case Report**

An 81-year-old woman was transferred to our Neurocritical Care Unit at the University of Miami Hospital (from another hospital), for severe encephalopathy in the setting of a right frontal mass. Her past medical history was significant for coronary artery disease (status post stent placement 1 year prior to admission to our hospital), type 2 diabetes mellitus (DM), endometrial carcinoma (status post hysterectomy and chemotherapy more than 30 years prior to admission to our hospital), and aseptic meningitis with no sequelae (20 years prior to admission to our hospital).

At the outside hospital, where she was admitted 1 week earlier with a history of possible syncope versus seizure, dysarthria, runny nose, nausea, and diarrhea, her course was as follows: she was initially started on ciprofloxacin for her gastrointestinal manifestations; subsequently, she experienced a rapid deterioration of her neurological status, described as profound lethargy followed by the onset of bilateral weakness (right-sided greater than left-sided weakness) and cranial nerve abnormalities; viral encephalitis was suspected on clinical grounds alone and the patient was started on empiric intravenous acyclovir. A subsequent brain CT showed a right frontal brain mass; additional CT imaging of the chest, the abdomen and the pelvis was unremarkable. The patient was then transferred to our institution for further workup and management.
Upon presentation, the patient was afebrile with stable vital signs (in room air). A neurological examination revealed that she was stuporous, opening eyes only to painful stimuli (with attempts to localize them with her left upper extremity), not following commands, and nonverbal. She had a left gaze preference and her pupils were anisocoric (left 6 mm and right 4 mm), but both reactive to light. Her right upper extremity and lower extremity were flaccid with no response to painful stimuli, and there was minimal withdrawal in her left lower extremity.

Her laboratory data were unremarkable except for hyponatremia (sodium level: 131), hyperglycemia (glucose: 248) and mild anemia (Hgb: 11).

A brain MRI, with and without gadolinium (fig. 1), was performed, and the neuroradiologist’s official report was as follows: large areas of high FLAIR signal and tubular/lobulated/ring enhancement in bifrontal regions with a smaller focus in the left anterior midbrain; overall, these findings indicate underlying multicentric glioma or multicentric primary CNS lymphoma.

A lumbar puncture was also performed on admission, but CSF studies for bacterial, viral and fungal etiologies were unrevealing. The CSF chemical profile was within normal limits except for a mildly elevated protein level of 78.

Nevertheless, a high index of clinical suspicion for brain abscesses was maintained and, in consideration of the patient’s age and her history of DM, empiric antimicrobial treatment was modified to include ampicillin, meropenem, vancomycin as well as voriconazole and pyrimethamine/sulfadiazine in order to prevent opportunistic infections. Intravenous dexamethasone was added due to significant perilesional vasogenic edema.

An open brain biopsy was performed, and on day 3 Gram-positive rods were reported to be growing in the surgical tissue culture. This eventually revealed the diagnosis of early abscess formation caused by *L. monocytogenes* infection.

All antibiotics but ampicillin were discontinued. Steroids were slowly tapered off and the patient was transferred to the regular floor 10 days after admission with no signs of neurological improvement. A percutaneous endoscopic gastrostomy was placed due to the patient’s inability to swallow safely. Thirty-two days after admission, the patient was transferred to a rehabilitation facility with instructions to complete an 8-week course of antibiotic treatment.

At the time of discharge from our institution, the final neurological examination documented: eye opening to verbal stimuli, command-following with left upper extremity, incomprehensible sounds, pupils 4 mm bilaterally and reactive to light, partial left third cranial nerve palsy and left nasolabial fold flattening. Also noticed was the persistent significant weakness in her right upper extremity, right lower extremity and left lower extremity (minimal withdrawal to pain).

**Discussion**

Our patient’s advanced age and her history of type 2 DM represent 2 well-established risk factors for invasive listeriosis and were the only clinical clues to our early suspicion of listeria CNS infection. In our case, both the brain MRI, silent regarding the possibility of brain abscesses, and the lack of bacterial identification in the CSF studies pinpoint the challenges in the early diagnosis of atypical presentations of listeria in the CNS, which can lead to a delay in appropriate treatment, with detrimental effects on the patient’s outcome.
Studies have shown up to 15% of cases with negative CSF and blood cultures in the setting of active infection. Mortality from brain abscesses can be as high as 50%, but lowered to 40% when appropriate treatment is timely started [4].

Based on ample clinical experience and proven clinical efficacy, high-dose ampicillin (2 g intravenously every 4 h) has been established as the first-line treatment when a CNS infection by listeria is suspected or confirmed [6]. In cases of severe infection and complicated clinical scenarios, meropenem has been identified as an alternative effective treatment, with or without the combination with other antimicrobial agents [6, 7].

Several authors have suggested the co-administration of an aminoglycoside with ampicillin for enhanced bactericidal activity for at least the first week of treatment [8, 9]; however, insufficient and conflicting data exist to recommend this adjunctive therapy, with some studies suggesting that it might actually worsen the outcome [10].

The recommended duration of antimicrobial therapy ranges from 3 to 8 weeks depending on the type of CNS involvement (e.g. meningitis vs. abscess) and the patient’s immunological status, keeping in mind that a shorter course of antibiotics can be complicated by infection relapses [4]. While patients with meningitis should be treated for at least 3 weeks, those with brain abscesses should receive antibiotic therapy for a minimum of 6 weeks and followed with serial MRI or CT studies, with the proviso that antibiotics should be continued until brain imaging studies document either a resolution of the lesions or a small, stable lesion.

Our patient survived her *L. monocytogenes* CNS infection presenting with multiple brain abscesses. Additionally, the findings of her neurological examination at the time of discharge to rehabilitation revealed an improvement. We attribute this favorable clinical course to the timely initiation of appropriate empiric antibiotic therapy, including ampicillin to target *L. monocytogenes*, in consideration of the patient’s risk factors for invasive listeriosis.

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**Fig. 1.** Brain MRI. Axial (a) and coronal (b) T1 weighted views with gadolinium.