CASE REPORT

A RARE ENCOUNTER: LISTERIA MONOCYTOGENES AND BRAIN ABSCESS

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Background: Listeriosis, caused by Listeria Monocytogenes, is a serious yet rare illness caused by consuming contaminated food and is considered a global public threat. Central nervous system (CNS) involvement often follows bacteraemia; common clinical presentation is usually meningitis. Listeria brain abscess is an uncommon pathology and can manifest in various clinical presentations, with fever and focal neurological deficits being the most common. Brain abscess secondary to listeria is associated with high mortality. We present a rare case of a 68-year-old immunocompromised female who developed a cerebral abscess due to Listeria monocytogenes. The presence of fever, increased somnolence prompted further investigation. This case underscores the need for brain imaging in the evaluation of listeria sepsis, even when clinical presentations are atypical. Additionally, in cases without major neurological deficits or swelling, postponing abscess drainage may be considered. A cautious approach with IV antibiotics, repeat imaging and close monitoring can be offered to patients who are unwilling to undergo aspiration and drainage of brain abscess or are poor surgical candidates.

Keywords: Listeriosis; Listeria Monocytogenes; Contaminated food; Central nervous system

Citation: Khanum I, Ibtesam I, Haroon A, Zafar A. A rare encounter: Listeria monocytogenes and brain abscess. J Ayub Med Coll Abbottabad 2025;37(1):189–92.

DOI: 10.55519/JAMC-01-13907

INTRODUCTION

Listeria Monocytogenes (*L. Monocytogenes*) is a rare, food-borne, opportunistic bacterial infection with a mortality rate of about 25%.[1] It has a predilection for brain parenchyma, brainstem, and meninges, with bacteraemia and meningitis as the two most common presentations.[2] CNS infection in some form occurs in 30–55% of the cases of listeriosis. However, Listeria-causing brain abscess is seen only in 1% of the cases.[3] Impairment of cell-mediated immunity (either as a part of the disease process as seen with HIV or the use of immunosuppressive drugs such as transplant recipients or autoimmune disorders) is an important cause of listeriosis.[4] In the following case report, we highlight a rare presentation of a cerebral abscess with listeria and a successful patient outcome.

CASE REPORT

A 68-year-old female with a history of diabetes mellitus, hypertension, and non-B non-C chronic liver disease presented with a chief complaint of high-grade fever, drowsiness and generalised body weakness over the past three days. The patient and her family reported that she was in her usual state of health until three days ago when she experienced a sudden onset of high-grade fever (documented at 102 °F), accompanied by chills, rigours, and mild burning during urination. The patient described her drowsiness as excessive somnolence and did not report any other specific complaints. Notably, there was no history of focal limb weakness. She was prescribed azathioprine and prednisolone for the last two months for autoimmune hepatitis.

Upon examination, she appeared lethargic in her responses, yet demonstrated full orientation to person, place, and time, scoring a Glasgow Coma Scale of 15/15. There was no focal neurological deficit, no signs of meningeal irritation and other systemic examination was also unremarkable.

Initially, she was managed for sepsis secondary to urinary tract infection (UTI) considering past history of recurrent UTI, dysuria and was administered intravenous (IV) meropenem at a dosage of 1 gram every 8 hours. A thorough workup was conducted to identify the source of the fever, encompassing blood cultures, urine cultures, and a chest x-ray. Her blood cultures grew *L.monocytogenes*. Due to increased somnolence and drowsiness MRI was done. A multi-sequential MRI study with contrast showed abnormal signal intensity areas in the left frontal lobe and left basal ganglia, showing T2/FLAIR hyperintense signals with intralesional T2/FLAIR crescentic hypo-intensity, demonstrating heterogeneous enhancement and patchy diffusion restriction. The lesion measured 18 x 7.5 mm and 8 x 10 mm in transverse and antero-posterior dimensions and was causing a mass effect and partial effacement of the left frontal horn. It was also associated with left frontal lobe leptomeningeal enhancement and minimal ependymal enhancement along the left frontal lobe Figure-1.

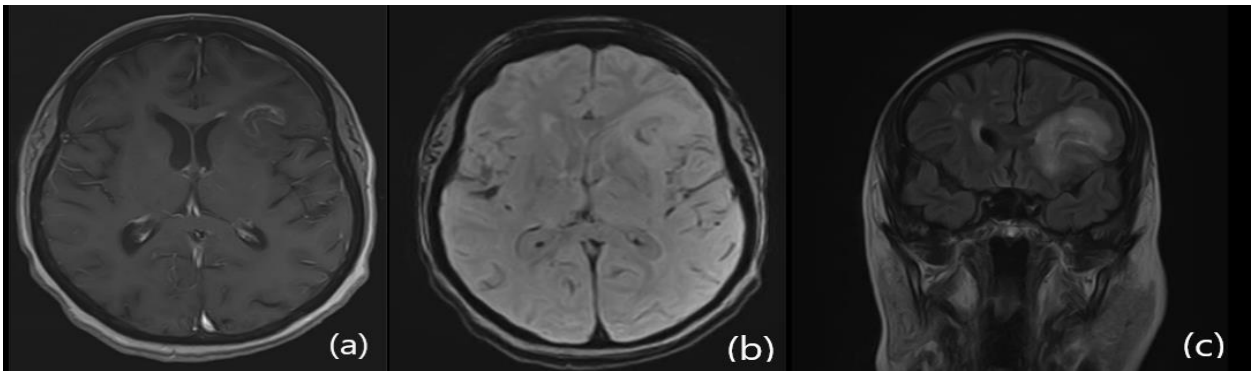

Figure-1 MRI brain with contrast. (*a*) T1 image showing a peripheral enhancing lesion with meningeal enhancement. (*b*) T2/FLAIR image showing abnormal signal intensity areas in the left frontal lobe and left basal ganglia showing hyperintense signals with intralesional T2/FLAIR crescentic hypo-intensity demonstrating heterogeneous enhancement and patchy diffusion restriction. (*c*) T1 coronal view showing the same lesion with perilesional oedema.

Based on the findings from the blood cultures and the observed lesion on her MRI, there was a suspicion of a cerebral abscess attributed to *L. monocytogenes*.

Consequently, the antibiotic regimen was intensified to intravenous Meropenem at a dose of 2 grams every 8 hours due to the non-availability of ampicillin in our pharmacy. A lumbar puncture was planned but deferred by the family and patient. The patient was referred to the Neurosurgery Department for a biopsy of the aforementioned lesion. However, the biopsy was postponed based on the preferences expressed by both the patient and their family.

Instead, a decision was made to continue with the trial of antibiotics.

MRI was repeated after one month, which revealed interval reduction in size of abnormal signal intensity areas in left frontal lobe and basal ganglia, showing interval reduction in abnormal signals and improvement in diffusion restriction. There was change in enhancement pattern, now seen as curvilinear intense enhancement and previously there was ill-defined patchy perivascular enhancement.

There is associated linear subcortical abnormal signal within it, returning low signals on T2, high signals on T1 and showing susceptibility, likely representing cortical laminar necrosis. Interval reduction in associated gyral swelling and vasogenic oedema with development of early gliosis with significant reduction in size of the lesion was also seen Figure-2. The patient improved clinically as well and had complete resolution of her neurological symptoms.

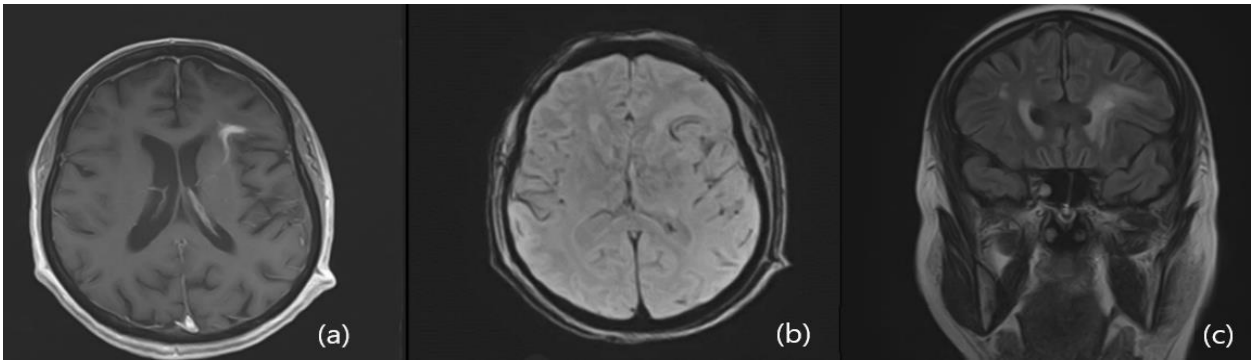

Figure-2 MRI brain with contrast (*a*) T1 image showing interval reduction in the ring enhancing lesion there is change in enhancement pattern, now seen as curvilinear intense enhancement (*b*) T2/FLAIR imaging showing linear subcortical abnormal signal intensity with intralesional hypo-intensity showing reduced size and improvement in diffusion restriction. (*c*) T1 coronal view showing reduced perilesional oedema and gyal swelling.

DISCUSSION

L. monocytogenes is a sporadic foodborne pathogen whose clinical manifestation ranges from subclinical febrile gastroenteritis to severe invasive disease. Invasive diseases can present in any of the following three forms (i) pregnancy-associated and neonatal listeriosis (ii) bacteraemia and (iii) central nervous system infection.[5] The incidence of neurolisteriosis reported in the literature is 30% but the occurrence of cerebral abscess secondary to *L. monocytogene* is only

3%.[6] The mechanism of invasion of *L.monocytogenes* into the brain has been proposed using mouse models. It can enter the CNS by at least three different mechanisms: (i) transport within infected monocytes that cross the blood-brain or blood-choroid barriers, (ii) direct invasion of endothelial cells that line the blood vessels in the brain, or (iii) retrograde migration along the axons of cranial nerves.

The bacterium can invade and replicate within various types of cells, such as epithelial cells, endothelial cells, and neurons, by using specific surface proteins called internalins. It can also escape from the phagosomes of immune cells and move from cell to cell by forming F-actin tails. *L. monocytogenes* can also infect the bone marrow and parasitize monocyte precursors, which then enter the bloodstream and carry the bacteria to the brain.[7]

The risk factors identified for listeriosis are pregnancy, age >60, immunocompromised patients such as those with HIV/AIDS, organ transplant, diabetes, or on steroid or immunosuppression therapy.[8] Our patient was also immunocompromised as she was taking azathioprine and prednisolone for autoimmune hepatitis. Trachuk *et al* reported a similar case of a patient who presented with focal neurological deficit and was found to have CT and MRI finding consistent with an abscess. Both blood culture and aspirate taken from the brain abscess grew *L. monocytogenes*.[9] Cirrhosis is a possible risk factor to develop Listeria brain infection, but it is relatively less common as compared to other co morbidities.[10]

Irrespective of the risk factor, it is well known that listeria brain abscess can present in a variety of manner with the most common presentation being fever and focal neurological deficit. The duration of symptoms varies between 1-2 weeks. The prevalence of solitary abscess is more common than multiple abscesses with predilection for frontal lobe.[2] Zang *et al.* documented a case resembling ours, featuring a listeria brain abscess.

In this instance, imaging revealed an irregular, well-defined lesion in the left frontoparietal area, alongside a positive blood culture for listeria monocytogenes. Unlike our patient, this individual exhibited a focal neurological deficit. Similarly to our case, no aspiration was performed, and treatment with ampicillin was administered for six weeks. Significant improvement was noted during the three-month follow-up visit.[1] The MRI findings in case of listeria brain abscess are variable but the curvilinear worm like pattern has been reported typical to listeria brain abscess and presence of such findings on MRI warrants testing for Listeria.[11]

Potential complications of listeria brain abscess include permanent neurological deficit and unfavourable responses to antibiotic therapy. In one reported case, there was permanent hemiparesis that did not resolve after antibiotic treatment and repeat imaging revealed an increase in the size of abscess precluding the need for surgical drainage.[12] In a multicenter prospective study of patients with listeriosis, 252 patients with neuro-listeriosis were included. Among them, 181 survived with 44% experiencing lasting neurological impairment such as encephalitis, limb weakness, cerebellar symptoms and eighth nerve palsies.[13]

Listeria spp are usually sensitive to penicillin, aminoglycosides, Carbapenem, first and second-generation cephalosporins & aminoglycoside. Generally, combination of ampicillin with gentamicin is found to be most effective as the addition of gentamicin confers bactericidal activity to the regimen. These antibiotics do not readily enter the blood-brain barrier and hence need to be given for a longer duration of time.[14] The general recommendation for treatment of *L. monocytogenes* brain abscess includes ampicillin 2 gm IV every four to six hours with gentamicin 1.7mg/kg IV every 8 hourly for 4-6 weeks or longer.[15] Second-line medications include trimethoprim-sulphathiazole but there are no definitive guidelines for listeria brain abscess treatment available.

Furthermore, unlike acute bacterial meningitis, the use of dexamethasone in listeria brain abscesses is associated with poor outcomes.[13]

Managing this case presented challenges due to the absence of treatment guidelines for Listeria monocytogenes brain abscess, an atypical clinical presentation, and reliance solely on positive blood culture results since pus culture from the brain abscess was unavailable. Based on the clinical presentation exhibited by our patient, we advocate for the performance of brain imaging in every individual with listeria bacteraemia, even in the absence of focal neurological deficits, particularly in those presenting with risk factors for disseminated infection.

Furthermore, in cases lacking significant neurological deficits or oedema, the aspiration of abscesses may be deferred. Instead, a trial of intravenous antibiotics should be initiated, followed by close monitoring of patient's clinical as well as radiological status.

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Submitted: October 5, 2024	*Revised: February 5, 2025*	*Accepted: February 5, 2025*

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