

Case Report: Meningitis a *Listeria monocytogenes* in a young, immunocompetent male

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Abstract:

Listeria Monocytogenes (LM) is a gram-positive intracellular pathogen usually transmitted to humans by ingesting contaminated food. It typically infects immunocompromised individuals, such as pregnant women, newborns, alcoholics, and the elderly.

LM is a rare cause of meningitis in immunocompetent, healthy adults.

However, its clinical presentation is similar to that of other viral or bacterial central nervous system infections and its course can be rapid and aggressive. Physicians should therefore always consider LM as a possible etiologic agent of meningitis, especially in cases that do not respond to first-line empiric antibiotic therapy.

We report a case of *Listeria Monocytogenes* meningitis in a previously healthy 16-year-old.

Key words: Immunocompetent adult, *Listeria Monocytogenes*, Meningitis, Antibiotics, Clinical presentation.

Introduction

Listeria Monocytogenes is a gram-positive, facultative intracellular bacteria transmitted to humans through ingestion of contaminated food.[1]

It generally affects pregnant women, newborns, the elderly and immunocompromised patients.[2]

Invasive forms of the disease mainly manifest as febrile gastroenteritis, endocarditis, maternal-neonatal infections and septicemia with or without neuromeningeal involvement.[3]

Meningitis is the most common manifestation when the central nervous system is involved.[3]

Rare cases of *Listeria* meningitis have been reported in previously healthy, immunocompetent individuals. These cases can be associated with serious complications and a high mortality rate, hence the importance of rapid diagnosis in order to initiate appropriate antibiotic therapy.[4], [5]

Case presentation:

The patient was a 16 years old male without a past medical history, admitted to the emergency department with severe headache, diarrhea and acute change in mental status 2 days before his admission.

On his admission, he was febrile a 40.1oC, heart rate was 126 bpm, blood pressure was 126/72 mmHg, polypneic a 24 breaths/min, and oxygen saturation was 94% on room air and capillary glucose 1.60 g/l.

On physical examination, the patient was confused, unresponsive to verbal stimuli but responsive to sternal rub and painful stimuli. A score of 12 was recorded according to the Glasgow coma scale (GCS). Kernig's and Brudzinski's signs were negative. Abdomen tender without palpable mass or externalized hemorrhage or cessation of matter and gas.

Cerebral CT scan (Figure 1): No acute abnormalities.

Abdomino-pelvic CT (Figure 2): Moderate peritoneal effusion associated with ileo-caecal thickening.

Blood tests on admission showed an elevated white cell count (WCC) 390/mm³, haemoglobin 12.4 g/dl, platelet count 128,000 el/mm, CRP 129 mg/l, Procalcitonin 0.92 g/l, the rest of the results were normal.



Figure 1 : Cerebral CT scan

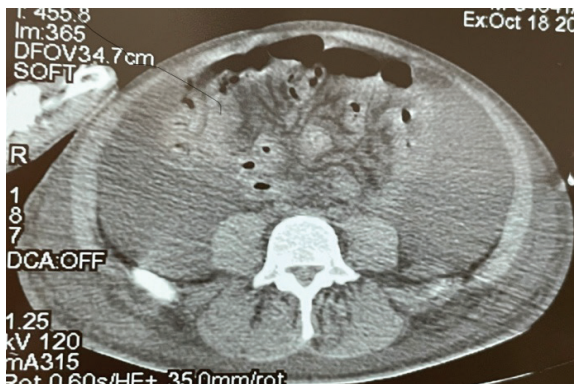


Figure 2: Abdomino-pelvic CT

Lumbar puncture (LP) was performed and the cerebrospinal fluid (CSF) was sent to the Laboratory for microbiological and biochemical investigations, it showed a clear CSF, normal protein and glucose levels, normal cytology with sterile culture. Cytobacteriological examination of urine was sterile. The patient underwent diagnostic laparoscopy, which revealed a small peritoneal effusion with no other abnormalities. Because of the lack of any significant improvement in his clinical status and due the clinical examination findings, an antibiotic therapy with a 3rd-generation cephalosporin in meningeal doses (3g× 2 per jour) and an antiviral agent (acyclovir 1000 mg) intravenous piggyback (IVPB) were started. In view of the neurological deterioration, the patient was intubated and put under mechanical ventilation. Then, brain MRI was indicated, it showed a moderate active triventricular hydrocephalus with central cerebral involvement and ptosis of the cerebellar tonsils through the foramen magnum.

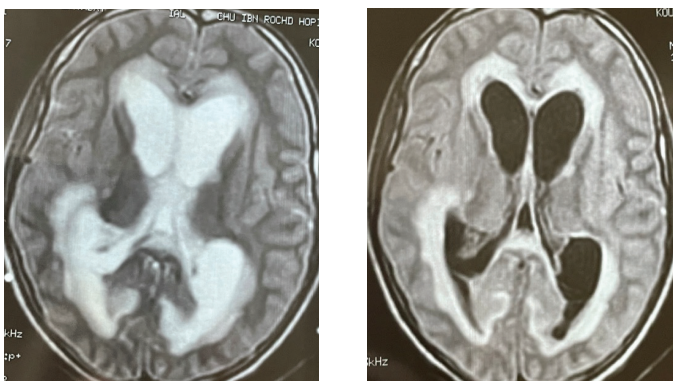


Figure 3:brain MRI

Blood tests were performed regularly during his hospitalization - Laboratory testing: WCC $18.280 \times 10^3/\text{mm}^3$ with 77.3% neutrophils, hemoglobin 11.4 g/dl, and platelet count $248,000 \text{ el}/\text{mm}^3$, CRP 172 mg/l, Procalcitonin 2.16 g/l. - A new LP was performed: a turbid CSF appearance, elevated CSF Leucocytes, CSF proteins a 1.95g/l, CSF glucose less than 0.05g/l and culture isolated *Listeria Monocytogenes*. - Blood culture from two sets of aerobic and anaerobic bottles grew *Listeria monocytogenes*.

A diagnosis of *Listeria monocytogenes* meningitis was therefore made, the antibiotics were then adjusted to ampicillin 2g/6h and aminoglycoside 1.5 g/day by the end of day two, and the patient was sent to the neurosurgical unit for an external ventricular drainage (EVD).

By the end of day 3 of his admission, and after receiving the adjusted therapy with EVD in place, the patient showed neurological improvement without any respiratory complications and the decision of extubation was made.

By the end of day 4, blood tests showed: WCC $13.220 \times 10^3/\text{mm}^3$ with CRP 88 mg/L and Procalcitonin 0.99 g/L. Neurological signs and symptoms improved.

On day 6, the patient was transferred to Neurosurgery department and both Neurology and Infectious Disease teams were informed.

Discussion:

Listeria Monocytogenes is a well-recognized opportunistic pathogen that primarily affects certain well-defined high-risk groups, including newborns, pregnant women, the elderly and immunocompromised individuals.[2]

Infection during pregnancy can lead to abortion, premature birth and amnionitis, while in newborns it can cause meningitis, conjunctivitis and pneumonia.[6] In immunocompromised patients, *Listeria* causes endocarditis and septicemia, with or without neuromeningeal involvement such as meningitis, meningoencephalitis, rhombencephalitis or brain abscess.[4], [7]–[9]

Rare cases of *Listeria* meningitis have been reported in previously healthy, immunocompetent individuals. These cases can be associated with serious complications and a high mortality rate. Rapid diagnosis is essential to initiate appropriate antibiotic treatment and achieve the best possible outcome.[10], [11]

CNS invasion by *Listeria Monocytogenes* can occur via at least three different routes: Transport across the blood-brain or blood-choroid barriers within parasitized leukocytes, direct invasion of endothelial cells by blood-borne extracellular bacteria, or retrograde (centripetal) invasion of endothelial cells into the brain in cranial nerve axons.[1], [5]

The clinical symptoms of *Listeria* meningitis are similar to those of other meningitides: fever, stiff neck, altered mental status, focal neurological signs and seizures.[10], [12], [13]

Biology shows leukocytosis with neutrophilia and elevated CRP. [10], [12]

Lumbar puncture is essential for diagnosis. In most cases, CSF analysis reveals pleocytosis with neutrophilia, reduced glucose concentration and increased protein levels.

Gram staining and CSF culture have been reported to be negative in a few cases at first lumbar puncture. Repeat CSF analysis is therefore recommended in cases of severe meningitis that do not respond to first-line antibiotic treatment, and when no specific organism has been identified on initial evaluation.[10], [13], [14]

Blood cultures are positive in 60% of cases.[12], [13]

Serological tests for *Listeria* meningitis lack specificity and

should only be used for retrospective diagnosis.[10], [12], [13] CT scans may show hypodense lesions, but MRI imaging is the radiological technique of preference.[12], [13] Empirical first-line treatment of meningitis often includes third-generation Cephalosporins and Vancomycin to target the most common pathogens. However, once *Listeria Monocytogenes* has been isolated from the CSF, treatment must be adjusted to include Ampicillin alone or in combination with an Aminoglycoside, such as Gentamicin or Amikacin. A single in-vitro study has demonstrated that this combination is synergistic.[9], [15], [16] Although Vancomycin may be effective against *Listeria* in in-vitro tests, it has a high clinical failure rate due to its low penetration of CSF. [15], [16] Clinical data are insufficient for the most recent Fluoroquinolones. Trimethoprim-Sulfamethoxazole has recently been described as an effective alternative for CNS listeriosis refractory to conventional treatment. Carbapenems, used alone or in combination with an aminoglycoside, have also given good results.[15], [16] The duration of treatment varied from 10 days to a maximum of 8 weeks, depending on the severity of the case.[9], [16] As *Listeria* mainly affects immunocompromised hosts, an immunological evaluation may be useful. Underlying causes of immunosuppression should be excluded, such as prolonged or inappropriate corticosteroid therapy, iron overload, etc.[9] Because of the high mortality rate, it is important that *Listeria Monocytogenes* be considered as one of the possible causes of community-acquired bacterial meningitis in any patient, even an immunocompetent adult, who does not respond to empirical antibiotic therapy.[9], [15]

Conclusion

Due to the rarity of *Listeria monocytogenes* meningitis and the absence of definitive guidelines for the best course of treatment, the type and duration of therapy, we think it is imperative to conduct further research on this complication. Immunocompetent patients should also be suspected of having *Listeria monocytogenes* infection, and novel molecular biology tools are crucial in the early detection of this uncommon condition. Since our patient was young and immunocompetent, ampicillin was not strictly necessary to provide *Listeria* coverage. However, we advise that ampicillin should be added to the regimen in case of meningitis, especially when the evolution is unfavorable under first line treatment and settings where a lumbar tap is not feasible.

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