CASE REPORTS

Bilateral Facial Nerve Palsy Following Antibiotic Treatment for *Listeria Monocytogenes* Meningitis

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Keywords: Facial nerve palsy, listeria monocytogenes, meningitis, community-acquired meningitis

https://doi.org/10.52504/001c.89403

Georgetown Medical Review

Vol. 7, Issue 1, 2023

Listeria monocytogenes (LM) is an intracellular, facultative, gram-positive bacillus transmitted mainly through contaminated food, particularly dairy products and cold deli meats. It typically affects immunocompromised patients, and a rarely neuroinvasive disease in immunocompetent healthy adults has been described in the literature. Despite the timely intervention, neurologic morbidity has been observed after extended periods of recommended antibiotic regimens. This case study highlights the challenges in diagnosing and managing LM meningitis, especially when atypical symptoms and complications arise. Herein, we describe the case of a 57-year-old healthy man admitted with symptoms of nausea, vomiting, headache, neck pain, and stiffness, who was eventually diagnosed with LM-related meningitis and treated with antibiotics. The patient's hospital course was complicated by bilateral facial nerve palsy, while other symptoms improved with antibiotic treatment, raising suspicion for parainfectious immunological response, which improved with a short course of steroids. This case contributes to the medical literature by providing insights into the immunological response for treating LM meningitis and managing LM meningitis–associated complications. To our knowledge, this could potentially be the first case of LM meningitis causing the parainfectious immunological response of bilateral facial nerve palsy after antibiotic treatment.

Introduction

Listeria monocytogenes (LM) is a facultative, gram-positive intracellular bacillus; human disease is acquired from the ingestion of unpasteurized dairy products and cold deli meats. It can grow at cold temperatures (4°C). Though the annual incidence of laboratory-confirmed listeriosis in the United States is rare (0.24 cases per 100 000 population), in recent years several large outbreaks have been reported.[1,2]

According to the World Health Organization, listeriosis is a very uncommon disease, with 0.1 to 10 occurrences per 1 million people per year and a death rate ranging between 20% and 30%,[3] particularly for those who acquire an invasive condition.[3] It typically affects immunocompromised patients or patients who are taking immunosuppressive agents, those who have diabetes, pregnant individuals, people older than 65 years, patients with substance abuse disorder, and neonates.[4] Of the clinical syndromes listeriosis causes, meningitis, sepsis, and meningoencephalitis are among the most difficult to treat.[5,6] However, there have been case reports of infections caused by *Listeria* in formerly healthy and immunocompetent individuals, which may be linked to serious consequences and a 26.6% 30-day death rate.[7]

Aside from bacterial meningitis, there are other infections that can potentially cause cranial nerve palsies, although the likelihood and specific manifestations may vary. For example, viral infections such as *herpes zoster* (shingles) can result in facial nerve palsy known as Ramsay Hunt syndrome. Lyme disease, caused by the bacteria *Borrelia burgdorferi*, is another infection that can lead to facial nerve palsy, among other neurologic symptoms.[8] The development of cranial nerve palsies in these infections can be attributed to various mechanisms, including direct nerve involvement, inflammation, compression, or immune-mediated processes. It is important to note that while cranial nerve palsies can occur in the context of certain infections, the occurrence of bilateral facial nerve palsy, the simultaneous involvement of both facial nerves leading to facial muscle weakness or paralysis, is a rare complication that has been infrequently reported in LM meningitis cases. This complication can occur due to the proximity of the facial nerves to the meninges and the inflammatory response in the surrounding area.[9]

Case Presentation

A 57-year-old man presented to the emergency department with a 3-day history of headache, fever, neck pain, dizziness, and lethargy. Recent travel included Germany a week prior. On initial assessment, he denied any shortness of breath, blurring of vision, nausea, or vomiting. However, he did mention consuming deli meat days prior to his illness. He had been positive for COVID-19 a few months prior but had tested negative prior to his arrival to the US. He was otherwise healthy, followed an active lifestyle, and rode a bike to work daily. He took over-the-counter fever medication for headache and fever, which did not improve his symptoms and led to his presentation.

On presentation, the patient was febrile (39.5°C) but otherwise hemodynamically stable. Physical examination was unremarkable except for neck stiffness. He had no additional neurologic deficits, papilledema, or rash. Laboratory diagnostics demonstrated leukocytosis (15.9 k/μL; range, 4.0-10.8 k/μL) with neutrophil predominance (85%; range, 43%-75%), an elevated lactate level (3.8 mmol/L; range, 0.5-2.2 mmol/L), and an elevated C-reactive protein level (28.15 mg/L; range, 0-10.0 mg/L). Diagnostic imaging included computed tomography of the head without contrast. A chest radiograph on admission demonstrated a left lower lobe consolidation for which the patient was admitted and received antibiotic therapy for presumed community-acquired pneumonia.

Despite appropriate management for presumed community-acquired pneumonia, the patient remained febrile. His headache had worsened and was associated with increasing neck discomfort over the course of the next day. Repeat neurologic examination showed progressive cranial nerve VI palsy with associated diplopia. Therefore, a lumbar puncture was conducted. Cerebrospinal fluid (CSF) was consistent with bacterial meningitis, showing low glucose levels (25 mg/dL), elevated white blood cell count (600/mm^3;

Table. Cerebral Spinal Fluid (CSF) Analysis

CSF Characteristic	Tube 1	Reference values	Tube 4
Color	Pink (ABN)		Pink (ABN)
Post spin	Xanthochromia absent		Xanthochromia absent
RBC	5200/mm^3	0-1/mm^3	5300/mm^3
WBC	600/mm^3	0-5/mm^3	560/mm^3
Segmented neutrophils	32/mm^3	0-7/mm^3	27/mm^3
Lymphocytes	49/mm^3	28-96/mm^3	50/mm^3
Monocytes	19/mm^3	16-56/mm^3	23/mm^3
Glucose	25 mg/dL	40-70 mg/dL	
Protein	145 mg/dL	15-45 mg/dL	
Cryptococcal antigen	Negative		

Abbreviations: RBC, red blood cell; WBC, white blood cell; ABN, Abnormal.

high segmented neutrophils, 32/mm^3), and high protein levels (145 mg/dL) (Table). The antibiotic regimen was transitioned to vancomycin, ceftriaxone, and ampicillin. He was prescribed adjunct steroid therapy, as the consulting neurology team recommended.

Despite broad-spectrum meningitis dosing, the patient developed progressive lethargy and was intubated for airway protection requiring transfer to the intensive care unit. Within the next few days, CSF cultures speciated LM, and his antibiotics were narrowed to gentamicin and ampicillin with discontinuation of adjunctive steroid therapy. After a period of initial improvement, he developed facial weakness, and an inability to close his eyes was noted. A detailed neurologic examination indicated signs of bilateral facial nerve palsy. Facial sensations were intact. A 3-day trial of methylprednisolone, 1 g/d, was initiated to which the patient eventually benefited with complete and continuous symptom regression.

Discussion

With 5% of cases worldwide, LM is the third most common reason for community-acquired meningitis globally.[10] The most significant predictors of death in individuals with Listeria meningitis include increasing age, chronic illness, sepsis, and cancers.[11] In 9% to 12% of adult patients with acute bacterial meningitis, cranial nerve palsies can occur either at the beginning or during the infection.[12] Particularly prevalent cranial nerve diseases include those affecting the oculomotor, abducens, facial, and glossopharyngeal nerves.[13] However, LM meningitis infrequently reports cranial nerve palsy.

Although the pathogenic cause of cranial nerve palsies is seldom known, it is thought that either elevated intracranial pressure or a meningeal inflammatory response close to the cranial nerves may be responsible. On the contrary, in our patient, antibiotics resolved the episode of meningitis, which was evident by improvement in his headache, neck stiffness, and diplopia. However, bilateral facial nerve palsy developed after a few days of starting antibiotics.

The Infectious Diseases Society of America recommends administering dexamethasone (0.15 mg/kg) for a period of 2 to 4 days in people with suspected acute bacterial meningitis, with the initial dose administered together with the first dose of antibiotic treatment. However, other than Streptococcus, there are no data to support the use of steroids in meningitis.[14] Hence, there were some uncertainties regarding the initiation of steroids after the establishment of Listeria meningitis and cranial nerve VII palsy according to the MONALISA national prospective cohort study. That study showed decreased survival in patients with neurolisteriosis when treated with concomitant dexamethasone; nonetheless, the number of patients who were treated was small.[15] But given the rarity of cases, the agreement was made to start methylprednisolone, 1 mg/d, for 3 days to which the patient effectively responded. A steroid medication, which has an immunosuppressive effect, helps patients with bilateral facial nerve palsy to recover. There have been previously reported cases of bacterial meningitis with similar sequelae after treatment. The decision to initiate a short course of intravenous methylprednisolone was based on the likelihood of a paradoxical immunological response causing sequelae of neurologic impairment well after the initiation of antibiotic treatment.[16]

Because of LM's predilection for the central nervous system, of all the cases of invasive listeriosis, half of these account for neurolisteriosis, which increases the mortality to more than 50%. Listeria rhombencephalitis is a rare and severe form of neurolisteriosis along with meningoencephalitis and cerebritis evolving into abscesses.[17-19] Up to 14% of adults diagnosed with Listeria meningitis develop hydrocephalus, and Listeria is the second most prevalent infection to cause this condition. Almost all patients have communicative hydrocephalus, resulting from a severe infection that prevents CSF resorption through arachnoid granulations.[20] To our knowledge, this might be the first case of LM meningitis causing the parainfectious immunological response to antibiotic treatment leading to bilateral facial nerve palsy. Furthermore we report it was resolved with pulse dose of methylprednisolone for 3 days.

L.M meningitis in review of related publications has failed to identify any reports of complications following antimicrobial administration for listeria meningitis. Bilateral facial nerve palsy can occur due to parainfectious immune response to antimicrobial agent as described in our case report. More work needs to be done at molecular level to find virulence determinates for specific parainfectious immune response to antibiotics.

Conclusion

LM meningitis is an uncommon cause of cranial nerve palsies, but this case emphasizes the value of early detection and effective treatment. The purpose of this case report is to raise awareness of the many clinical symptoms and potential adverse effects of LM meningitis. When dealing with uncommon neurologic symptoms, it emphasizes the importance of early detection, proper

antibiotic therapy, and evaluation of additional therapies because delay in treatment is independently associated with unfavorable outcomes. It is necessary to conduct further studies to improve our knowledge of LM meningitis and its treatment, ultimately leading to better patient outcomes.

Acknowledgment

The authors declare that they have not received substantial contributions from non-authors or by any sponsor. The manuscript has not been published previously and all the authors contributed substantially to writing it at any step. We would like to thank our patient who gave his consent for his clinical information to be reported in the journal.

Conflicts of Interest

None reported.

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