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CASE REPORT

Infection with *Listeria monocytogenes* meningoencephalitis: A case report

Da-Zhen Xu, Quan-Hui Tan

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Abstract

BACKGROUND

Listeria meningitis is an infectious disease of the central nervous system caused by *Listeria monocytogenes*. This bacterium is widely present in the natural environment and can be transmitted through channels such as food and water. Patients usually show symptoms such as fever, headache, and neck stiffness. In severe cases, coma, convulsions, or even death may occur. Traditional diagnostic methods, such as cerebrospinal fluid (CSF) culture and serological tests, have certain limitations. Although CSF culture is the "gold standard" for diagnosis, it is time-consuming and has a relatively low positivity rate. Serological detection may also result in false positive or false negative results. The emergence of metagenomic sequencing (mNGS) technology has led to a significant break-through in diagnosing Listeria meningitis, allowing quick and accurate detection of various pathogens in samples.

CASE SUMMARY

Here, we present the case of a previously healthy 64-year-old woman diagnosed with Listeria meningitis using mNGS. She was successfully treated with intravenous ampicillin and meropenem, without any complications.

CONCLUSION

Listeria meningitis must be considered, especially in patients who fail to show improvement with first-line antibiotic treatments. mNGS significantly reduces the diagnosis time, supporting timely treatment of patients.

Key Words: *Listeria monocytogenes*; Meningitis; Diagnosis; Treatment; Immune function; Case report

Core Tip: Listeria meningitis is a potentially serious condition with a high associated mortality rate, making its early diagnosis crucial. In immunocompromised patients, active administration of first-line antibiotics can help achieve better clinical outcomes.

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INTRODUCTION

Listeria monocytogenes (LM) is a facultative anaerobic bacterium that causes Listeria meningitis[1]. LM is an intracellular, parasitic, Gram-positive, non-spore-forming bacillus and a zoonotic pathogen[2]. This bacterium is widely present in natural environments such as soil and water, and has a strong tolerance to low temperatures and high salinity[3]. It is a foodborne bacterium that easily contaminates various foods such as dairy products, seafood, meat and eggs, poultry, fruits, and vegetables. It is widely present in refrigerated foods and is classified as an opportunistic pathogen[4]. Patients often have a history of catching a cold and directly consuming refrigerated food before the onset of the disease. Susceptible populations include newborns, pregnant women, the elderly, and individuals with low immunity[5]. After consuming food contaminated with LM, the bacteria enter intestinal epithelial cells by binding to the adhesive proteins on the surface of the cells, multiply within the cells, and use actin to spread between cells, thereby passing through the intestinal barrier and the blood-brain barrier and spreading to any part of the body[6].

LM meningitis usually has an acute onset. The first symptom in 90% of cases is fever, often exceeding 39 °C and accompanied by severe headaches, dizziness, nausea, and vomiting. There is obvious meningeal irritation, and patients often exhibit altered consciousness, including numbness, delirium, and seizures. In severe cases, loss of consciousness may occur within 24-48 h[7]. A few cases have a slow onset, a long disease course, and repeated episodes. If the lesion involves the brain parenchyma, symptoms of encephalitis or brain abscess may develop[8]. Some patients may experience double vision, difficulty in pronunciation or swallowing, facial nerve paralysis, and hemiplegia. Cerebrospinal fluid (CSF) examination typically reveals a cloudy appearance, increased WBC count and multinucleated cell count, elevated protein levels, and reduced sugar and chloride concentrations[9]. LM can also be found in blood and CSF cultures. LM is sensitive to penicillin, ampicillin, gentamicin, streptomycin, chloramphenicol, quinolones, rifampin, and sulfamethoxazole/trimethoprim, among others. Penicillin or ampicillin are the primary therapeutic drugs, although they do not show bactericidal effects *in vitro*. In severe cases, these two antibiotics are often used in combination. The combination of ampicillin or penicillin with aminoglycoside antibiotics has a synergistic effect. The clinical manifestations and CSF findings in LM meningitis are not significantly different from those of other forms of suppurative meningitis, making its diagnosis and treatment challenging for clinicians. This requires careful clinical attention. Our department has successfully treated a critically ill patient with LM meningitis. The diagnosis and treatment process are reported below.

CASE PRESENTATION

Chief complaints

Intermittent fever and headache for 1 month and convulsions and confusion for 2 days.

History of present illness

On May 8, 2021, the patient developed a fever for no apparent reason, with a maximum body temperature of 39.3 °C, accompanied by headache and forceful vomiting, without any loss of consciousness or convulsions.

History of past illness

The patient had a history of systemic lupus erythematosus (SLE) for more than 10 years. During the 6 months when she was abroad, she had stopped taking steroids on her own. Upon returning to China 2 months prior to admission, she underwent a comprehensive examination in the nephrology department, followed by a re-examination that showed decreased complement C3 and C4 levels. Three doses of methylprednisolone had been administered orally to control her SLE.

Personal and family history

All members of her family were in good health and had no history of tuberculosis or other related diseases.

Physical examination

The patient's body temperature was 38.9 °C, heart rate was 137 beats/min, respiratory rate was 26 times/min, and blood pressure was 155/81 mmHg. She was in a deep coma, with pupils that were equal, round, reactive to light, and measured 3 mm bilaterally. Neck stiffness was noted. Slightly thick breathing sounds were observed in both lungs, but no dry or wet rales were heard. The heart rate was 137 beats/min, regular, with no pathological murmur. The abdomen was flat and soft, and the liver, spleen, and ribs were unaffected. The left upper limb was ankylosed, and there was no edema in both lower limbs. No pathological reflex was elicited.

Laboratory examinations

Blood tests revealed a rapid c-reactive protein level of 31.65 mg/L, a WBC count of 5.5 × 10^9/L, and a neutrophil percentage of 90.9%. The CSF pressure was 250 mmH_2O, total WBC count was increased, protein levels were significantly increased, and glucose and chlorine levels were decreased.

On the 4th day of admission, mNGS of the CSF indicated the presence of LM (Table 1), and both blood and CSF cultures were positive for LM. Serum complement C3 and C4 levels were normal. Other diagnostic tests, such as the T-cell spot of tuberculosis test (T-SPOT), CSF latex agglutination test, CSF tuberculosis (TB)-PCR, and autoimmune brain antibody tests were all negative.

Imaging examinations

Electroencephalography: Display exception.

Head magnetic resonance imaging: Multiple small ischemic lesions in the bilateral frontal, parietal, temporal, and occipital lobes, as well as the lateral ventricles.

Chest computed tomography revealed local callus formation after fractures of the right fourth to ninth ribs. In addition, a review of electroencephalography showed abnormalities, with irregularities on the video electroencephalography topographic map. Head CT and MRI showed scattered small ischemic foci in the bilateral frontal and parietal lobes, basal ganglia, and lateral ventricles.

MULTIDISCIPLINARY EXPERT CONSULTATION

After admission, the patient received electrocardiographic monitoring, oxygen therapy, and critical illness reporting. A lumbar puncture was performed, revealing CSF pressure of 250 mmH_2O, an increased total WBC count, significantly increased protein levels, and decreased glucose and chlorine levels (Table 2). Central nervous system (CNS) infections (excluding bacteria, tuberculosis, fungi, and viruses) and lupus encephalopathy were considered possible diagnoses. Samples were sent for three smears (bacterial, fungal, and acid-fast staining), three cultures, latex agglutination tests, TB-PCR, CSF mNGS, autoimmune brain antibodies, and blood T-SPOT tests, with consultation from the Department of Rheumatology and Immunology. The patient was treated with anti-infection therapy (1.0 g q8h of meropenem + 0.5 g qd of ribavirin), dehydration treatment to reduce intracranial pressure (125 mL q8h of 20% mannitol + 250 mL of glycerol fructose q12h), diagnostic anti-tuberculosis treatment (0.3 g qd of isoniazid, 0.45 g of rifampin, 0.75 g of ethambutol, and 0.5 g of pyrazinamide; all *via* nasal feeding), 500 mg × 3 days of methylprednisolone intravenous infusion, 20 g × 5 days of intravenous immunoglobulin therapy, and control of epileptic seizures (diazepam and levetiracetam).

FINAL DIAGNOSIS

Infection with LM meningoencephalitis and SLE.

TREATMENT

On the 4th day of admission, mNGS of the CSF indicated LM (Table 1), and the blood and CSF cultures also tested positive for LM. Serum complement C3 and C4 levels were normal, and tests for T-SPOT, CSF latex agglutination, CSF TB-PCR, and autoimmune brain antibodies were all negative. The diagnostic evidence for tuberculous meningitis, cryptococcal meningitis, lupus activity, and lupus encephalopathy was insufficient, and the diagnosis was finally confirmed as sepsis caused by LM and LM meningoencephalitis. Therefore, the anti-tuberculosis drugs were discontinued, and the anti-infective regimen was adjusted to meropenem 2 g q8h + ampicillin 3 g q6h *via* intravenous infusion. The methylprednisolone dosage was reduced to 3 capsules qd *via* nasal feeding. Antiepileptic treatment, dehydration treatment to reduce intracranial pressure (125 mL q6h mannitol + 250 mL q12h glycerol fructose intravenous infusion), and symptomatic supportive treatment were continued.

Table 1 Metagenomic sequencing results of cerebrospinal fluid

Name	Number of detected sequences	Gene percentage	Copies/mL
Listeria monocytogenes	1317	195077 bp/6.34%	9.90^{E+02}

Table 2 Results of cerebrospinal fluid examination during hospitalization

Cerebrospinal fluid (reference interval)	Day 1	Day 7	Day 14	Day 30
Pressure (80-180 H_2O)	210	150	100	80
Color (transparent and clear)	Primrose	Colorless transparent	Colorless transparent	Colorless transparent
Leucocyte (0-8 × 10^6/L)	75	18	5	1
Erythrocyte (10^6/L)	110	7	5	3
Lymphocyte	16	90	3	1
Neutrophils	82	8	1	0.5
Macrophage	1	2	0	0
Eosinophilic granulocyte	1	0	0	0
Basophilic granulocyte	0	0	0	0
Sugar mmol/L	3	2.53	2.79	4.1
Chlorine	123	131	125	130
Protein	3.32	1.16	0.66	0
Bacterial culture	*Listeria monocytogenes*	Negative	Negative	Negative

OUTCOME AND FOLLOW-UP

The patient's consciousness gradually improved, and cognitive functions, such as the ability to calculate, gradually recovered. She could follow instructions and perform simple movements, and her body temperature remained normal. Repeated blood and CSF cultures showed no bacterial growth, and her condition improved. On July 24, the patient was discharged to a rehabilitation hospital for further rehabilitation treatment. During the treatment period in this hospital, lumbar punctures were re-examined in the 1st and 2nd weeks after admission. The biochemical and routine CSF findings were normal, and the CSF culture was negative. The patient experienced no fever, had clear consciousness, and was able to care for herself. She was considered clinically cured.

DISCUSSION

Infections of the CNS are still associated with high morbidity and even mortality[10]. LM belongs to the group of bacteria that have a specific potential to invade and access the CNS, mainly causing meningitis and rhombencephalitis. Neurolisteriosis typically affects immunocompromised individuals but can also occur in immunocompetent patients[11]. Particularly vulnerable populations include pregnant women, unborn babies, newborns, and the elderly[12]. The mechanisms by which LM invades the CNS and its associated tropism toward neural tissues are still poorly understood[13]. Since LM is mostly acquired orally through the ingestion of contaminated food, it seems apparent that the bacterium can be transported by the trigeminal nerve to the brainstem, resulting in LM-induced rhombencephalitis[14]. This pathology has been observed in more than 200 cases of neuropathological investigations in cattle, sheep, and goats, and in autopsies of patients who died from LM brainstem encephalitis[15-16]. These investigations revealed inflammation and infiltration of cranial nerves and their pathways[17-18].

The patient in this case was an elderly woman with weakened immune function owing to a history of SLE and prolonged hormone therapy, which she had recently discontinued. The onset of the disease was characterized by fever, headache, and neurological symptoms, such as altered consciousness and epileptic seizures. These symptoms, along with brain parenchymal damage, neck stiffness, and ischemic lesions on head CT and MRI, raised concerns of CNS infection, although lupus encephalopathy could not be ruled out. After admission, a complete lumbar puncture was performed as soon as possible, and routine CSF analysis, biochemical tests, smear and culture, mNGS, cryptococcal latex agglutination test, TB-PCR, and autoimmune brain antibody tests were conducted. Based on the patient's medical history and CSF findings, tuberculosis, bacterial, fungal meningitis, and lupus encephalopathy could not be ruled out. The patient's condition was critical and was progressing rapidly. Without timely and active treatment, there was a high risk of life-threatening conditions such as continuous epilepsy, respiratory failure, or brain herniation. Therefore, before determining

the cause of the infection, a broad treatment approach was initiated to take into account bacterial infections, tuberculosis, and lupus encephalopathy. Active antibacterial treatment, diagnostic anti-tuberculosis therapy, high-dose hormone shock, and immunoglobulin therapy were given. Through tests such as serum complement, T-SPOT, CSF latex agglutination, CSF TB-PCR, autoimmune brain antibodies, CSF mNGS, and both CSF and blood cultures, a final diagnosis of LM meningoencephalitis was confirmed. The treatment plan was adjusted as follows: High-dose hormones and anti-tuberculosis drugs were stopped and replaced by low-dose hormone maintenance. The patient was treated with meropenem 2 g q8h, ampicillin 3 g q6h, and measured to reduce dehydration to reduce intracranial pressure. Nutritional support was also provided, which gradually improved her condition.

In the treatment of patients with CNS infections, it is necessary to closely observe changes in the patient's body temperature, consciousness, breathing, pupil response, and blood gas levels. In case of brain herniation or respiratory failure, it is necessary to reduce intracranial pressure promptly, along with the provision of tracheal intubation and assisted ventilation *via* a ventilator to save the patient's life. During the treatment process, attention should be paid to maintaining airway patency, preventing phlegm obstruction, and strengthening nursing and nutritional support.

In addition, the patient had a history of untreated SLE, complicating the diagnosis. It was necessary to distinguish between SLE encephalopathy and secondary CNS infection related to SLE, as there are significant differences in the treatment methods for these two conditions. SLE encephalopathy typically requires high-dose hormone shock therapy, whereas CNS infections require timely and effective anti-infective drug treatment. However, the use of high-dose hormones in cases of infection may exacerbate the spread of infection, making it more difficult to control.

Identifying the pathogen is an important aspect of the diagnosis and treatment of infectious diseases. In this case, the patient's CSF routine and biochemical manifestations were atypical, which made it difficult to identify the type of pathogen. Therefore, after admission, in addition to routine blood and CSF cultures, the patient underwent a CSF mNGS examination as soon as possible. This quickly and accurately detected LM, which was later confirmed by blood and CSF cultures. Multi-disciplinary consultations and a thorough examination of SLE-related indicators helped rule out other CNS diseases such as SLE encephalopathy, autoimmune encephalitis, and cerebrovascular disease.

CONCLUSION

Listeria meningitis is an infectious disease of the CNS caused by LM, a bacterium widely present in the natural environment and transmitted through contaminated food and water sources. Patients usually show symptoms such as fever, headache, and neck stiffness. In severe cases, coma, convulsions, or even death may occur. Traditional diagnostic methods include CSF culture, serological detection, *etc.*, but these methods have certain limitations. Although CSF culture is the "gold standard" for diagnosis, it is time-consuming and has a relatively low positivity rate. Serological detection may result in false positive or false negative results, further complicating the diagnosis.

The emergence of mNGS technology has revolutionized the diagnosis of LM. It can quickly and accurately detect various pathogens in samples, including bacteria, viruses, and fungi. It offers several significant advantages over traditional methods. First, it is fast and accurate. mNGS can analyze a large number of nucleic acid sequences in a short time and quickly determine the pathogen species in the sample. Compared with traditional CSF culture, mNGS can greatly shorten the diagnosis time and provide strong support for the timely treatment of patients. Second, it has high sensitivity and specificity. It can detect pathogen nucleic acids at extremely low concentrations, improving diagnostic sensitivity. Moreover, it distinguishes between different pathogens through precise analysis of nucleic acid sequences, improving diagnostic specificity. In some complex cases, there may be infections caused by rare pathogens or mixed infections. Traditional diagnostic methods often struggle to identify such infections, whereas mNGS can comprehensively detect various pathogens in samples and provide more information for accurate diagnosis.

FOOTNOTES

Author contributions: Xu DZ and Tan QH jointly collected the data and co-authored this paper. Both authors have read and approved the final version of the manuscript for publication.

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