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Meningoencephalitis due to *Listeria monocytogenes* in a Young Immunocompetent Patient: Case Report with Literature Review

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Abstract

Meningoencephalitis secondary to *Listeria monocytogenes* (*L. monocytogenes*) mainly affects newborns, the elderly and immunocompromised people; there are extremely rare cases in which said infection occurs in immunocompetent individuals. We present the case of a young adult patient without immunocompromise, who developed meningoencephalitis due to *L. monocytogenes*; This case is exceptional, since it occurred in an individual outside the classic age group, in addition to not having risk factors, which is why it should be considered an atypical causal agent.

Keywords

Meningitis, Encephalitis, Rhombencephalitis, *Listeria monocytogenes*, Immunocompetent

1. Introduction

L. monocytogenes was first isolated by E.G.D. Murray *et al.* in 1926 during an epidemic in animals, mainly guinea pigs and rabbits. It was originally called Bacterium monocytogenes because of the increase in monocytes found in the blood of infected animals; it was later named Listerella hepatolytica, in honor of Joseph Lister, the pioneer in the field of antisepsis, and finally in 1940, it adopted the name *Listeria monocytogenes* [1].

It is frequently acquired through consumption of unpasteurized dairy prod-

ucts. It is isolated from wastewater from containers and the soil, where it can live for more than 295 days [2].

In the risk factors, it is mentioned that it is more likely to be contracted in pediatric patients, pregnant women, over 65 years of age, and immunocompetent patients. For this reason, this study was found where it is stated that in pediatric age there is an estimated incidence of invasive listeriosis of 3 - 6 cases per million inhabitants per year [3]. In pregnant women, *L. monocytogenes* infection occurs 13 to 20 times more frequently than in the general population, with an incidence of listeriosis in pregnancy of 12 per 100,000 compared to an incidence of 0.7 per 100,000. in the general population [4].

The EU prevalence of *L. monocytogenes* varied amongst different food products with the highest recorded in fish (10.4%), followed by meat products (2.07%) and cheese (0.47%). The notification rate of human listeriosis in the US was reported to be approximately 0.3 cases per 100,000 population. In the US, approximately 1600 individuals get listeriosis annually with 21% case fatality rate. Almost all the fatalities were reported in high-risk groups, such as older adults, pregnant women and people who were immunocompromised [5].

In *L. monocytogenes* leukocytosis frequently occurs together with meningoencephalitis. With a leukocyte count of 100 to 10,000 cells/mm^3, polymorphonuclear predominance, and protein morrachia, the cerebrospinal fluid (CSF) is typically purulent; in 60% of cases, glucorrhachia is normal. 60% - 75% of blood cultures yield favorable results. Although gram-positive coccobacilli are often confused with other microbes, gram staining in CSF can reveal them and guide the first empirical treatment against this agent. It can occasionally be negative due to the stain's limited sensitivity (0% - 40%) for these particular bacteria [6].

The CSF results of meningitis caused by *L. monocytogenes* resemble those of other bacterial meningitis. Differentiating between bacterial meningitis causes can be achieved using CSF culture and microscopy. Nevertheless, the yield of Gram stain is typically low, and the diagnosis is validated only upon isolation of *L. monocytogenes* from CSF culture. Due to its sluggish growth, the identification process takes roughly five to seven days in total [7].

L. monocytogenes can cause more serious invasive diseases, including sepsis, central nervous system (CNS) infection, and endocarditis. Serotypes 1/2a, 1/2b, and 4b are responsible for most human cases [8].

The objective of this report is to consider *L. monocytogenes* as a possible atypical etiological agent of meningoencephalitis in patients without immunocompromise.

2. Clinical Case

We present the clinical case of a 23-year-old male patient, previously healthy, without comorbidities, without history of chronic diseases, without history of immunocompromise, with the only relevant history of having ingested unpasteurized dairy products one month before the onset of his symptoms.

He began his illness 7 days prior to his admission, with fever, headache holocranial, nausea and vomiting; He went to receive medical attention for alteration of alertness. In the neurological examination upon his admission, no alteration of the cranial nerves was identified, the extremities were normotrophic and normotensive, muscle stretch reflexes were present and normoreflexic, Babinski signs and negative substitutes, nuchal rigidity was evident. At the time of his admission, the patient presented neurological and ventilatory deterioration, which is why it was decided to perform advanced management of the airway. Given a condition compatible with bacterial neuroinfection, it was decided to start empirical treatment with meropenem to cover the most common pathogens.

Within the diagnostic approach, it was decided to perform a CT scan, which showed dilation of the ventricular system, as well as transependymal leak and loss of the arachnoid mantle (Figure 1), compatible with obstructive hydrocephalus, so it was decided to admit the patient to the operating room for placement of an External Ventricular Drainage (EVD). During the surgery, cloudy cerebrospinal fluid was observed, which was analyzed showing (add cytochemical data), in the same way Gram stain was requested, in which bacilli were identified gram positive.

The patient remained febrile and without improvement in his neurological status. Eight days after the surgical procedure, there was evidence of expulsion of cellular debris through the EVD, which caused his dysfunction, so it was decided to admit the patient for a replacement of the system. However, the patient developed hydrocephalus again, which probably caused herniation and the patient's death 9 days after his admission.

The CSF culture result demonstrated the presence of *L. monocytogenes* (Figure 2). However, this could be analyzed after the patient's death, so the change

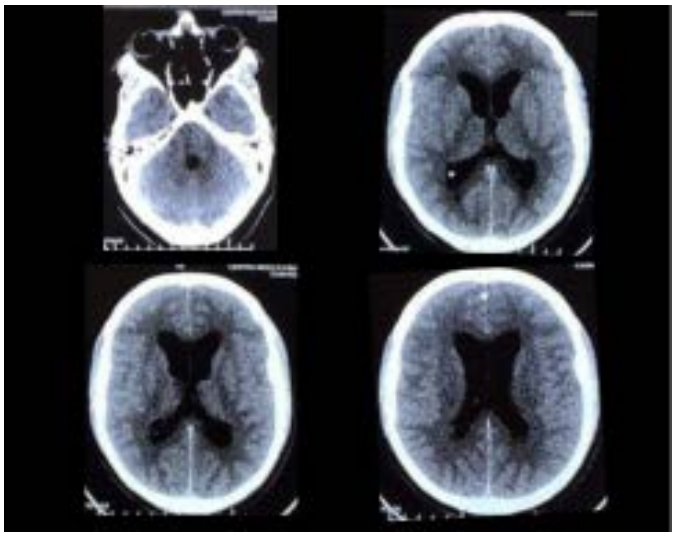

Figure 1. Simple axial computed tomography of the skull, in which a dilated fourth ventricle is observed, as well as a rounded third ventricle, lateral ventricles with predominance of occipital horns, with evidence of transependymal leak, as well as loss of the arachnoid mantle towards the convexity.

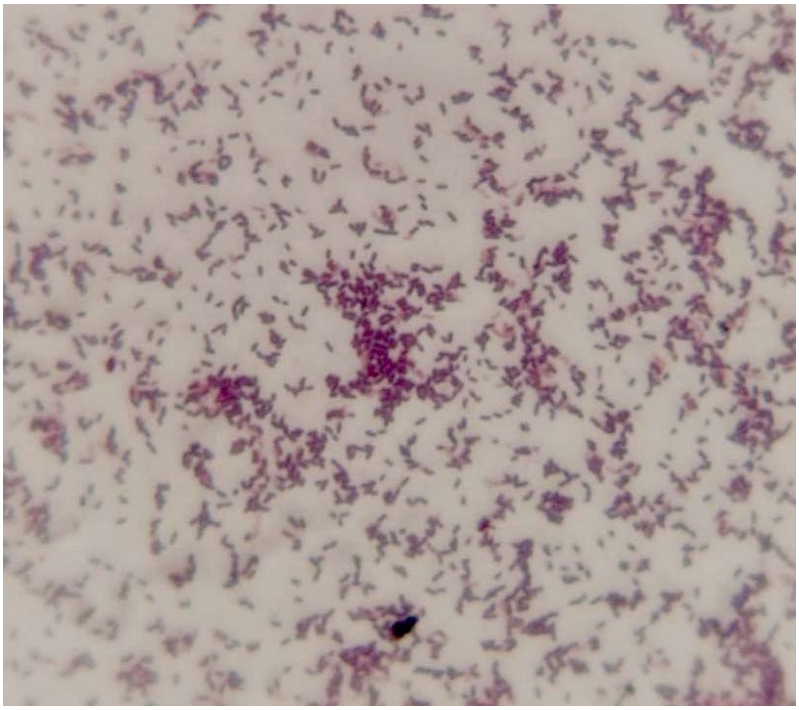

Figure 2. CSF sample with Gram stain, in which Gram-positive bacilli are identified.

to an antibiotic regimen directed at the identified pathogen could not be made.

3. Discussion

In the literature, only isolated cases of *L. monocytogenes* infection in immunocompetent patients have been reported. A search was carried out in Pubmed using the words "*Listeria monocytogenes*" and "immunocompentent" obtaining 159 results. It was identified that most reported cases are cases of infection by *L. monocytogenes* in its presentation as neurolisteriosis in cases of immunocompetent patients.

Mohan *et al.* reported the case of a 56-year-old man with no history of immunocompromise who developed cerebritis secondary to *L. monocytogenes* to which was added the development of endocarditis. The patient died 3 weeks after admission (Mohan 2023) [9].

Zhao *et al.* reported the case of a 23-year-old female with no previous history of immunocompromise or other chronic diseases, only with a history of ingesting poorly cooked meats, who developed meningitis complicated with ventriculitis and obstructive hydrocephalus, who died 12 hours after her income [10].

Cao *et al.* reported the case of a previously healthy 50-year-old man, with no comorbidities or history of immunocompromise, who was misdiagnosed with facial neuritis, treated unsuccessfully with dexamethasone. In the following days the patient developed signs of neuroinfection, MRI was performed. in which lesions were observed in the brainstem, compatible with rhombencephalitis, *L. monocytogenes* was identified in CSF [11].

Richards *et al.* presented the clinical case of a 46-year-old, previously healthy

male who developed signs of neuroinfection accompanied by cranial nerve deficits, with signs of rhombencephalitis on MRI, in which *L. monocytogenes* was identified in the CSF, treated With ampicillin, the patient progressed favorably and 6 months after his illness, he only presented spasticity on the right side of his face [12].

L. monocytogenes infection is acquired after ingestion of contaminated dairy products, the bacillus crosses the intestinal epithelium and spreads by lymphatic and hematogenous routes to the spleen and liver, finally crossing the blood-brain barrier [13].

There is a wide variety of clinical syndromes caused by listeriosis. *L. monocytogenes* infection characteristically has 2 forms of presentation; it can appear as meningoencephalitis, with the characteristic triad of fever, headache and meningeal irritation [14]. The second is the development of rhombencephalitis with the development of cranial nerve abnormalities and cerebellar dysfunction [15].

For patients with *Listeria* central nervous system (CNS) or bloodstream infection, we treat with either ampicillin or penicillin, each combined with gentamicin. Regimens without gentamicin may be preferable for patients who have impaired renal function or are taking other nephrotoxic drugs, such as cyclosporine. If gentamicin cannot be used, we generally favor the combination of ampicillin plus trimethoprim sulfamethoxazole intravenously per day divided every 6 to 12 hours, with the higher end of the dosing range used in patients who are severely ill [16].

L. monocytogenes infection has a poor prognosis; some studies claim that *L. monocytogenes* is the third leading cause of death from foodborne illnesses in the United States [17].

4. Conclusion

L. monocytogenes infection occurs mainly in immunocompromised patients; However, it should be considered as an atypical agent in immunocompetent patients who present important risk factors and meet the clinical manifestations.

5. Ethical Considerations

The authors declare that they have met al. ethical responsibilities regarding data protection, right to privacy and informed consent.

Authorization from the institution's ethics committee is not necessary since at no time do they fail to comply or violate patient anonymity rules, nor is any experimental procedure performed that puts the patient's integrity at risk.

The authors declare that this article does not contain personal information that would allow the patient described to be identified, which makes the patient's informed consent unnecessary for the publication of the article.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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