Case Report

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Listeria monocytogenes Meningoencephalitis in Immunocompetent Patient- Clinical Course and Outcome

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Abstract

Listeria monocytogenes is the third most frequent cause of bacterial meningitis. It was an unusual etiological agent in immunocompetent patients. We described a 29-years-old male patient with *L. monocytogenes* sepsis with meningoencephalitis hospitalized in the Clinic of Infectious Diseases, University Multiprofile Hospital of Active Treatment, Stara Zagora, Bulgaria. His initial complaints were headache, nausea and vomiting. At the admission, clinical examination showed severe meningeal irritation-neck stiffness and signs of Brudzinski, Kernig and Babinski. Cerebrospinal fluid (CSF) was slightly opalescent with changes as with viral infection of the central nervous system (CNS). Causative agent *L. monocytogenes* was proven from CSF and blood. The disease was very severe. Changes in consciousness and acute respiratory and cardiovascular failure imposed a two-month stay in intensive care unit. The patient was discharged after a hospital stay of 125 days. Within two years thereafter he was fully recovered without residual manifestations

Keywords: *Listeria monocytogenes*; Meningoencephalitis; Immunocompetent patient

Introduction

L. monocytogenes is a motile, non-spore-forming, gram-positive bacillus that has aerobic and facultative anaerobic characteristics [1]. *Listeria monocytogenes* emerged as an important foodborne pathogen in the latter part of the 20th century. Primary clinical picture manifests as intestinal infection - diarrhea, nausea and vomiting [2].

Clinical syndromes caused by this microorganism include also sepsis in the immunocompromised patient, meningoencephalitis in infants and adults and febrile gastroenteritis [3].

In the literature, meningitis due to *L. monocytogenes* is described as a disease in immunocompromised patients, pregnant women, newborns and elderly individuals with high case-fatality rates (24% to 62%) [4]. At least 90 percent of affected patients are in one of these risk groups. It is observed casuistically rare in young, healthy people whit no comorbidities [5].

This is one of the causes of infection in the CNS, along with the *Str. pneumoniae*; *H. influenzae*; *N. meningitidis* [6].

Case Report

We reported a case of a 29-year-old man with *Listeria monocytogenes* sepsis with meningoencephalitis. He lived in a little village near Stara Zagora town, Bulgaria. He was in a previously good health status. Clinical, laboratorial, microbiological, instrumental and epidemiological investigations were done. On April, 2015 he was admitted at the Clinic of Infectious Diseases at the University Hospital, Stara Zagora, Bulgaria with complaints consisted of nausea, vomiting, fever and chills from the previous day. There were general weakness, muscle and joint pains, too. Neurological examination showed positive meningeal presentations-severe neck stiffness, Brudzinski neck sign, Brudzinski symphyseal sign, Kernig's sign. Initial lumbar puncture was with slightly opalescent CSF under pressure. Biochemical investigation showed parameters as with aseptic meningitis **(Table 1)**. The diagnosis on admittance was acute meningitis with unspecified causative agent. Etiological therapy with antibiotics-ceftriaxone and amikacin was immediately started. Treatment included also Sol. Mannitholi 10% and dexamethasone as well as glucose-saline solutions, antipyretics vitamins and probiotic.

Two days after the patient's status had improved with normal temperature, decreased headache and meningeal signs.

On the third day there was a second temperature pick up to 38.6°C. Nausea and vomiting recovered. Another lumbar puncture (LP) was made but results were similar to the first one.

The patient became nervous, with mental confusion. Paralysis of cranial nerves-n. abducens and n. oculomotorius-with ptosis of the eyelids, anisocoric pupils and horizontal nystagmus had been noticed. New LP showed data for a bacterial neuroinfection. Microbiological examinations proved *Listeria monocytogenes.*

Magnetic resonance imaging (MRI) was performed and massive cerebral edema appeared. The initial antibiotics were changed with a combination of Ampicillin plus Meropenem.

Over the next three days the patient has no changes in general condition, he is afebrile, conscious but inadequate most of the time. The control LP shows a relatively good influence on the laboratorial CSF parameters. *L. monocytogenes* had not been established anymore. On the beginning of the second week after hospitalization the patient sharply worsened, there was no verbal contact and comatose status was established. Because of dysrhythmias in breathing, necessitating relocation in ICU and putting him on artificial pulmonary ventilation were done.

Diagnosis was formed as sepsis due to *L. monocytogenes* with purulent meningoencephalitis after receiving the result of blood culture with the same causative agent.

The temperature moved in values from 37.3°C to 40°C. Intermittent return of consciousness, as attempts to implement the verbal commands-open eyes, showing tongue, clenching his fists regain was observed for a short time.

Computer tomography imaging with contrast showed persistent cerebral edema and CSF's data confirmed them with persistent elevated levels of albumin up to 1.8 g/l. The condition is considered as hydrocephalus. Surgery with insertion of external ventricle drainage was performed. Two weeks later was made a second neurosurgery by placing ventricle - peritoneal anastomosis. The general condition of the patient however deteriorated and tendon reflexes did not appear. Cardiovascular function was getting worse and hypotension persisted despite of the application of dopamine. There was no response to pain. GCS was 3-4 points with sluggish pupil reaction. There were oral automatisms.

Performed a month later brain tomography showed a persistence of mild cerebral edema. This required revision of the ventricle - peritoneal shunt. After that the patient's status began to improve. He performed verbal commands, but did not speak and reacted with pain when bending of the limbs. There was good pupil photoreaction. The patient remained permanently afebrile.

Three months after hospitalization the patient was disconnected from artificial pulmonary ventilation. He was in consciousness and was completely adequate. There was a muscular hypotension and contracture of the fingers of the right hand. Four limbs were moving in a limited volume. The patient was discharged with improvement in overall physical and mental condition after 125 days of hospitalization. Conducted physiotherapy led to improved motor abilities. A control CT revealed no pathological changes.

A month after discharge the patient could move on his own. He could talk, but with vocabulary deficits. EEG made in an outpatient setting did not show deviations.

Table 1 Laboratorial findings in CSF in the patient with *L. monocytogenes* meningoencephalitis.

Variable (unit)	Reference range	At the admittance	2nd day	8th day	15th day	40th day	76th day
Glucose (mmol/L)	2.5-3.9	2.9	2.3	1.9	2.4	3.5	2.86
Protein (g/L)	0.15-0.45	1.2	0.65	2.54	1.7	1.8	0.86
White blood cells	0-6	347	320	1669	800	25	1
Neutrophils (%)	/	41	43	73	19	--	--
Erythrocytes	/	23	16	96	80	12	10
Chlorides (mmol/L)	115-132	114	118	102	111	--	112
Pandy's	-	+	+	++	+	--	-

Discussion

At the largest percentage of cases, *Listeria monocytogenes* infection affected immunocompromised patients [7]. Despite of our patient had no such data, the disease had severe clinical course with the both of purulent meningoencephalitis and sepsis. According to the literature data this was only observed in 6% of all cases of *Listeria monocytogenes* infection of the CNS.

Meningitis/meningoencephalitis had been observed in more than half of adults with Listeriosis. Symptoms occurred in about four days with flu-like manifestations and expressed in fever, MRS, tremors, convulsions and mental changes. The symptoms in neuro-infections due to *L. monocytogenes* were

not significantly different from those in other bacterial meningitis. As regards the results of biochemical investigation of CSF leukocyte counts may vary from a few tens to a few thousand, but CSF glucose not always shows change [8].

As in the described case, microbiological testing could be positive shift in the later stages of the disease. The examination of blood culture greatly assisted the diagnosis [9].

At the largest percentage of cases with purulent meningitis/ meningoencephalitis the therapy started with third generation cephalosporin in combination with an aminoglycoside or vancomycin. After isolation of the causative organism *Listeria monocytogenes* had to be included ampicillin or penicillin [10]. There was no conclusive data in the literature for poor prognosis and outcome of the disease due to the delay in etiological therapy.

Most frequent neuroimaging studies of brain was brain edema, expressed in varying degrees, as was the case with our patient.

L. monocytogenes was one of the most common bacteria causing hydrocephalus [10]. In about 5% of the cases the complication was cerebral abscess [7].

Regarding the outcome of the disease, the best idea gave Glasgow outcome scale. 1. Death; 2. Persistent vegetative state. Severe damage with prolonged state of unresponsiveness and a lack of higher mental functions; 3. Severe disability. Severe injury with permanent need for help with daily living; 4. Moderate disability. No need for assistance in everyday life, employment is possible but may require special equipment; 5. Low disability. Light damage with minor neurological and psychological deficits [11].

In the mentioned clinical case the outcome on this scale is 5. According to the literature in *Listeria monocytogenes* infection occurring as meningoencephalitis, mortality was up to 35%. [11].

Conclusion

Diagnosis *Listeria monocytogenes* meningoencephalitis was a challenge, especially in immunocompetent patients. Epidemiological study was also very important. Focal infections at other sites are less frequent. Listeria species are commonly found in raw and unprocessed food products. Major outbreaks of listeriosis, with high morbidity and mortality, have been caused by a variety of foods, including soft cheeses, delicatessen meats, and vegetable products.

Microbiological testing of cerebrospinal fluid and blood cultures were the methods for prompt and adequate diagnosis and appropriate treatment.

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