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LISTERIOSIS INFECTION IN PREGNANT WOMEN AND CHILDREN (LITERATURE REVIEW AND CASE OBSERVATIONS)

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Summary.

Listeriosis is classified among highly hazardous infectious diseases. The infection predominantly affects immunocompromised individuals, particularly neonates and pregnant women, in whom it frequently manifests with severe central nervous system (CNS) involvement, including meningitis, meningoencephalitis, or brain abscess, and may culminate in recovery with disabling neurological sequelae or death.

Objective. *To emphasize the clinical significance of listeriosis, delineate its major clinical manifestations, and present contemporary diagnostic and therapeutic approaches, integrating data from the literature with the authors' own clinical observations.*

Materials and Methods. *A comprehensive literature review on listeriosis in pregnant women and children was conducted, supplemented by a case report of congenital listeriosis in a neonate whose infection originated from a mother with subclinical disease. Bacteriological examination confirmed Listeria monocytogenes in the cerebrospinal fluid. The clinical course was characterized by moderate systemic intoxication and meningoencephalitis complicated by hydrocephalus, which had been detected prenatally, pointing to intrauterine transmission. Listeria monocytogenes exhibited resistance to combination antibacterial therapy with ampicillin and gentamicin.*

Conclusions. *Listeriosis presents with a broad clinical spectrum, ranging from mild infection to fulminant sepsis, with the highest risk associated with CNS involvement. Neonates and pregnant women represent the most vulnerable groups due to impaired host defenses. Evaluation for listeriosis should be considered in all neonates with CNS disease to enable timely diagnosis and initiation of appropriate therapy.*

Keywords: *Children; Pregnant women; Newborns; Listeriosis; Meningitis; Sepsis; Diagnostics; Treatment.*

Introduction

Listeriosis is an infectious disease characterized by diverse transmission routes, polymorphic clinical manifestations, and frequent involvement of the central nervous system (CNS) with the potential development to septic states. In some cases, the disease may progress as prolonged asymptomatic carriage, while in neonates and immunocompromised patients, it is often associated with high mortality rates [3, 8, 13, 20].

According to the Public Health Center of the Ministry of Health of Ukraine, 22 cases of listeriosis were registered in Ukraine between 2007 and 2017, including 11 cases in 2017. Six patients died, of whom four were neonates. Listeriosis incidence in Ukraine remains sporadic, with isolated cases reported across various regions. In 2018, two cases were documented: one involved a young woman in the Lviv region with the anginoseptic form of listeriosis, who fully recovered after treatment; the other was fatal, involving a 69-year-old man in Kyiv who succumbed to listerial encephalitis. In the same year, the European Food Safety Authority reported 47 cases of listeriosis in Europe, 10 of which were fatal [5, 7, 16].

Data from the Ukrainian Public Health Centre indicate that the risk of developing listeriosis in pregnant women exposed to *Listeria monocytogenes* is approximately 20-fold higher than in the general adult population, and 300-fold higher in individuals with HIV/AIDS [3,4].

In recent years, *Listeria monocytogenes* has been recognized as a TORCH pathogen, capable of inducing pathological pregnancy outcomes and congenital abnormalities in the fetus and neonate [3].

Currently, two species of the genus *Listeria* are considered pathogenic: *L. monocytogenes*, which affects both humans and animals, and *L.* ivanovii which is primarily pathogenic for animals and only rarely for humans. *L. monocytogenes* are motile, Gram-positive bacilli with flagella and do not produce endospores. They are facultative anaerobes capable of forming L-forms and surviving intracellularly,, a feature that can reduce the efficacy of antibiotic therapy in certain cases. Seven distinct lineages of *L. monocytogenes* have been identified [2, 3].

L. monocytogenes exhibits substantial environmental resilience, with the capacity to grow across a wide range of temperatures (3-42 °C), pH levels (5.5-9.5), and humidity. The bacterium can replicate at refrigeration temperatures (4-6 °C) in soil, water, plant material, and the tissues of deceased humans and animals. In plant-derived feed, including leaves, stems, and roots, the pathogen may persist for 2-3 years. Thermal inactivation occurs at 70 °C for 20-30 minutes or at 100 °C for 5 minutes. The pathogen is susceptible to 5% phenol and 10% chlorine solutions [3].

Principal virulence factors comprise listeriolysin O, internalins A and B, phosphatidylinositol- and phosphatidylcholine-specific phospholipases, ActA protein,

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metalloproteases, and the transcriptional regulator Prf A. Sixteen serological antigenic types have been described, based on combinations of 15 somatic and 4 flagellar antigens. Globally, as in Ukraine, approximately 90% of listeriosis cases are attributable to serotypes 1a, 1b, and 4b [3, 10].

Upon entry into the host, *L. monocytogenes* induces phagocytosis in multiple cell types, including non-phagocytic cells, mediated by the bacterial proteins internalin A (InlA) and internalin B (InlB). The pathogen then escapes from the phagosome into the cytoplasm of the host cell using the pore-forming toxin listeriolysin O (LLO). During the acute phase of infection, *Listeria* elicits a cascade of histopathological tissue changes, often resulting in granuloma formation [10, 18].

Chronic asymptomatic intestinal carriage is possible; according to published reports, *L. monocytogenes* has been detected in the stool of approximately 5% of clinically healthy individuals [3].

Transmission of *Listeria* occurs via fecal-oral, aerogenic, and transplacental routes. Risk factors include contact with infected animals or birds, consumption of unpasteurized milk, raw or smoked meat and fish products, and unwashed vegetables and fruits. Between 15% and 70% of fast-food meals may harbor the pathogen. *Listeria* is capable of persisting and even proliferating in refrigerated foods. Human-to-human transmission occurs primarily through fecal-oral mechanisms [4, 7, 9, 13].

Occupational exposure is possible through handling of animal raw materials, including leather, wool, bristles, hides, fluff, and feathers. Direct contact transmission may occur via cuts or abrasions contaminated with biological fluids from infected animals. Vertical transmission from mother to fetus during intrauterine development is also possible. Most pregnant women infected with *Listeria* remain asymptomatic; however, if clinical manifestations do occur, typically in the third trimester, the disease course is generally mild. In contrast, neonatal listeriosis is characterized by a severe course and is frequently fatal [9, 11, 20].

The incubation period may extend up to 3 weeks. Following bacteremia, *Listeria* exhibits marked tropism for the CNS and placenta. Additionally, the respiratory mucosa, conjunctiva, and damaged skin may serve as portals of entry [1, 2, 6, 8, 20].

The clinical course of listeriosis can be categorized as acute (1-3 months), subacute (3-6 months), or chronic (more than 6 months). Clinical classification distinguishes the following forms: glandular; gastroenteric; neurological (meningitis, meningoencephalitis); septic; and bacteria carrying. Rare manifestations include endocarditis, arthritis, dermatitis, osteomyelitis, parotitis, and urethritis. Listeriosis in pregnant women and the septic-granulomatous form in infants are classified separately. In full-term healthy neonates, a delayed form of listeriosis may present as meningitis or sepsis. The incubation ranges from 1 day to 4 weeks, and less commonly up to 1.5-2 months [6, 17].

The glandular form (anginal-glandular and ocular-glandular) is more frequently described in the literature, occurring in 15-20% of cases. The anginal-glandular form is characterized by a mononucleosis-like syndrome, featuring fever, intoxication, ulcerative-necrotic or membranous tonsillitis, enlargement of the submandibular, cervical, and axillary lymph nodes, and less commonly, hepatosplenomegaly. Peripheral blood analysis reveals monocytosis. The ocular-glandular form of listeriosis is characterized by unilateral purulent conjunctivitis with manifestations of general intoxication and lymphadenopathy (enlargement of the parotid and submandibular lymph nodes; less commonly, the cervical and occipital lymph nodes). The disease duration ranges from 1 to 2 months [9, 20].

The neurologic form of listeriosism, also called neurolisteriosis, occurs predominantly in children under 3 years of age and adults over 50 years of age. This form presents with typical manifestations of meningitis or meningoencephalitis. Although listeriosis accounts for only 1% of all bacterial meningitis cases, the prognosis for the neurologic form is generally poor, with a mortality rate of up to 30%; furthermore, 7% of patients experience recurrent meningitis. Severe complications of neurolisteriosis include hydrocephalus [8, 13, 14, 19].

In severe cases, listeriosis sepsis may develop, with a mortality rate of approximately 60%. This form is typically characterized by the formation of numerous necrotic nodules (listeriomas) in the lymph nodes, internal organs, and central nervous system. These nodules consist of the pathogens themselves, reticular and monocyte cells, nuclear debris, and altered polymorphonuclear leukocytes [12, 13].

The septic form of listeriosis presents with characteristic clinical symptoms: pronounced intoxication, hepatolienal syndrome, hectic fever, and skin rash – particularly around large joints (maculopapular or erythematous exanthema) and on the face (in a 'butterfly' distribution). Most patients with the septic form develop hepatitis with signs of jaundice, pneumonia, pyelitis, gastroenteritis, polyserositis, infectious-toxic shock (ITS), and disseminated intravascular coagulation (DIC) syndrome [12, 13].

L. R. Shostakovich-Koretska et al. (2021) described a case of listeriosis that manifested as sepsis and was not recognized ante mortem in a 4-month-old child due to the rapid progression of the infectious process.

The case history indicated that the mother had cystitis at 20 weeks of gestation [7], a threatened miscarriage at 22 weeks, pyelonephritis at 27 weeks, and an acute respiratory disease with a temperature rise to 38 °C at 38 weeks. Upon admission, the child presented with a temperature of 38.3 °C, watery diarrhea (10-12 episodes per day), adynamia, and episodes of agitation alternating with lethargy and drowsiness. Subsequently, the child's condition progressively worsened. A vesicular-papular rash developed on the trunk and extremities. Pulmonary examination revealed mixed dyspnea, SpO_2 90% on room air, respiratory arrhythmia, tachypnea, grunting respiration, and marbled skin that was cold to the touch. Twenty-four hours after the onset of septic shock symptoms, signs of a brain crisis emerged in the form of clonic-tonic seizures against the background of meningoencephalitis. The child was transferred to mechanical ventilation. The disease course was unfavorable, and biological death was confirmed on the third day of hospitalization. *Clinical diagnosis:* enterovirus infection, unspecified, complicated by multiple organ failure

syndrome, acute liver and kidney failure, disseminated intravascular coagulation syndrome; cerebral edema; intestinal paresis; grade 3 respiratory failure; circulatory failure grade 2B; anaemia grade 2, and cryptogenic hepatitis. The pathological diagnosis was entirely inconsistent with the clinical findings: listeriosis septicemia with numerous *Listeria* in various organs; multiple organ failure syndrome; cerebral edema; pulmonary edema; dystrophic damage to the abdominal organs, brain, lungs, and skin; erosive colitis; peritonitis; encephalitis; polysegmental pneumonia; necrotic changes in the dermis; and granulomatous hepatitis. Histological examination revealed multiple granulomas (listeriomas) in the liver, spleen, meninges, pancreas, kidneys, thymus, soft meninges, and vascular plexuses of the lateral ventricles of the brain. The granulomas consisted predominantly of macrophages, with secondary necrosis present in the center of some granulomas. Postmortem bacteriological examination of the blood, lungs, intestines, liver, and spleen identified *Listeria monocytogenes*, type I, in the lung tissue and in the small and large intestines. These findings highlight the diagnostic challenges associated with listeriosis [7].

O. V. Prokopiv et al. (2021) described a rare case of listeriosis manifesting as meningitis in a 4-year-old girl, characterized by a protracted disease course and an absence of clear correlation between the general condition and cerebrospinal fluid (CSF) dynamics. The administration of combined antibiotic therapy resulted in the complete clinical recovery of the patient on the 28th day of inpatient treatment. This case underscores the utility of considering the results of bacterioscopic CSF examination (detection of gram-positive rods on microscopy and isolation of *L. monocytogenes* on bacteriological culture) [6].

In pregnant women, the pathogen can adhere to placental tissue and form listeriomas, which represent the primary cause of intrauterine fetal infection. A decrease in cellular immune function during pregnancy creates a favorable environment for listeriosis manifestation. Consequently, the incidence among pregnant women exceeds 25% of all reported listeriosis cases, with a higher frequency of infection registered during the third trimester [15,17,18].

The risk of transplacental fetal infection arises not only in cases of manifest listeriosis but also during bacterial carriage. At the peak of clinical symptoms, typically occurring in the third trimester, women exhibit influenza-like manifestations, including short-term fever, catarrhal symptoms, conjunctivitis, and myalgia. Initially, the pregnant woman acts as the source of infection for the fetus through the formation of placental listeriomas; subsequently, a vicious cycle is established within the «mother–placenta–fetus» system, wherein the fetal compartment of the placenta becomes a source of secondary infection to the mother – a presentation often misinterpreted as fever of unknown origin [15, 17].

In approximately one-third of cases, listeriosis in pregnant women remains asymptomatic; however, vaginal colonization with the pathogen occurs, posing a fetal infection risk. Transplacental infection may occur at any gestational stage. Intrapartum infection occurs during passage through the birth canal [15].

In cases where transplacental infection does not lead to intrauterine fetal demise, the neonate typically presents with manifestations of congenital listeriosis, including prematurity and intrauterine growth restriction. Clinical deterioration may occur precipitously within the first 48 hours of life. A papular or papular-hemorrhagic rash frequently develops, accompanied by the onset of multiple organ failure syndrome. The septic-granulomatous form of neonatal listeriosis is marked by a severe clinical course and high perinatal mortality (approximately 25%). Among survivors, 5-20% exhibit persistent neurological sequelae [5, 9, 11].

Aspiration of Listeria-contaminated amniotic fluid leads to severe pulmonary damage in newborns, resulting in extremely high mortality (50%). When clinical manifestation occurs on days 10-12 of life, the disease typically presents as listeriosis meningitis with high mortality rates reaching 25% [9, 15, 17].

Listeriosis meningitis demonstrates several characteristic features distinguishing it from other meningitis types: low-grade fever, typical symptoms of focal cranial nerve involvement (ptosis, anisocoria, strabismus, pathological reflexes, and occasionally paralysis and mental disorders), with rarely pronounced meningeal signs. Cerebrospinal fluid typically demonstrates a lymphocytic profile with moderately elevated protein conten; occasionally, neutrophilic-lymphocytic cytosis is observed. Glucose and chloride levels typically remain unchanged [21].

Definitive diagnosis requires positive results from bacteriological examination of blood, cerebrospinal fluid, synovial fluid, tonsillar lacunae contents, lymph node aspirates, ocular discharge, amniotic fluid (obtained via amniocentesis), or placental tissue (obtained via chorionic villus sampling), followed by antibiotic susceptibility testing.

Serological testing may yield false-positive results due to antigenic similarity between *Listeria* and staphylococci, enterococci, and erysipeloid pathogens. Consequently, currently available test systems lack sufficient specificity for reliable detection of Listeria-specific antibodies. Therefore, direct cultural methods (culture) and polymerase chain reaction (PCR) represent the primary diagnostic modalities in clinical practice. Complete blood count often reveals leukocytosis with monocytosis and occasionally atypical mononuclear cells. Cerebrospinal fluid analysis typically shows neutrophilic-lymphocytic cytosis with neutrophil predominance and characteristic cell-protein dissociation [1, 2].

For the treatment of severe disease forms (septic and neurological) and neonatal listeriosis, a combination of penicillins and aminoglycosides is employed. Etiotropic therapy is administered throughout the entire febrile period, typically for 10-14 days, and for listeriosis meningitis – for 14-21 days. In severe cases with central nervous system involvement and/or visceral organ damage, glucocorticosteroids are prescribed concomitantly with antibacterial therapy at a dosage of 1-2 mg/kg/day (prednisolone equivalent) for 7-10 days [3].

For localized forms with mild symptoms, outpatient treatment may be implemented using ampicillin, azithromycin, doxycycline, or sulfamethoxazole/trimethoprim. Pregnant women are prescribed ampicillin. Women who deliver an

infected child, regardless of their clinical status, receive a therapeutic course of ampicillin or doxycycline [3, 9].

Awareness of listeriosis clinical manifestations and diagnostic approaches remains limited; cases are not consistently documented in statistical reports or medical literature, contributing to underestimation of its relevance. However, listeriosis is more prevalent than recognized, particularly with application of modern diagnostic methods. Furthermore, literature indicates mortality rates ranging from 20% to 60%, primarily attributable to delayed diagnosis.

The objective of our work is to highlight the clinical significance of listeriosis infection, describe its clinical manifestations, and present contemporary diagnostic and therapeutic approaches based on our clinical experience and literature data.

Materials and methods. We present a case of congenital listeriosis in an 11-day-old newborn treated at the Khmelnytskyi City Cildren's Hospital in October 2024, a twin aged 11 days. This work utilizes modern diagnostic recommendations for listeriosis and analyzes laboratory (clinical, biochemical, bacteriological) and instrumental (ultrasonography, radiography) examination methods performed on the newborn.

Clinical observation. The child was born at the Khmelnytsky Regional Perinatal Centre, where on the third day of life he was diagnosed with prematurity and being underweight for his gestational age. Additional diagnoses included bacterial meningitis, bacterial sepsis of the newborn, respiratory distress syndrome, respiratory disorder syndrome, grade II ventriculodilation, and hypoxic-ischaemic central nervous system damage.

The patient was born from the mother's first pregnancy and first delivery as the second of dichorionic diamniotic twins, in breech presentation, via cesarean section. Birth weight was 2400 g, body length 46 cm, and gestational age 35 weeks. The amniotic fluid was clear. Apgar scores were 6,6, and 7.0. Owing to the severity of his condition, the neonate remained in the neonatal intensive care unit for 48 hours, after which he was transferred to the post-intensive care unit. Respiratory support was required for 12 hours. Enteral feeding was initiated on the first day of life, and parenteral nutrition was administered for 3 days. The pregnancy was physiologically normal, although persistent bacteriuria was identified on routine examination. A prenatal diagnosis of hydrocephalic syndrome was established.

The child exhibited signs of depressed nervous-reflex activity, altered consciousness, and moderate respiratory disorders. Neonatal reflexes were immature and selectively elicited. Spontaneous motor activity was reduced, with present muscle hypotonia; the child did not open his eyes during examination, but pupillary light reflex was preserved. The anterior fontanelle measured 0.5 by 0.5 cm, was non-tense and level with the skull bones, but with overriding cranial sutures. No seizures were observed. Visible mucous membranes and skin were clean, pale pink, without edema. Spontaneous breathing was effective without oxygen dependence. Auscultation revealed weakened vesicular breath sounds with conductive rales over both lung fields. Percussion elicited a boxy tone with areas of dullness over the posteroinferior lung regions. Haemodynamics in the post-intensive care unit were relatively stable, compensated by volemia. Capillary refill time was 3 seconds. Heart sounds were weakened but rhythmic, with a systolic murmur audible over the cardiac apex. The abdomen was slightly distended, symmetrical, and accessible to deep palpation. Peristalsis was depressed; liver was palpable 1 cm below the costal margin; spleen was not enlarged. No gastric stasis was present. Enteral feeding was administered via nasogastric tube with 33 ml of age-appropriate milk formula every 3 hours. Diuresis was adequate; bowel movements were normal. Table 1 presents data from laboratory and instrumental examinations of the newborn.

Table 1

Data from laboratory studies and instrumental examinations

Complete blood count	RBC – 4.01x10¹²/L, Hb – 138 g/L, Color index (CI) – 1.03, Tr. – 355x10⁹/L, WBC – 26.2x10⁹/L, Eosinophils – 2%, Band neutrophils – 0%, Segmented neutrophils – 48%, Lymphocytes – 37%, Monocytes – 13%, Ht – 38.7%.
General urine analysis	Within normal limits
Biochemical blood test	Total bilirubin – 149.5 μmol/L; Direct bilirubin – 9.9 μmol/L; Indirect bilirubin – 139.6 μmol/L; Total protein – 40 g/L; Albumin – 29 g/L; ALT – 9 U/L; AST – 36 U/L; Creatinine – 43.6 μmol/L; Urea – 1.9 mmol/L
Acid-alkaline balance	pH – 7.34; pO_2 – 32 mm Hg; pCO_2 – 28.6 mm Hg
Cerebrospinal fluid analysis	Volume – 2 mL; Color – light yellow; Turbidity – moderate; Protein – 2.2 g/L; Glucose – 0.8 mmol/L; Cell count – 180 cells/μL (microscopy not performed)
Neurosonography (NSG) – Day 3 Neurosonography (NSG) – Day 10	Signs of hypoxic-ischaemic CNS damage; multiple organ dysfunction with grade II ventriculodilation (dilation of the cerebral ventricles); hyperechoic content in ventricles III-IV with formation of of platelet masses.
Ultrasound examination of the abdominal cavity, kidneys, lungs	No pathological changes detected
Echocardiography	PFO (Patent Foramen Ovale), VSD (Ventricular Septal Defect)

There is leukocytosis in the blood and pronounced protein-cell dissociation in the cerebrospinal fluid, indicating a risk of hydrocephalus development.

The results of the bacteriological examination of the child at the perinatal regional centre are presented in Table 2.

Table 2

Data from bacteriological examination of the oropharyngeal and gastric mucosa, and blood (probe).

Bacteriological blood culture	Listeria monocytogenes sensitive to ampicillin, penicillin G, gentamicin, erythromycin, trimethoprim/sulfamethoxazole, and meropenem was isolated.
Bacteriological culture from a throat swab	Klebsiella pneumoniae (resistant only to ampicillin) and Streptococcus mitis (resistant only to erythromycin) were isolated.
Gastric aspirate culture (obtained via nasogastric tube)	Polyresistant Staphylococcus epidermidis, Klebsiella pneumoniae, and Staphylococcus aureus (MRSA) were isolated.

Histological examination of the placenta revealed purulent chorioamnionitis, deciduitis, subdecidual intervillous inflammation, vascular-stromal funiculitis, compensatory angiomatosis, and perivascular haemorrhages in the umbilical cord, indicating intrauterine hypoxia and pronounced inflammatory changes in the placenta of both fetuses.

Based on clinical, laboratory, and instrumental examination methods, bacterial meningitis was diagnosed. Treatment with intravenous immunoglobulin, ampicillin, and gentamicin was initiated; upon receipt of bacteriological results, therapy was switched to meropenem with colomycin and fluconazole (due to inadequate penetration of ampicillin across the blood-brain barrier).

Upon admission to the Khmelnytsky City Children's Hospital of the Khmelnytsky City Council, the child's condition was assessed as serious. The patient was hospitalized in the neonatal pathology department, receiving enteral nutrition via tube with a combination of formula and breast milk (40 ml of adapted milk formula). Examination revealed reduced spontaneous motor activity, decreased muscle tone, and exhausted reflex activity. Vesicular breath sounds were diminished bilaterally**.** The child was partially oxygen-dependent (SpO_2 92% on room air).

NSG *on admission* (11th day of life) – the pattern of gyri and fissures was smoothed; the subarachnoid space and interhemispheric fissure were not enlarged; lateral ventricles measured 12.5 mm on the left and 12 mm on the right; at the level of the thalami – 21/21 mm. Single strands were present in the ventricular lumen, and punctate echogenic inclusions were observed in the lumen of the insula and basal ganglia. Gastric index was 0.46%. Signs of Grade II-III ventriculodilation (obstruction at the level of the interventricular foramina).

Repeated cerebrospinal fluid examination: cytosis increased to 560 cells/μL, with lymphocytes accounting for 59%, neutrophils 37%, monocytes 2%, and macrophages 2%.

Clinical diagnosis: bacterial sepsis (P36.8); neonatal listeriosis, meningitis caused by *Listeria monocytogenes* (P37.2); respiratory distress syndrome, neonatal apnea (P22.8); hypoxic-ischaemic central nervous system damage, neonatal cerebral depression (P91.4); persistent fetal circulation syndrome: persistent fetal circulation (P29.3); ventricular septal defect (Q25.0); anaemia of prematurity, grade II, of mixed etiology (P61.2); premature infant at 35 weeks of gestation (P07.32); infant with low birth weight (2400 g), from twins (P07.13).

The child continued to receive meropenem and was also administered linezolid for 20 days.

NSG dynamics: gastric index 0.36%; dense structures in the ventricles resolved; signs of grade II ventriculodilation persisted; ventricular walls were thickened.

Repeated cerebrospinal fluid analysis showed positive dynamics: cytosis 56 cells/μL, 84% lymphocytes (protein was not examined). Complete blood count: leukocytosis decreased but persists (17.6×10^9/L), with lymphocyte predominance (58%); thrombocytosis (764×10^9/L).

Cerebrospinal fluid examination on the 18th day after admission: cytosis 5 cells/μL. No microbial growth was detected in the cerebrospinal fluid or blood.

Women whose children are diagnosed with neonatal listeriosis are prescribed a course of antibiotic therapy with ampicillin or doxycycline. As the child was breastfed, the mother was prescribed ampicillin. The father, who was in contact with the mother and children and cared for the healthy breastfed twin, also received a course of antibiotic therapy. The second twin did not develop the disease.

The child was discharged home on the 19th day after admission to the Communal Enterprise «Khmelnytskyi City Children's Hospital» of the Khmelnytskyi City Council (30th day of life) in satisfactory condition, with a body weight of 3370 g (+960 g), but with manifestations of grade II hydrocephalic syndrome. Follow-up monitoring revealed progression of hydrocephalic syndrome, which became an indication for ventricular shunting at the age of 11 months.

Conclusions

1. In neonates with suspected sepsis or neuroinfection, bacteriological investigations of blood, cerebrospinal fluid, and other specimens should include testing for *Listeria monocytogenes*, given its intrinsic resistance to cephalosporins.

2. Listerial meningitis is frequently associated with pronounced protein-cell dissociation in cerebrospinal fluid (CSF), which predisposes to hydrocephalus;; therefore, timely diagnosis and rational antimicrobial therapy are critical.

3. Neonatal listeriosis is associated with high mortality rates; however, modern diagnostic and therapeutic approaches, including combined antibiotic therapy and intravenous immunoglobulin administration, may improve outcomes. Hydrocephalic syndrome developed in, and its progression could not be prevented.

4. Clinical management should include treatment of both the infant and the mother, who often serves as an infection carrier, to prevent reinfection. Although listeriosis may confer long-term immunity, this does not apply to neonates, particularly preterm infants with physiological immunodeficiency.

Conflict of Interest. The authors declare no conflict of interest and no financial involvement in the preparation of this article.

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ЛІСТЕРІОЗНА ІНФЕКЦІЯ У ВАГІТНИХ ЖІНОК ТА ДІТЕЙ (ОГЛЯД ЛІТЕРАТУРИ ТА ВЛАСНІ СПОСТЕРЕЖЕННЯ)

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Резюме.

Лістеріозна інфекція відноситься до групи особливо небезпечних інфекційних хвороб. Захворювання вражає переважно людей зі зниженим імунітетом, у тому числі немовлят та вагітних жінок, в яких може проявитись важким ураженням центральної нервової системи з розвитком менінгіту, менінгоенцефаліту, абсцесу мозку і закінчитись одужанням, але з важкими резидуальними наслідками зі сторони нервової системи і навіть летальним кінцем.

Мета – висвітлити актуальність лістеріозної інфекції, її клінічні симптоми та продемонструвати сучасні методи діагностики і лікування лістеріозу, виходячи з власного досвіду та даних літературних джерел.

Матеріали та методи. Проведено аналіз літературних джерел, присвячених лістеріозній інфекції у вагітних жінок і дітей, а також результатів власних спостережень лістеріозу у новонародженої дитини з вродженим лістеріозом, інфікування якої відбулося від мами, яка перенесла дану інфекцію в латентній формі. При бактеріологічному дослідженні з ліквору дитини виділено збудник лістеріозу – Listersa monocytogenes. Захворювання протікало з помірно вираженим інтоксикаційним синдромом та з розвитком менінгоенцефаліту, який ускладнився гідроцефальним синдромом. Останній було діагностовано ще пренатально, що свідчить про внутрішньоутробне інфікування дитини. Лістерія була резистентною до комбінованої антибактеріальної терапії з ампіциліну та гентаміцину.

Висновки. Клінічні прояви лістеріозної інфекції поліморфні – від стертих до важких, особливо при ураженні нервової системи або розвитку сепсису. Переважно маніфестація лістеріозу з ураженням ЦНС має місце в ослаблених осіб, до яких слід відносити також новонароджених дітей та вагітних жінок. При будь-яких проявах ураження ЦНС в даної когорти осіб, їх слід обстежувати, в тому числі, на лістеріозну інфекцію.

Ключові слова: діти; вагітні жінки; новонароджені; лістеріоз на інфекція; менінгіт; сепсис; діагностика; лікування.

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