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REVIEW

Listeriosis during pregnancy

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Abstract
Purpose Listeriosis is a rare foodborne illness caused by *Listeria monocytogenes*. It can be transmitted by consuming contaminated ready-to-eat food, long shelf-life products, deli meats, and soft cheeses. *Listeria* has a predilection to affect immunocompromised patients, elderly people, pregnant women and neonates. In particular, pregnant women are at ~18 times greater risk of infection than general population due to specific pregnancy-related suppressed cell-mediated immunity and placental tropism of *L. monocytogenes*. The purpose of this review is to summarize the current knowledge regarding listeriosis during pregnancy.
Methods A literature search on Medline and Embase was done for articles about listeriosis during pregnancy. A detailed review of published data on epidemiology, pathogenesis, diagnosis, treatment and prevention of listeriosis during pregnancy was performed.
Results Listeriosis during pregnancy encompasses maternal, fetal and neonatal disease. Maternal listeriosis during pregnancy usually presents as a mild febrile illness. Fetal listeriosis has a high mortality rate of 25–35%, depending on the gestational age at the time of infection. Neonatal listeriosis may present as sepsis or meningitis with severe sequels and high case fatality rate of 20%. Adequate treatment of maternal listeriosis prevents and treats fetal disease and it is of imminence importance in the treatment of the neonates. Amoxicillin or ampicillin are the first line of treatment alone or in combination with gentamicin, followed by trimethoprim/sulfamethoxazole.
Conclusions Pregnancy-associated listeriosis should be considered as a cause of fever during pregnancy and appropriate treatment should be initiated preemptively. Prevention remains the best way to control listeriosis and should be reinforced among patients, health care professionals, and regulatory agencies.

Keywords Listeriosis · Pregnancy · Outcomes · Treatment

Introduction

Listeria monocytogenes is an intracellular, aerobic and facultative anaerobic, Gram-positive bacteria [1]. It can be spread by consumption of contaminated food and is responsible for cases and outbreaks of listeriosis [2, 3]. Pregnant women, neonates, elderly people, and immunocompromised persons are at higher risk of the infection [4]. Case fatality rate related to listeriosis is approximately 20% [5].

It has been reported that the annual incidence of listeriosis in the world is between 1 and 10 per million population [4]. In the USA, the incidence of *L. monocytogenes* infection is 2.4 cases per million population with mortality rate of 16.9% [6, 7], leading to more than 1520 listeriosis-related hospitalizations annually and resulting in up to 252 listeriosis-related deaths per year [8]. In Canada, there were 132 confirmed cases reported in 2014 [9]. In Europe, the incidence rates vary between 0.1 and 11.3 per million population, and around 20% of the cases are perinatal or involve neonates [10, 11]. During pregnancy, the incidence of listeriosis varies in different ethnic groups depending upon their food consummation habits, socioeconomic

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status, and place of residence [3, 4, 12] accounting for approximately 8–17% of the listeriosis cases [12–14].

The estimated incidence of pregnancy-related listeriosis is 3 cases per 100,000 births, ranging from 1 to 25 cases per 100,000 births (up to 35% of all *Listeria* infections) [6, 8, 12, 14, 15]. When the infection occurs in neonates, about half of them have no apparent immunocompromising condition [16].

Listeria monocytogenes has been categorized in 13 serotypes but only few serotypes (1/2a, 1/2b, and 4b) are involved in the majority of reported human listeriosis outbreaks and sporadic cases [10, 17, 18]. Genetically, it is a heterogeneous organism [19] with a relatively stable genome [20–22]. It has a low rate of variants acquisition in virulence genes evolutionary clustering into four phylogenetic lineages I, II, III, and IV [21, 23–25]. Epidemic clone (EC) classification system is based on genotyping of the *L. monocytogenes* strains involved in more than one outbreak using the multi-locus sequence typing (MLST) with grouping based on identical virulence type of six virulence locus sequences or whole-genome sequencing (WGS). The EC classification has ten types (from I–X), each serotype can belong to different EC originating from the same ancient clone (e.g., serotype 4b has three EC: EC I, EC II, and EC Ia) all involved in outbreaks and in majority of sporadic cases [23, 26–28]. Clonal complex (CC) system classifies *L. monocytogenes* regardless of its involvement in outbreaks into group of sequence types (ST) based on 7-gene MLST where groups differ no more than one allele from at least one other ST group [22, 24, 29]. Lineage-specific genotyping and grouping of *L. monocytogenes* isolates using core genome MLST is implicated as successful in both epidemiologically related and unrelated strains [26].

Pathogenesis of *L. monocytogenes*

L. monocytogenes can enter the host without disturbing the integrity of gastrointestinal tract, although symptoms of gastroenteritis may represent a concomitant infection that disrupts mucosal integrity and facilitates *L. monocytogenes* invasion [10]. The invasiveness of listeriosis is determined by the host immunity, virulence of the infecting strain, and the size of inoculum [2]. The infective dose related to clinical listeriosis is estimated to be 10^4–10^6 organisms/g of ingested product which might be lower in groups at risk [10]. After ingestion, the first site of *Listeria* invasion is small intestine. From there bacterium invades the mesenteric lymph nodes to disseminate to the spleen and the liver and from there to the brain or the placenta [30].

Internalization of *L. monocytogenes* into the epithelial cells is through phagocytosis, being mediated by the interaction of bacterial surface protein internalin (Inl, 25 different groups) and the epithelial transmembrane protein, E-cadherin that promotes formation of phagosomes [31]. This internalization marks the intracellular cycle of *L. monocytogenes* where transcriptional factor PrfA is controlling expression of several virulent factors that *Listeria* uses to escape the phagosomes through the action of listeriolysin O (*Listeria*-specific virulence factor), phospholipid A (PlcA), phospholipid B (PlcB), and metalloproteinase, finally entering the cytoplasm where the proliferation occurs [31, 32]. The organism spreads from cell to cell by production of pseudopodia-like structures by actin tail formation, orchestrated by ActA, which extrudes from the host cells and is phagocytosed by adjacent cells, so that the intracellular cycle of *Listeria* continues [31]. This unique intracellular cycle of *Listeria* make antibodies, complement and neutrophils to have little or no effect in host protection [33].

Listeria activates T-cell-mediated immunity which, under the influence of cytokines (IL-18), attracts macrophages that produce inflammatory granulomata where bacteria are destroyed. Memory T-cell provides an acquired resistance to *Listeria* infection (this explains why listeriosis is linked with malignancy, immunosuppressive therapy, AIDS, pregnancy, and neonates) [4].

Maternal listeriosis

Pregnant women have 18 times higher risk of contracting listeriosis after ingestion of contaminated food compared to general population [3]. This is partly attributed to pregnancy-related maternal T-cell suppression and their low number in the decidua due to epigenetic silencing of chemokines, in addition to the placental tropism (ActA and InlP virulence factors) and the protected intracellular life cycle of *Listeria* which enables invasion of the bacterium beyond the gastrointestinal system [3, 34–41]. Decidua is the initial site for placental colonization of *L. monocytogenes* [42], after passing beyond the restrictive barrier of invasive extravillous trophoblast (EVT) that can inhibit the vacuolar escape of the bacterium [43]. Placenta has specific immunologic properties partially regulated by immunosuppressive human leukocyte antigen G (HLA-G) (expressed on EVT) silencing the cytolytic functions of decidual natural killer cells, macrophages, and T-cells [44]. That makes placenta a protective environment for growth of the bacterium and formation of microabscesses, from where it can be chronically discharged into maternal and fetal bloodstream [44–47]. In the early stages, placental infection is dependent on the inoculum size and virulence of circulating bacteria that will resist the protective barrier of EVT and syncytiotrophoblast [43, 48–50]. The

inflammatory reaction is usually based between the chorionic villi in the intervillous space causing intervillositis which is characteristic of *Listeria* infections [51].

Maternal infection, clinically, is usually silent, while fetal or neonatal infection can cause severe outcomes including miscarriage, stillbirth, neonatal sepsis, and meningitis [52–54].

Listeriosis can occur any time during pregnancy but it is most often recognized in the third trimester of pregnancy (from 28 weeks of pregnancy) [12, 55].

Diagnosis of listeriosis during pregnancy is challenging because of its non-specific clinical presentation [56]. Approximately, one-third of infected pregnant women are asymptomatic [13, 52, 57]. The rest usually presented with non-specific, mild flu-like symptoms such as fever, backache, headache, vomiting/diarrhea, muscle pains, or sore throat [13, 14, 56, 58, 59]. Rarely, it can invade maternal brain leading to meningoencephalitis [18].

When *Listeria* escapes the first immunologic barrier at the maternal gastric and intestinal lymph nodes, it causes maternal bacteremia [31]. *Listerial* bacteremia can cause chorioamnionitis and can invade the placenta thus becoming a source for cyclic reinfections [46]. Transmission of the bacteria to the fetus can cause intrauterine fetal demise, preterm labor, and prematurity [12, 13, 60]. Maternal mortality is rare and often reported with other coexisting medical conditions of the mother [12, 15]. Overall mortality rate after neonatal and fetal listeriosis including abortion and stillbirth increases to more than 50% [10, 18].

Fetal listeriosis

Symptoms of maternal listeriosis usually occur 1–14 days prior to the appearance of fetal distress, resulting in fetal disseminated disease with high mortality rate of 27–33% [15, 61, 62]. Gestational age at the onset of infection is an important predictor for survival of the fetus [14]. Fetal infections in the first trimester of pregnancy have poor prognosis with approximately 65% risk for miscarriage [15]. On the other hand, the fetuses who have survived the infection in the first trimester (∼35% of cases) or if the infection occurs later in the second or third trimester of pregnancy are at 26% of risk for still birth, miscarriage, or fetal death [15]. In about 10–15% of the cases, listeriosis induces premature labor [12, 14]. In a study involving 166 cases of fetal listeriosis, fetal survival was 0, 29.2, and 95.3% in the first, second, and third trimesters, respectively [12]. A recent prospective cohort study analyzing 107 cases with pregnancy-related listeriosis, the transmission of the infection to the fetus was 96%, and major fetal or neonatal complications have been seen in 83% of infected mothers with a cutoff of 29 gestational weeks for increased risk for fetal loss [13]. The overall perinatal mortality rate of listeriosis is reported to be around 50% [18].

Although there are reports on association between recurrent miscarriages and chronic or asymptomatic listeriosis, the causative link remains controversial as *L. monocytogenes* was not isolated from the reproductive tract of any of the women with previous miscarriages in those reports [56, 63].

Neonatal listeriosis

Neonatal illness due to listeriosis is often severe and may be fatal in contrast to maternal illness that tends to be mild. Vertical transmission of the bacterium is the source of neonatal infection in the most of the cases. *L. monocytogenes* can be transmitted from mother to fetus and consequently to the neonate by ingestion of infected amniotic fluid during intrauterine life, transplacentally from the maternal circulation, by ascending infection from vaginal colonization and rarely by horizontal infection after birth.

The incidence of neonatal listeriosis is approximately 8.6/100,000 of live births, with a high mortality rate (20–60%) and is one of the most common causes of neonatal meningitis [15, 18, 64].

Neonatal cases of listeriosis are classified into early onset (day 1–6) and late onset (day 7–28) depending on the time of development of symptoms after birth [14, 65]. Early onset cases are usually preceded by mild maternal symptoms [66]. Clinical features of neonatal listeriosis include septicemia (81–88%), respiratory distress or pneumonia (38%) and meningitis (24%) [18]. The formation and dissemination of abscesses and granulomas in multiple organs known as granulomatosis infantiseptica is pathognomonic for neonatal listeriosis [61]. Mortality rate is high (∼20%) with severe neurological and developmental sequelae in 40% of the surviving neonates [67].

In the late onset type, a newborn develops symptoms approximately 1 week after birth, and it is typically seen in term neonates born to asymptomatic mothers [10, 18]. Horizontal transmission is an extremely rare mode of infection causing late-onset neonatal listeriosis which occurs during passage of neonate through birth canal or as a nosocomial infection from another early-onset cases (most common way of horizontal transmission) [14]. The neonate usually presents with septicemia (17–95%) and meningitis (67–93%), but the clinical features may be non-specific [66]. The mortality rate associated with late-onset disease is around 10% [3], and frequently seen severe complications include physical growth retardation, mental retardation, and blindness [18]. Neonatal listeriosis is one of the few congenital infections in which antibiotic therapy can improve outcome [10, 56].

Diagnosis

Early recognition and diagnosis of listeriosis in pregnancy is important as the treatment with antibiotics is directed toward improving neonatal outcome [10, 68]. The incubation period for listeriosis can be long; a median incubation period of invasive listeriosis is 8 days, ranging from 1 to 67 days [3]. For febrile gastrointestinal disease, the median incubation period was reported as 24 h, ranging from 6 h to 10 days [3, 69].

The diagnosis of listeriosis relies on culturing and detecting the growth of the microorganisms in body fluids [3]. The standard approach to establish a diagnosis of listeriosis during pregnancy is through a maternal blood culture [54], but also vaginal swabs, cerebrospinal liquor or placental tissue and placental smears can be employed [13]. In case of maternal listeriosis, an amniocentesis should be performed with subsequent Gram staining [70]. If the amniotic fluid is infected with *L. monocytogenes*, it is usually meconium stained with Gram-positive rods [3, 5, 13]. Rapid identification and characterization of *L. monocytogenes* is also possible, using serological testing for anti-listeriolysin, monoclonal antibody tests based on latex agglutination, enzyme immunoassay (EIA) and enzyme-linked immunosorbent assay (ELISA), as well as direct molecular genetic identification using specific primers based on pulse field gel electrophoresis, DNA hybridization, real-time PCR, or sequencing of the bacterial genome [10, 54, 71]. Stool cultures of *Listeria* can be used for diagnostic evaluation in pregnant women although not recommended due to relatively frequent intermittent fecal carriage of the bacterium and low sensitivity [54, 72–74], especially when *Listeria* exposure is suspected in pregnant women, their household and during *Listeria* outbreaks [72].

Treatment

Summary of treatment options for *Listeria* and their safety in pregnancy and breastfeeding is given in Table 1.

Preemptive treatment with active antimicrobials should be administered to prevent overt listeriosis in all cases with unexplained fever in pregnancy, and/or gastrointestinal symptoms with or without possible *L. monocytogenes* exposure, based on the insignificant clinical presentation of listeriosis during pregnancy and the imperative of the adequate and timely treatment for favorable maternal and fetal/neonatal outcomes [68, 72]. The treatment should be *L. monocytogenes*-specific when there is a suspicion of an exposure from contaminated food, during outbreaks and in all cases of microbiologically confirmed *L. monocytogenes* [54, 72].

Although there are no randomized controlled trials to assess type, efficacy, and duration of antibiotic treatment, and most data are based on reports of clinical experience [75], the current antimicrobial treatment and prevention of listeriosis consist of a β-lactam antibiotic as a first line, given in high doses for at least 14 days or until delivery, normally ampicillin (intravenously 6–12 g/day) or amoxicillin (orally 100 mg/kg/day) [68, 76]. The American College of Obstetricians and Gynecologists guidelines on management of pregnant women with presumptive exposure to *L. monocytogenes* recommend the above-mentioned treatment in symptomatic, febrile patients; however, the recommendation for the afebrile cases with mild symptoms and febrile without listeriosis symptoms is expectant management [54]. Ampicillin/amoxicillin can be given alone or in combination with an aminoglycoside, usually gentamicin 5 mg/kg/day for 3–5 days [54, 76–78]. This combination of antibiotics has synergistic bactericidal effect on *L. monocytogenes* in vitro; however, it has no documented intracellular efficacy in macrophages [79]. Clinical experience is also conflicting where smaller size retrospective studies show unfavorable effect of aminoglycosides [80, 81] and couple of recent studies, bigger in size, showing strong evidence of efficacy and supporting the use of gentamycin in combination with ampicillin as a first-line therapy of invasive listeriosis [13, 82]. Ampicillin has a long-standing fetal safety record [83–86]. Ampicillin is also acceptable for breastfeeding when the treatment is to be extended or initiated in the puerperium with relative infant dose (RID) of 0.2–0.5% (RID: an infant dose of drug via breast milk per body weight, expressed as % of maternal dose per body weight) [87–90].

Data available for fetal safety of gentamicin are limited, but there are no reports of increased risk for congenital malformations or eighth cranial nerve toxicity that is described with other aminoglycosides [91]. Gentamicin is poorly excreted in breast milk with RID of 2.1% thus making it acceptable for breastfeeding [89, 90, 92].

For those patients who are allergic to β-lactams, second-line agents are erythromycin (4 g/day intravenously for 14 days or until delivery), trimethoprim/sulfamethoxazole (80/400 mg four times/day for 14 days or until delivery), vancomycin, and the fluoroquinolones [77]. Resistance of clinical isolates of *L. monocytogenes* to these antibiotics is shown to be low [93].

Erythromycin has a favorable fetal safety profile and is a good option in cases of penicillin allergies [94–100]. The only drawback is the lower placental transfer of the drug requiring a usage of higher doses. It is compatible for breastfeeding as well (RID 1.4–1.7%) [89, 90].

Fetal safety data on trimethoprim/sulfamethoxazole suggest an increased risk for neural tube defects, cardiovascular malformations, and oral clefting after

Table 1 Treatment options for *Listeriosis* during pregnancy and their safety in pregnancy and breastfeeding

Antimicrobial agent	Treatment dose	Duration of treatment	Safety during pregnancy	Safety during breastfeeding (BF)
Ampicillin	First-line: 6–12 g/day IV	14 days or until delivery	Safe [83–86]	RID = 0.2–0.5% compatible with BF; observe for disturbances of neonates' GIT flora[a] [87–90]
Amoxicillin	First-line: 100 mg/kg/day PO	14 days or until delivery	Safe [83–86]	RID = 1% compatible with BF; observe for disturbances of neonates' GIT flora[a] [87–90]
Gentamicin	First-line: in combination with ampicillin/amoxicillin 1.5–2.5 mg/kg q8 h IV	3–5 days	Relatively safe [91] (limited data available, no reports of increased risk for congenital malformations or eight cranial nerve toxicity)	RID = 2.1% compatible with BF; observe for disturbances of neonates GIT flora[a], candidiasis or antibiotic associated colitis [89, 90, 92]
Erythromycin	Second-line: 4 g/day IV (alternative option in cases of penicillin allergies)	14 days or until delivery	Safe [94–100]	RID = 1.4–1.7% compatible with BF; observe for irritability and gastrointestinal tract (GIT) effects such vomiting, diarrhea, candidiasis, or rush [89, 90]
Trimethoprim/sulfamethoxazole [TMP (80 mg)/SMX 400 mg)]	Second-line: 200–320 mg (3–5 mg/kg q6 h IV) of TMP component	14 days or until delivery	Increased risk for neural tube defect, cardiovascular defects and oral clefting in 1st trimester; Supplement Folic acid 1 mg/day [101]	RID = 3.9–9% compatible with BF of full-term babies. Caution in premature and infants with hyperbilirubinemia; observe for disturbances of GIT flora[a] and candidiasis [89, 90, 103]
Vancomycin[b]	Third-line: limited efficacy 1 g, q8 h, IV	7–14 days	Relatively safe (limited safety information for the fetus) [62, 75, 104]	RID = 6.7% compatible with BF [89, 90]
Quinolones[b] (ciprofloxacin)	Fourth-line: limited efficacy (200–400 mg, q8–12 h, IV)	7–14 days	Relatively safe in 1st trimester (cartilage toxicity in animal studies) [105]	RID = 2.1–6.34% compatible with BF only for short term use [89, 90, 105, 106]

PO per os, *IV* intravenous, *IM* intramuscular, *TMP/SMX* trimethoprim/sulfamethoxazole, *RID* relative infant dose, *BF* breastfeeding, *GIT* gastrointestinal tract, *NT* neural tube defect

[a] Disturbances of GIT flora = diarrhea

[b] Only an option after exhausting other treatment alternatives

trimethoprim exposure, possibly due to its antifolate activity. Supplementation with 1 mg/day of folic acid is recommended when trimethoprim is used in the first trimester [101]. Because of potential neonatal toxicity of sulfamethoxazole and the risk for hemolytic anemia and kernicterus, it should be avoided in late third trimester [102]. This antibiotic is compatible with breastfeeding of full-term babies, with trimethoprim RID of 3.9–9% [89, 90]. It should be avoided in breastfeeding mothers whose infants are diagnosed with G6PD deficiency, premature born, and neonates with hyperbilirubinemia, and all infants should be observed for irritability or GIT effects such diarrhea and candidiasis [103].

Data on efficacy of vancomycin in treating listeriosis are based on clinical reports with limited safety information for the fetus [62, 75, 104]. This drug passes into breast milk in small amounts (RID 6.7%) and has very poor oral bioavailability making it unlikely to cause any adverse effects in breastfed babies [89, 90].

Quinolones are relatively safe in the first trimester of pregnancy, with animal studies showing cartilage toxicity. Their efficacy is also questionable and should be used in cases where other alternatives are exhausted [75]. It can be used in breastfeeding mothers only for short term because of theoretical interference with baby's joint development [105]. Calcium in milk reduces its resorption thus minimizing infants' exposure (RID 2.1–6.34%) [89]. Avoiding breastfeeding for 3–4 h after a dose can decrease the exposure of the infant to ciprofloxacin in breast milk [89, 90]. The infants should be monitored for possible adverse effects on the gastrointestinal flora, such as diarrhea or candidiasis [105, 106].

The length of treatment and the choices of antibiotics depend on the clinical manifestation and the findings of the active fetal surveillance [54], but should not be less than 14 days. The efficacy of the antimicrobial treatment will still depend on the amount that would penetrate the cell, distribute within the cells, and remain stable in the intracellular environment [54, 66]. *Listeria monocytogenes* is resistant to cephalosporins, clindamycin, and chloramphenicol [54].

Prevention

There is no vaccine against listeriosis. Prevention should be the utmost priority in control of listeriosis as the bacterium is widely distributed in the soil, mud, water, and decaying organic material [16]. Raising awareness of the risks of listeriosis among pregnant women, health professionals, and governmental health authorities should be all part of a proactive prevention system with strict regulations of safety in production and distribution of food and with appropriate rapid alert messaging system with information concerning *Listeria*.

The cornerstone in prevention is proper food preparation and handling; proper food storage and general food safety, hygiene and sanitation for both producers and consumers [107, 108]. Good sanitary and hygiene habits with improved standards and surveillance for *Listeria* have reduced the prevalence of contaminated foods at grocery stores [64].

The following are the most common foods at risk of *L. monocytogenes*:

- Soft cheeses made from unpasteurized milk: feta, Brie, Camembert, blue-veined cheeses, and Mexican-style cheeses such as queso fresco, queso blanco, and panela that do not state they are pasteurized. Hard cheeses (cheddar), semi-soft cheeses (mozzarella), and pasteurized processed cheese products (slices, spreads, cream cheese, and cottage cheese) can be safely consumed.
- Hot dogs, cold cuts, or deli meats if consumed without reheating to steaming (or 160 °F).
- Refrigerated pates or meat spreads (especially with long shelf life).
- Refrigerated smoked seafood (salmon, trout, tuna, cod, and whitefish) if consumed without reheating to steaming. Canned fish or shelf stable smoked seafood are safe to consume.
- Raw and unpasteurised milk or any product made from unpasteurised milk.
- Raw and unwashed fruit and vegetables (e.g., leafy salad, and cantaloupe).
- Refrigerated perishable food not used within 2–3 days.

Prevention tasks for health care professionals: Hygiene measures and strict adherence to disinfection polices should be practised in hospitals and units caring for infected patients to prevent nosocomial infection [16]. Special attention in preventative measures is required in the neonatal and delivery units where an outbreak of nosocomial listeriosis was linked to contaminated mineral oil [109].

Patient education through *prenatal counseling* [110] and active involvement of practitioners in raising the awareness of the risks of foodborne diseases are necessary in proper prevention of listeriosis in pregnancy [111]. Heating or cooking food is the best way to inactivate foodborne pathogens. Therefore, pregnant women should ensure that their food is obtained from reputable establishments; stored, handled, and cooked properly; and consumed within a couple of days of purchasing [112].

The following advises should be given to pregnant women and their household to reduce the chances of contracting listeriosis:

- Avoid consumption of food at risk for *Listeria* contamination.
- Practice safe food handling:
  - Wash all fruits and vegetables even if not consumed at the moment and to be kept refrigerated.
  - Separate uncooked meats from cooked meats and vegetables.
  - Avoid cross-contamination between uncooked food and raw food (including hot dog juices).
  - Wash thoroughly all preparation surfaces, cutting boards, knifes, and hands after contact with uncooked foods.
- Practice proper food storage:
  - Clean refrigerator regularly and often.
  - Keep your refrigerator thermometer at 40 °F (4 °C) or below.
  - Consume ready-to-eat products as soon as possible and always read labels and follow recommended storage conditions.
  - Refrigerate and freeze food promptly (freeze leftovers if not consumed within 1–2 days).
- Thoroughly cook raw meats and other foods:
  - Recommended internal temperatures (rare cooked meat is not recommended): chicken—165–180 °F, egg dishes—160 °F, ground meat—160–165 °F, beef—160–170 °F, pork—160–170 °F, ham (raw)—160 °F, ham (precooked)—140 °F.

Prevention tasks for health authorities: Health and food control authorities have major role in prevention and control of foodborne diseases. After several large outbreaks, the standards are set high for food processing industry and safety

surveillance has been raised for prevention of listeriosis, including reduction of *Listeria* contamination to zero of all ready-to-eat food (such as processed deli meats; pasteurized soft cheeses and pates) (*HACCP-Hazard Analysis and Critical Control Points, ISO standards and zero tolerance for Listeria, Public Health Agency of Canada, US Food and Drug Administration, 2003*) [64]. European standards for *L. monocytogenes* tolerance depend on the type of food (able/unable to support *Listeria* growth with <100 cfu/g food) and intended consumer population (zero tolerance for food for infants) [113]. Rapid alert system and surveillance has prompted numerous voluntary recalls of products at risk regardless of the huge financial losses for the industry, marking a substantial increase and clear evidence of improved awareness of public health risks of listeriosis [16].

Conclusion

Listeriosis during pregnancy is a foodborne disease, with non-specific or absent symptoms of maternal infection, which is vertically transmitted to the fetus and neonate posing a high risk of adverse pregnancy outcome: miscarriage, stillbirth, fetal death, premature labor, or neonatal disease with serious sequels. After establishing the diagnosis, listeriosis in pregnancy should be treated with high doses of intravenous ampicillin for 14 days or until delivery as a first-line and trimethoprim/sulfamethoxazole or erythromycin as a second-line antibiotic. Unexplained flu-like symptoms in pregnancy should be carefully examined with listeriosis taken into consideration together with initiation of appropriate listeriosis directed empiric therapy. Continuing education of pregnant women for proper food selection and adopting the proper hygienic conditions in food preparation should be emphasized.

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Compliance with ethical standards

Conflict of interest The author Mitko Madjunkov declares that he has no conflict of interest. The author Shahnaz Chaudhry declares that she has no conflict of interest. The author Shinya Ito declares that he has no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

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