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## 26.1 Introduction

Listeriosis is a sparse foodborne illness caused by a gram-positive bacterium called *Listeria monocytogenes*. The disease was first identified in laboratory animals as early as 1926. A decade later, Burn identified it as a cause of perinatal infections in humans in 1936 [1]. The name “Listeria” was coined under the name of the Father of antiseptics, Lord Joseph Lister, in 1940 [2]. The bacteria are ubiquitous in nature. It usually infects the population that is at the extremes of the age, i.e., newborns and elderly, immunosuppressed patients, pregnant women and sometimes can affect previously healthy individuals. In comparison to the general population, listeriosis is seen 13–20 times more commonly in pregnant women, and infections in pregnant women contribute to 16–27% of all listeria infections [3–5]. Most of the times, the infection is seen in otherwise healthy pregnant women with no prior risk elements [6].

## 26.2 Epidemiology

*L. monocytogenes* is known to cause foodborne illness reported in sporadic cases, outbreaks, and food recalls all over the world. It became one of

the major pathogens causing foodborne illnesses in 1980s which actually resulted in the execution and further improvisation of surveillance strategies in the Western world [7]. Overall, due to the extensive reporting, microbiological surveillance through reference laboratories and systematic food quality control procedures, there was significant decrease in the cases, especially in pregnancy. However, recently new changes were observed in the overall epidemiology of the disease.

A recent large outbreak (2017–2018) caused by this bacterium in South Africa (the largest outbreak worldwide) had an effect on a wider population over a wider geographical area as compared to that caused by the usual local foodborne illness. There were more than 1000 laboratory-confirmed cases with more than 200 case fatalities with 42% infected [8]. The source of the illness was identified as “polony” that is an instant processed meat. The strain of listeria associated with the outbreak was also recognized in the processing environment of the manufacturer of this processed meat. The outbreak had a widespread impact affecting more than 15 African countries [9]. The factor responsible for such a large distribution of the disease was mainly because of the widespread distribution of the processed food product across national as well as international borders.

The largest outbreak in the USA was reported in 2011, which affected multiple states with alto-

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gether 147 people being affected. This outbreak has accounted for the largest number of deaths ( $n = 33$ ) as seen by any foodborne illness in the USA in greater than the last 80 years. The food product identified was the whole cantaloupe melons from a single farm [10].

Over the last few years, there has been a recent change in the foods/food products identified as sources of *Listeria* outbreaks, and there has been involvement across the borders, national as well as international. This can be attributed to the free exchange of goods, including food (processed as well as unprocessed), its constituents, and the processing supplies across states as well as countries. The main factors responsible for these mass food outbreaks are the microbiological characteristics of this pathogen, its ability to survive, grow, and then multiply in different environments [11].

The most common vehicles of transmission of *Listeria* infection are dairy associated products like unpasteurized milk, soft cheese (like queso fresco, feta and camembert), cooked and processed instant meats and sausages, refrigerated foods like seafood, pates, and spreads. The recent outbreaks have challenged our old time understanding of the foods associated with this infection, and foods like raw sprouts and whole cantaloupe melons have been added to the list now [12–14].

*Listeria* infection is considered as a notifiable disease in some countries. The Center for Disease Control and Prevention estimates that approximately 1600 cases occur annually and considers *L. monocytogenes* as the third leading cause of death from foodborne illness in the USA, causing about 260 deaths [15]. On the European front, 28 European countries documented 2206 confirmed human cases of listeriosis and 270 deaths in 2015. This large number accounted for the highest yearly deaths since 2008, making this disease the most severe human zoonotic disease under European Union (EU) Surveillance [16].

### 26.2.1 Listeriosis in Pregnancy

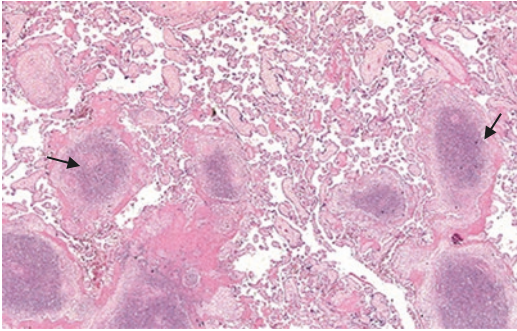
Listeriosis in pregnancy is associated with significant morbidity, including fetal loss and severe neonatal infection. The incidence of listeriosis in pregnant women is reported as 11% in Italy, 12% in UK, 16.9% in USA, and 17.7% in France [17]. Mylonakis et al. [6], in their case series of 11 pregnant women affected with listeriosis, along with a review of 222 cases from the literature, observed that around 1 out of 5 pregnancies resulted in spontaneous miscarriage or still birth, and around 66% of the infants who survived had neonatal listeriosis [6]. The reported case fatality rate from *Listeria* is around 60% for fetal or early neonatal infection vs. around 35% for late-onset neonatal infection [15].

Listeriosis during pregnancy is seen more commonly in certain ethnic groups. It is seen more frequently in Hispanic women as compared to non-Hispanic women in USA [6, 18], in women with African origin as compared to French origin [19] and in non-English women compared to those born in the UK [20].

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## 26.3 Microbiology and Pathogenesis

The only species of *Listeria* that is commonly associated with infection in humans is *Listeria monocytogenes*. Rare cases of infection with *Listeria ivanovii* and *Listeria grayi* have also been reported [21, 22]. *L. monocytogenes* is an aerobic and facultative anaerobic, intracellular gram-positive bacillus that is beta hemolytic with characteristic tumbling motility, which can be visualized on light microscopy [23, 24]. On gram staining, *Listeria* can be mistaken with other gram positive bacilli like diplococci, diphtheroid, enterococci or *H influenzae* [23]. Because of its intracellular nature, gram staining is not a preferred method for diagnosing *Listeria*. It is ubiquitous and can survive through wide temperatures



**Fig. 26.1** *Listeria* placentitis: Histopathological image of placenta showing microabscesses with neutrophilic infiltration

varying from 4 to 37 °C. At room temperature, it is highly motile and can replicate actively in diverse medium: soil, water, vegetable decaying matter and animal feed, making them its primary habitat. It can also grow well in low temperatures, allowing it to survive and grow in refrigerated food like deli meats and soft cheese.

*Listeria* grows easily in most of the commonly used culture media like broth or blood agar. It usually does not need enrichment media when specimen is obtained from aseptic sites. However, if specimen is obtained from the vagina or is a stool culture, selective media are required to inhibit the growth of other commensals inhabiting such sites [25, 26]. On histopathology of infected placental specimens' micro abscesses are found (Fig. 26.1). The mode of transmission is the consumption of foods contaminated with the organism.

### 26.3.1 Genomic Characterization of Strains of *Listeria Monocytogenes*

Isolates of *L. monocytogenes* have been grouped into lineages, PCR serogroups, multilocus sequence typing (MLST), clonal complexes (CCs), and core genome MLST (cgMLST) sub lineages and types (CTs) [27, 28]. Overall, 13 serotypes are recognized in literature. A difference in the virulence of these lineages and sub-

types have been noted. In particular, hypervirulent MLST clones with high clinical frequency have been identified, such as CC1, CC2, CC4, and CC6 (belonging to lineage 1 and serotype 4b) [29]. Maury et al. [29] reported that these particular strains were seen more commonly in maternal-neonatal *Listeria* infection isolates in France, were also more frequently isolated from milk products [29]. The most common association was seen with CC4 type, 20% of these were obtained from maternal-neonatal infections. On the contrary, CC9 and CC121 (lineage 2) are the most common isolates obtained from food and are rarely seen in maternal-neonatal infections [30]. This is seen because of the difference in the virulence observed in the different groups and their adaptability to the mammalian gut. Of note, CC1, CC4, and CC6 adapt and replicate well and thereby are hypervirulent in comparison to isolates CC9 and CC121 that are hypovirulent as seen in the mouse model of *Listeria* infection. It is seen that these strains are hypovirulent since they express a short and non-functional InlA [29, 30]. The CC4 strain also exhibits placental tropism when used for intravenous inoculation rather than the usual strains (CC7 and CC9) used for reference in experiments [30].

### 26.3.2 Pathophysiology

The incubation period of *Listeria* infection varies between 24 h and 70 days, with a median of 21 days. The infection results after consumption of food contaminated with the bacteria. The bacteria penetrates through the enteric mucosa into the systemic circulation. Both innate and acquired immune responses help in combating this infection. The immune system responsible for protecting against *Listeria* is cell-mediated immunity, where the circulating T-cell lymphokines activate the local macrophages leading to the removal of the bacteria from the circulation [31–33]. Interleukin (IL)-18 appears to play a role in protection against *Listeria* by enhancing bacterial clearance, even in the absence of interferon

(IFN)-gamma, and by stimulating macrophages to secrete tumor necrosis factor (TNF) and nitric oxide [32]. On the other hand, factors that impair macrophage survival or function are associated with increased susceptibility to listeria infection. Pregnancy is associated with decreased cell-mediated immunity, thereby providing home to this virulent bacterium. The pregnant uterus gets inoculated by the circulating listeria leading to placental involvement. Once infected, the ability of the bacterium to survive intracellularly makes the placenta a perfect reservoir resulting in active multiplication and increasing overall active bacterial load.

### 26.3.3 Placental Tropism

It has been seen that maternal-neonatal (MN) Listeriosis with *L. monocytogenes* does not correlate well with any immunosuppressed state apart from the pregnancy itself. The clinical manifestation of listeriosis in pregnant women is usually mild with no mortality from listeria itself but has high rate of complications and mortality in fetus and neonates. This may indicate a high affinity of listeria for placental tissue [19].

Experimental studies using in vitro, ex vivo, and in vivo models have helped in understanding the pathophysiology of placental infection of this bacterium. Human placenta is formed with cytotrophoblasts that are epithelial cells that join together and form the syncytiotrophoblast (SYN). This SYN layer comes in straight contact with maternal blood, and this is called hemochorial placentation. Studies using human placental explants of the first and third trimester have shown that *L. monocytogenes* invades the extravillous trophoblasts (EVT), [34] and syncytiotrophoblasts (SYN) [35] in these explants. Along with that, immunohistochemical studies have shown that it is seen in SYN of placental specimens acquired from women infected with listeriosis.

Multiple studies have documented that listeriosis is most often reported in late second and third trimesters. These studies support the fact

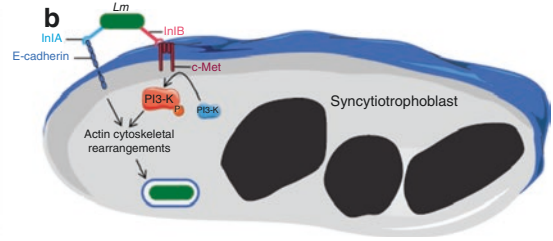
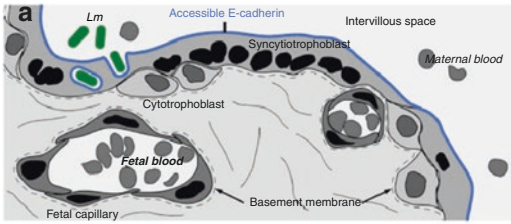
that this organism's mode of entry to the placenta is the SYN [19].

The more common incidence of listeriosis in the late second or third trimester can be explained by many factors.

The most important explanation of this association is that the human placenta becomes hemochorial only in the second trimester [36]. Also, there could be a low-grade infection due to the long incubation period of *L. monocytogenes* in the placenta, which actually becomes the home for this bacterium and further causes multiplication and reinfection [37]. Another hypothesis could be the increase in the percentage of cardiac output to the uterine blood flow, which increases as the pregnancy advances, thereby increasing the chances of seeding into the placenta. Along with that, there is an increase in the critical immune tolerance mechanisms at the maternal-fetal interface in advanced gestation. Listeriosis is also more often seen to affect multiple fetuses (with increased physiological burden), thereby suggesting that the increased physiological burden of advanced gestation can also be a possible explanation [38].

Contrary to these above explanations, one should remember that there might be an underreporting of listeria in early pregnancy losses since the products of conception are not often sent for culture and histopathology, resulting in underreporting. Also, as in early pregnancy, placenta directly invades the decidua, i.e., the uterine wall, making EVT as the portal of entry for *L. monocytogenes* during that period.

The entry of this bacterium into the host cells is species specific. In vivo studies have reported InlA and InlB are the two proteins of *L. monocytogenes*, which are crucial in invasion of epithelial cells used for culture. Their receptors are also host specific. InlA interacts with E-cadherin (hEcad, adherens junction protein human) while InlB interacts with hepatocyte growth factor receptor (c-Met in human, mouse, and gerbil) [39, 40]. As previously mentioned, the histological sections obtained from cases of MN listeriosis (human) depicted SYN and villous stroma [35]. Ecad and c-Met are both seen on the surface of SYNs and EVT (Fig. 26.2). InlA interacts with



**Fig. 26.2** Pathogenesis of Listeria infection of Placenta. InlA and InlB dependent breaching of the placental barrier by Listeria monocytogenes. (a) The placental barrier between the maternal blood and the fetus lies in an epithelium, the syncytiotrophoblast, which results from the fusion of underlying cytotrophoblast cells. Syncytiotrophoblast expresses E-cadherin, which is

accessible for bacteria in the maternal blood. (b) Listeria monocytogenes adheres to syncytiotrophoblast via InlA interaction with Ecad. InlB is required for L. monocytogenes entry by activating PI3-K via c-Met in the syncytiotrophoblast, leading to the actin cytoskeleton rearrangements needed for bacterial internalization

Ecad to adhere the bacteria on the host cell, and then InlB interacts with c-Met and thereby activates PI3-K (phosphoinositide 3 kinase) in the syncytiotrophoblast, which helps in its internalization. The potential role of InlA can be emphasized by the epidemiological fact that 100% of the clinical isolates associated with MN listeriosis demonstrate a non-truncated InLA in comparison to the isolates from the cases of bacteremia (93%) or those obtained from food (65%) [19, 30, 41].

To summarize, MN listeriosis is due to the organism’s specific placental tropism that is subsequent to the interaction of InlA and InlB with their respective species-specific receptors at the placental surface. Other pathogenic factors like Listeriolysin O (LLO), which is a pore-forming toxin that helps the organism to avoid its internalization vacuole and modifies host signaling pathways [42] and ActA (Actin assembly-inducing protein) that arbitrates actin polymerization that leads to the creation of comet cells which helps in movement of Listeria intercellularly [43]. Both these molecules play important role in their intracellular survival and multiplication.

**Table 26.1** Clinical manifestations of listeria in pregnancy and neonatal period

Pregnant women	Neonatal infections
<ul style="list-style-type: none"> <li>• Influenza-like symptoms: fever, malaise, headache</li> <li>• Gastrointestinal symptoms</li> <li>• Back pain</li> <li>• First trimester: Miscarriage</li> <li>• Second/third trimester: Preterm labor, Stillbirth, Meconium stained amniotic fluid, Neonatal infections</li> </ul>	<p><i>Congenital/Early onset infection (0–7 days):</i></p> <ul style="list-style-type: none"> <li>• Sepsis</li> <li>• Congenital pneumonia and respiratory distress</li> <li>• Skin rash</li> <li>• Neurological symptoms</li> <li>• Jaundice</li> <li>• Disseminated granulomatous lesions called granulomatous infantisepsis</li> <li>• Death (60%)</li> </ul> <p><i>Late-onset infection (8–28 days)</i></p> <ul style="list-style-type: none"> <li>• Meningitis</li> <li>• Sepsis</li> <li>• Death (20%)</li> </ul>

## 26.4 Clinical Manifestations (Table 26.1)

Severe maternal disease due to Listeria in pregnancy is rare, though it has been reported. Most of the women present with mild to minimal

symptoms or may be asymptomatic as well. In most case studies, fever has been reported as the most common symptom. It can affect all three trimesters in pregnancy equally, and the overall outcome depends upon the stage of pregnancy affected. Theoretically, co-morbidities affecting the cell-mediated immune system, for example, HIV, diabetes, and use of immunosuppressed agents, may predispose pregnant women to this infection, but most of the cases reported are seen in healthy pregnant women with no predisposing risk factors [6]. As mentioned above in the text, infections are more often seen in the late second or third trimester.

### 26.4.1 Maternal Manifestations

The estimated incidence of listeria in pregnancy is 3–4 per 100,000 cases. It usually presents with nonspecific flu-like illness with symptoms: fever, myalgia, headache, backache, vomiting, diarrhea, or sore throat, leading to delay in the diagnosis. Women with confirmed listeriosis are more likely to have fever (71% vs. 28%), flu-like symptoms (42% vs. 9%), preterm labor (100% vs. 1.7%), and premature contractions (57% vs. 5%) compared to suspected listeriosis [44]. Mortality is rare in pregnant women, but the outcomes in the fetus as well the newborn can be potentially grave.

### 26.4.2 Fetal Infection

Listeria is one of the infections that has shown a high transmissibility rate from the mother to the fetus in the intrauterine period. Close to 95% of the mother infected with listeria can transmit the infection to the fetus in the first 14 days of infection [45]. Compared to other common infections like TORCH, Listeria is not teratogenic with no associated fetal anomalies based on the current literature. It however carries significant morbidity and mortality compared to infection in the neonatal period, with multiple studies reporting a high rate of fetal losses in the early pregnancy: 65% compared to 26% in the second or third trimester [15]. In a study looking at the 166 cases of listeria infection acquired during pregnancy, the fetal mortality rate was 100%, 71%, and 5% with infection acquired in the first, second, and third trimester, respectively [46]. The reason for high mortality in the first trimester is not very well clear and is most likely confounded due to better reporting of cases resulting in fetal losses and missing out on the mild or moderate infection due to the nonspecific clinical presentation of the listeria in the pregnancy (flu-like illness).

### 26.4.3 Listeria in Newborn Period (0–28 Days) or Neonatal Listeriosis

The incidence of listeria in the newborn period is about 8 per 100,000 live births, with an overall reported fatality rate of 20–30% [47]. Infection in a newborn can be transmitted by different routes: trans-placental by infected amniotic fluid or perinatal through the vagina [48]. Early onset within first 7 days can present with neonatal pneumonia and respiratory distress (61%), fever (48%), neurological symptoms (24%), skin rash (20%) and jaundice (5%) [6]. The rate of mortality in early onset is significantly high compared to late-onset (60% vs. 20%) [6]. The infants in late-onset cases (days 8–28) are usually term and are born to asymptomatic mothers with no perinatal complications, with meningitis more commonly reported (90%) compared to early onset [49]. Maternal demographics and outcomes in both early and late-onset cases are comparable, while the overall yield of listeria detection is higher in the early onset cases. The maternal blood culture or genital culture is usually positive for Listeria (44–90%) in the early onset cases [50].

### 26.4.4 Granulomatosis Infantiseptica

It is a widespread presentation of *Listeria monocytogenes* where diffuse granulomas are seen in various systemic organs like liver, spleen, lungs, kidneys, and brain of the fetus or neonate. There can be associated skin lesions. Mortality is very high in these cases [51]. On histopathological examination of the placenta, there is distinctive chorioamnionitis with acute villitis along with macro abscess formation. Listeria can be seen clearly within the amniotic epithelium with Gram stain or silver methenamine stain. It is considered as a pathognomonic for Listeriosis [52].

## 26.5 Diagnosis

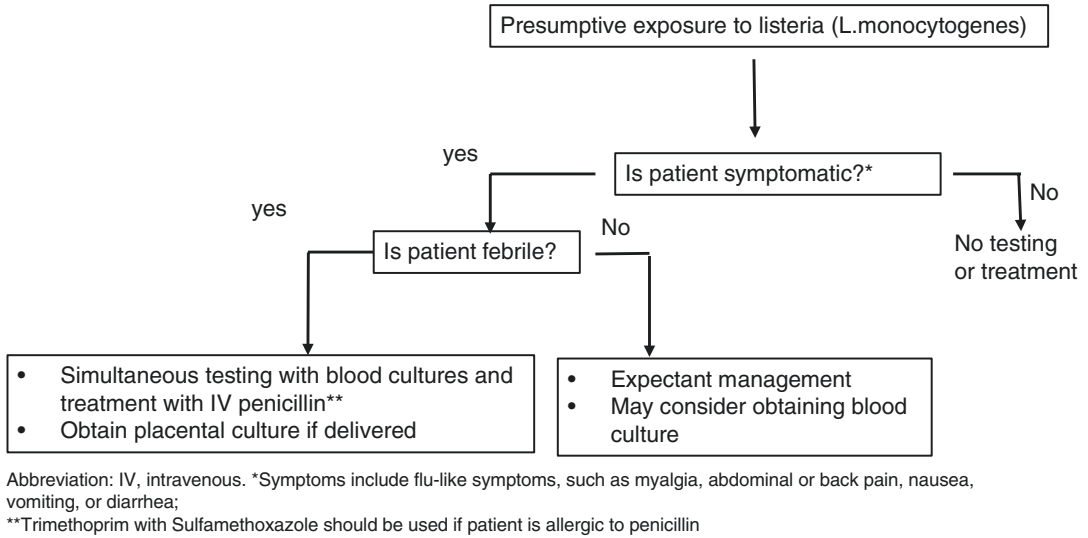
Listeriosis can be diagnosed by obtaining cultures from sterile areas like amniotic fluid, blood, or cerebrospinal fluid [53]. Gram staining is not a very sensitive method to diagnose *Listeria* infection. It gives results in only 33% of cases. It is thereby important to inform the microbiologist about the clinical suspicion of the diagnosis, which can improve the yield from cultures obtained from the contaminated sites like the vagina or rectum [25, 26]. It is always recommended that if a pregnant woman presents with fever and there is a high suspicion of Listeriosis, blood is the specimen of choice. If the amniotic fluid is obtained following amniocentesis, it is mostly meconium stained. Gram-positive rods are seen on staining. Stool culture has not been advocated as a screening or diagnostic specimen

for Listeriosis during pregnancy. This is because bacteria are usually present in the food and the environment. It is frequently shed in stool with fecal carriage of around 5% and may not indicate true infection resulting in poor sensitivity for the diagnosis of listeriosis. Polymerase chain reaction (PCR) and antibody testing for listeria are not available currently. Once the diagnosis is confirmed in any individual, it should be reported to appropriate authorities since it is considered as a notifiable disease in many countries.

The aim of listeria infection detection and treatment is to improve the fetal and neonatal outcomes. Guidelines from various countries (Ireland [54], USA [18], Canada [55], and Australia [56, 57]) have been approached to compile in this chapter (Table 26.2). All of these guidelines are based on expert opinions. They have divided the pregnant women with presumed

**Table 26.2** Guidelines on the management of women with presumptive exposure to *L. monocytogenes* (from mildest to the most aggressive approach), left to right [58]

Symptoms	Country			
	Ireland [54]	USA [18]	Canada [55]	South Australia [56, 57]
Asymptomatic	No testing, No fetal surveillance, Counsel regarding potential symptoms	No testing, No fetal surveillance, Counsel regarding potential symptoms	No testing, No fetal surveillance, Counsel regarding potential symptoms	Consider to start antibiotic therapy—oral amoxicillin/ampicillin (2–3 g/day for 7 days)
Mildly symptomatic	Consider blood Culture, Consider antibiotic Therapy, Consider fetal surveillance	Consider antibiotic Therapy, Consider fetal surveillance	Stool for routine culture, Consider stool cultures, Counsel regarding potential symptoms	Start antibiotic therapy—oral amoxicillin/ampicillin (2–3 g/day for 7 days)
Severe infection: febrile with or without other symptoms consistent with Listeria	Simultaneously test by blood culture and start the antibiotic therapy. Obtain placental cultures after delivery. If blood culture results negative, consider whether to continue the antibiotic therapy based on clinical status	Simultaneously test by blood culture and start the antibiotic therapy, Obtain placental cultures after delivery, If blood culture results negative, consider whether to continue the antibiotic therapy	Two sets of blood cultures taken from different sites, Another set of blood culture after 48 h if the previous cultures are negative, Culture of other potentially involved anatomic site (e.g., joint, pleural, pericardial fluid, sputum)	Start antibiotic therapy—Intravenous amoxicillin/ampicillin (4–6 g/day for 14 days)



**Fig. 26.3** Management of pregnant women with presumptive exposure to listeria

infection with listeriosis into three different groups. The following recommendations from the American College of Obstetricians and Gynecologists (ACOG), Fig. 26.3 [18] are described in the text, whereas all the others are enumerated in Table 26.2.

### 1. Asymptomatic Pregnant Women

For women who are asymptomatic and their only complaint is a possible exposure to *Listeria* during an outbreak or when noted during recall, no testing is recommended. However, they should be kept on follow-up and should be asked to report if they have symptoms in the 2 months following the exposure. Also, no fetal monitoring is required in such cases, and regular antenatal care is provided.

### 2. Mild Symptoms with *Listeria*-Like Illness Without Fever

Limited evidence is available to support management in this subgroup. When a pregnant woman presents with gastrointestinal or flu-like symptoms, i.e., myalgias, nausea, vomiting and/or diarrhea with a history of eating food that could be contaminated like *Listeria*, but does not have fever, then she can be managed using two

approaches. The first approach is expectant management, like in asymptomatic pregnant women with a note for follow-up in case she develops fever or symptoms worsen. Another approach is to test the patient followed by treatment if she comes as positive. The test of choice should be blood culture. At the time of delivery, placental cultures can be sent. Of note, it is important to inform the microbiologist with the suspicion of *Listeria* as the causative organism, which helps in increasing the yield of the testing.

Again, the need for treatment is based on clinical judgment; some clinicians would like to initiate the treatment with intravenous penicillin without waiting for the results, while others would prefer to wait for the definitive diagnosis and then start the treatment. There is no data to prove the superiority of either of the above lines of treatment.

### 3. Fever With/Without Other *Listeria*-Like Symptoms

It is recommended to screen and treat the pregnant women with a history of possible exposure to *Listeria* if she presents with fever (temperature > 100.6 °F). The other symptoms and signs of listeriosis may or may not be present at the time of presentation. With no other identifiable



causes of fever in such women, listeria infection should be presumed, and the appropriate treatment should be initiated. Blood cultures at presentation and placental cultures at delivery should be sent. Since the blood culture has a low sensitivity (0–55%) for listeria, the continuation of treatment if the cultures are negative should be individualized based on the overall clinical status of the patient. Amniocentesis can be considered if the patient is clinically stable, and the laboratory should be informed for the targeted amniotic fluid culture. A multidisciplinary team assessment involving an Infectious diseases specialist and Maternal-fetal specialist is also warranted.

## 26.6 Treatment

Listeria can survive and grow within host cells, so the infection may not respond favorably to bacteriostatic antibiotics. High dose of Intravenous (IV) ampicillin (6–12 g/day) for 14 days is the recommended treatment of choice. In case of the previous history of penicillin allergy, Trimethoprim with Sulfamethoxazole IV (200–320 mg for 14 days) is the first line of treatment, followed with IV erythromycin (4 g/day for 14 days) or IV vancomycin (1 g/day for 14 days) as a second line of treatment. In an afebrile pregnant woman with a history of exposure to listeria, a 14 days oral amoxicillin regimen of 1 g three times a day is suggested. Initiating a program of fetal surveillance seems prudent for women in whom listeriosis is diagnosed or strongly suspected because of exposure and fever with or without other symptoms, although studies and data do not exist to point to one best plan for such testing.

## 26.7 Prevention and Surveillance

Prevention and increased surveillance are essential for reducing the fetal and neonatal complications. Different agencies have recommended the following guidelines to minimize the exposure to Listeria:

### 26.7.1 Infection Prevention in Pregnancy (Table 26.3)

The following recommendations for the prevention of Listeriosis in pregnancy are available on the Center for Diseases Control and Prevention (CDC) website (<https://www.cdc.gov/listeria/prevention.html>).

Health Protection Surveillance Centre, HPSC Ireland, describes various food safety measures to prevent foodborne illnesses.

Foods should be used for a short period of time and read the labels for dates of manufacture and dates of expiry, including the directions for storage.

Raw meat should be cooked properly and through and through till the center.

Separate uncooked meat from cooked food, vegetables, and ready to eat things.

All fruits, vegetables, and fruits should be washed properly before consuming, including those which are to be peeled and cut items.

Once used, the cutting materials (board, knives, peelers) should be washed properly.

Refrigeration temperatures should be checked periodically.

While heating food in microwave follow instructions given by manufacturer.

**Table 26.3** CDC recommendations for pregnant women for food safety to prevent Listeriosis during pregnancy

Foods to be avoided while being pregnant

- Hot dogs, lunch meats, cold cuts (when served chilled or at room temperature; heat to internal temperature of 74 °C [165 °F] or steaming hot)
- Refrigerated pâté and meat spreads
- Refrigerated smoked seafood
- Raw (unpasteurized) milk
- Unpasteurized soft cheeses such as feta, queso blanco, queso fresco, Brie, queso panela, Camembert, and blue-veined cheeses
- Unwashed raw produce such as fruits and vegetables (when eating raw fruits and vegetables, the skin should be washed thoroughly in running tap water, even if it will be peeled or cut)

Data from Centers for Disease Control and Prevention. Listeria (listeriosis). Prevention. Available at: <http://www.cdc.gov/listeria/prevention.html>. Retrieved July 25, 2014 [18]

Once reheated, food should be consumed. Leftover should not be kept for reuse. If cooked food is to be consumed again, it should be cooled and then refrigerated readily.

If the pregnant women cannot avoid contact with ewes during lambing time, hand hygiene can be really helpful in preventing infection.

## 26.8 Conclusion

Listeriosis is a foodborne illness caused by *Listeria monocytogenes* which is a gram-positive bacterium. The estimated incidence of listeria in pregnancy is 3–4 per 100,000 cases. Maternal infection manifests as non-specific flu like syndrome to febrile illness, but in 95% cases transplacental infection of the fetus is seen within 14 days of the infection. It is an important cause of fetal demise in the first trimester and is responsible for upto 30% fetal mortality in the newborn period. For symptomatic patients, intravenous ampicillin for 14 days is the treatment of choice and all pregnant women should follow CDC food safety guidelines for prevention of listeriosis.

### Key Points

1. Listeriosis is primarily a foodborne disease. It is caused by an intracellular organism which is seen to affect individuals with depressed cell-mediated immune system.
2. Pregnant women are more prone to this infection, so they need to be informed regarding the avoidance of possible foods that are at risk of contamination with the organism, and this should be a part regular antenatal counseling.
3. Listeria is usually asymptomatic in pregnant women but can present with flu-like illness. Its effects on fetal and neonatal life can be devastating, and fatality rates vary between 20 and 30% in the newborn period.

4. For asymptomatic pregnant women who report exposure to a potential food product involved in a food recall or outbreak, no testing or treatment is recommended. She should be followed up for 2 months post-exposure for symptoms.
5. Nonfebrile pregnant women with mild flu-like illness can be managed with two acceptable approaches. First approach is to have a close follow-up for at least 2 months for fever or other signs and symptoms. The other approach is to evaluate for listeria infection and treat if positive along with fetal surveillance.
6. In case of febrile symptomatic women with high clinical suspicion, testing, and treatment should be promptly initiated.
7. In countries where Listeriosis is a notifiable disease, all positive cases should be notified to appropriate authorities.
8. Pregnant women should be given leaflets to read about Listeria infection and measures that can be taken to prevent it during pregnancy.

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