# Obstetric Infections

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**KEYWORDS**

• Pregnancy complications • Puerperal infection • Sepsis • Pregnancy complications • Infectious • Critical care

**KEY POINTS**

- Severe sepsis and septic shock remain among the leading causes of pregnancy-associated death around the world.
- Altered cell-mediated immunity in pregnancy may predispose to infection or increased severity of infection by certain organisms (eg, *Listeria monocytogenes*, influenza, and malaria).
- The microbiology of obstetric sepsis includes enteric bacteria as well as vaginal and sexually transmitted organisms, and *L monocytogenes*.
- Management should take into account the safety of the fetus, but investigations (eg, radiological) and therapy (eg, antibiotics, vasopressors) that may be lifesaving for the mother should not be avoided.

## BACKGROUND

### *Physiology*

The pregnant woman undergoes several physiologic changes affecting various systems relevant to the assessment and management of infectious complications (**Table 1**). From a respiratory perspective, the following changes occur:

- Upper airways become edematous and hyperemic because of the effects of estrogen, which is relevant during endotracheal intubation.
- A 10% to 25% decrease in functional residual capacity (FRC) occurs, whereas total lung capacity decreases only minimally as the thoracic cage widens to compensate.[1]
- Forced expiratory volume in 1 second ($FEV_1$) and lung compliance remain unchanged, but chest wall and total respiratory compliance are reduced.[2]
- The increased progesterone levels stimulate an increase in ventilation; tidal volume and minute ventilation increase, reaching 20% to 40% more than baseline by term.[3] This increase results in a mild respiratory alkalosis with compensatory renal excretion of bicarbonate ($Paco_2$ 28–32 mm Hg; $HCO_3^-$ 18 to 21 mEq/L).

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**Table 1**
**Physiologic changes in late pregnancy**

| Parameter | Change |
|---|---|
| Respiratory | |
| Functional residual capacity | Decreased 10%–25% |
| Minute ventilation | Increased 20%–40% |
| Arterial Blood Gases | |
| $Pa_{O_2}$ | Unchanged |
| $Pa_{CO_2}$ | Reduced to 28–32 mm Hg |
| Serum bicarbonate | Reduced to 18–21 mEq/L |
| Cardiac | |
| Cardiac output | Increased 30%–50% |
| Systemic vascular resistance | Decreased 20%–30% |
| Pulmonary vascular resistance | Decreased 20%–30% |
| Renal | |
| Glomerular filtration rate | Increased 50% |
| Creatinine | Decreased (to 24–68 μmol/L; 0.29–0.77 mg/dL) |
| Hematology | |
| Hemoglobin | Small decrease (5%–10%) |
| White cell count | Small increase (8%), increased further during labor |
| Platelet count | Small decrease |

- Oxygen consumption increases because of the demands of the fetus and the maternal metabolic processes, and reaches a level up to 33% more than baseline by term.

Maternal blood volume and cardiac output increase during pregnancy, reaching a peak at 30% to 50% more than baseline levels by about 28 weeks' gestation.[4] Hemodynamic measurements in the near-term patient show this increased cardiac output, associated with a reduced systemic vascular resistance and pulmonary vascular resistance.[5] During labor and the immediate postpartum period, cardiac output is further augmented by the return of 300 to 500 mL of blood to the central circulation.

Oxygen delivery to the fetus depends on the maternal arterial oxygen content and the uterine blood flow. Maternal hypotension, alkalosis (eg, hyperventilation), and endogenous or exogenous catecholamines can vasoconstrict the uterine artery and adversely affect fetal oxygen supply.[6] Umbilical venous blood returning to the fetus has a low oxygen tension, but a high oxygen content is maintained by the increased oxygen carrying capacity (left shift of the oxygen dissociation curve) of fetal hemoglobin.

With respect to renal function, glomerular filtration rate increases early in pregnancy, reaching a value about 50% more than prepregnancy levels by the second trimester.[7] The normal serum creatinine level in late pregnancy is therefore in the range of 0.5 to 0.7 mg/dL (45–60 μmol/L). Mild ureteric dilation and mild hydronephrosis may occur as a result of uterine compression and smooth muscle relaxation.

### *Immunology*

Several changes occur in a pregnant woman's immune system to allow tolerance to paternally derived fetal antigens. Downregulation of cell-mediated immunity occurs, and maternal lymphocytes show a diminished proliferative response to soluble antigens and to allogeneic lymphocytes.[8] Decreased numbers of T-helper cells have

been documented, either because of an absolute decrease in numbers or a reduction in the CD4/CD8 ratio.[8] These effects are balanced by an intact or upregulated humoral immune response.[9] The TH1/TH2 cytokine ratio is altered in pregnancy with a predominant TH2 response, although the process is likely to be more complex.[10] The effect of these alterations on maternal immunity is a predisposition to more severe manifestations of certain infections, including some viral and fungal infections, as well as *Listeria monocytogenes*.

The pregnant woman seems to be more susceptible to the development of acute respiratory distress syndrome (ARDS) than the nonpregnant patient, which may be related to factors such as increased circulating blood volume and hypoalbuminemia, but an immunologic effect may also play a role. An inflammatory change seems to occur in the lungs as a result of the pregnant state or the process of labor and delivery, priming it for the development of ARDS.[11]

### *Microbiology*

The pregnant woman is at risk of infection by a different spectrum of infectious agents compared with the nonpregnant patient (**Table 2**).[12] This difference is caused by the altered cell-mediated immunity and the risk of organisms arising from the genital tract. The immunologic changes in pregnancy increase the risk and severity of infections caused by viruses such as influenza and varicella, as well as some systemic fungal infections such as coccidioidomycosis. Pregnant women are at risk of human immunodeficiency virus infection and the associated opportunistic infections. Urinary tract infections in pregnancy are caused by the usual gram-negative fecal flora, as well as staphylococci and group B streptococci. Pneumonia is caused by the same

**Table 2**
**Microbiological source of severe infections occurring in pregnancy. Organisms listed represent examples and are not comprehensive**

| | Predisposing Factor/Source | Organisms |
|---|---|---|
| Obstetric sepsis | Enteric | *Escherichia coli*<br>*Enterobacter* spp<br>*Enterococcus* spp<br>*Clostridium* spp<br>*Bacteroides fragilis* |
| | Vaginal | *Mycoplasma hominis*<br>*Peptostreptococcus* spp<br>*Streptococcus* (group A and B)<br>*Staphylococcus aureus* |
| | Sexually transmitted | *Neisseria gonorrhea*<br>*Chlamydia trachomatis* |
| | Hematogenous<br>Altered cell-medicated immunity | *L monocytogenes* |
| Respiratory | Usual organisms | *Streptococcus pneumonia*<br>*Haemophilus influenzae*<br>*Mycoplasma pneumoniae*<br>*Legionella* |
| | Altered cell-mediated immunity | Influenza virus<br>Herpes zoster virus<br>Coccidioidomycosis |
| Renal | Urinary stasis<br>• Smooth muscle relaxation<br>• Ureteric obstruction | *E coli*<br>*Klebsiella*<br>Group B streptococci |

organisms that affect nonpregnant patients, namely *Streptococcus pneumoniae*, *Haemophilus influenzae*, and streptococci.

The spectrum of organisms responsible for obstetric infections include vaginal, enteric, and sexually transmitted organisms, as well as anaerobes, and hematogenously spread infection with *L monocytogenes*.

## EPIDEMIOLOGY

Sepsis is a common cause of morbidity and mortality worldwide, but sepsis in the pregnant woman is infrequent. Bacteremia has been reported to occur in less than 1% of obstetric patients, and a small percentage of these develop severe sepsis or septic shock.[13] Septic shock has been estimated to occur in 0.002% to 0.01% of all deliveries.[12] Nevertheless, sepsis remains one of the 5 leading causes of pregnancy-related death around the world. Data from the Confidential Enquiries into Maternal Deaths in the United Kingdom show that most sepsis-related mortality is attributable to genital tract sepsis, and sepsis is the most common cause of direct maternal death (ie, directly related to pregnancy).[14] Their most recent data (2006–2008) generate a maternal mortality caused by sepsis of 1.13 out of a total of 11.39 deaths per 100,000 maternities. A systematic review of 40 reports of obstetric admissions to the intensive care unit (ICU) identified sepsis as the cause for admission in a median of 5% of cases, with no difference between developed and developing countries.[15]

## DIAGNOSIS

### *Obstetric Sepsis*

#### *Chorioamnionitis*

Infection of the uterine contents may be found histologically in about 20% of term pregnancies and up to 60% of preterm pregnancies,[16] but clinically evident infection only occurs in 1% to 2% of term pregnancies.[17] Infection is usually ascending in origin, often following rupture of membranes with entry of vaginal or intestinal organisms, but rare cases may occur following invasive obstetric procedures or by hematogenous spread (eg, *L monocytogenes*). Infection is usually polymicrobial in nature (see **Table 2**), and genital mycoplasmas are the most frequent organisms identified.[18] The patient may present clinically with signs of sepsis with uterine tenderness and purulent discharge. Diagnosis is confirmed by Gram stain and culture of amniotic fluid. Fetal risks of chorioamnionitis include premature delivery and neonatal sepsis.[18]

Treatment requires early administration of antibiotics to resolve maternal sepsis and prevent neonatal bacteremia. At first, broad-spectrum coverage is used until cultures are available, and may be directed according to the suspected organism (eg, ascending infection in which group B streptococci and intestinal gram negatives are a concern vs hematogenous infection by *Listeria*). Common regimens include ampicillin and gentamicin or a third-generation cephalosporin (**Table 3**). Clindamycin is sometimes added for patients undergoing cesarean section, after clamping of the cord, because the hysterotomy repair creates a local anaerobic environment.[12] A short course of antibiotics, ending after 1 postpartum dose, may be adequate.[19] Delivery provides source control but the time to delivery does not seem to significantly influence outcome. Persistent sepsis after delivery and antibiotic therapy should prompt a search for necrotizing myometritis or pelvic abscess.

#### *Postpartum endometritis*

Uterine infection is most common after cesarean delivery, particularly following prolonged rupture of membranes, and less frequently after vaginal delivery.[20] The

**Table 3**
**Examples of antimicrobial regimens for some causes of severe infection in the pregnant patient**

| Infection | Therapy |
|---|---|
| Chorioamnionitis | Ampicillin and gentamicin (with addition of clindamycin for cesarean section)<br>Ceftriaxone or cefotaxime |
| Postpartum endometritis | Clindamycin and gentamicin<br>β-Lactam/β-lactamase inhibitor combination |
| Necrotizing fasciitis | Penicillin and clindamycin (group A streptococci)<br>Ceftriaxone and clindamycin |
| Bacterial pneumonia | Similar to nonpregnant patient<br>• Hospitalized patient: ceftriaxone<br>• ICU patient: ceftriaxone and azithromycin<br>• Avoid tetracyclines and quinolones if possible |
| Tuberculosis | Isoniazid, rifampin, ethambutol<br>Pyrazinamide recommended by some authorities<br>Avoid streptomycin |
| Fungal | Amphotericin B<br>Limited data in pregnancy for newer drugs |
| Viral | Influenza: oseltamivir<br>Varicella: acyclovir |
| Severe malaria | First trimester: quinine + clindamycin<br>Second, third trimester: artemisinin |

microbiology includes vaginal and intestinal organisms and the patient may present with signs of sepsis accompanied by lower abdominal pain and purulent discharge. Prophylactic antibiotics are commonly administered before cesarean section and this should be considered in choosing a therapeutic antibiotic regimen. For example, enterococcus infection may occur in a woman who received a cephalosporin for prophylaxis. A Cochrane Review of antibiotic regimens identified numerous regimens with broad-spectrum coverage that were equivalent in efficacy to a combination of clindamycin and gentamicin.[21] Regimens with activity against *Bacteroides* and other penicillin-resistant anaerobes were superior to those lacking this activity. Persistent fever may suggest the development of a complication: an enlarged tender uterus suggests myometrial microabscesses, and subcutaneous gas or gas in the uterine walls on radiology suggests gas gangrene. Surgical intervention including hysterectomy may be necessary.

***Puerperal ovarian vein thrombophlebitis***

This is a rare complication following postpartum endometritis, and needs to be considered in the woman with an acute deterioration with fever, chills, and lower abdominal pain. The hypercoagulable state of pregnancy accompanied by vascular injury by direct trauma or infection predisposes to thrombosis and thrombophlebitis of the ovarian veins.[22] Abdominal examination may reveal a tender elongated mass and the diagnosis may be confirmed by ultrasound, computed tomography (CT), or magnetic resonance imaging.[23] Treatment involves broad-spectrum antibiotics and anticoagulation, and may require surgical intervention.

***Necrotizing fasciitis***

Post–cesarean section wound infections with cellulitis are common, and microbiology involves skin and lower genital tract organisms. Treatment with drainage, exploration,

and antibiotics is usually sufficient. Simple wound infections need to be differentiated from necrotizing fasciitis, characterized by extensive necrosis of the superficial fascia and subcutaneous tissues. Necrotizing vulvitis may complicate an episiotomy or perineal laceration wound.[12] Necrotizing fasciitis may be caused by group A streptococci, or may be polymicrobial in nature.

Group A *Streptococcus* is the organism historically responsible for puerperal fever, but infection by this organism decreased dramatically during the twentieth century. However, a resurgence has occurred over the past 20 years with reports of necrotizing fasciitis and toxic shock syndrome. Infection may occur unexpectedly following uncomplicated pregnancy and delivery and may be associated with toxic shock caused by production of exotoxins.[12,24] The patient is usually systemically ill with marked pain, out of proportion to local findings.[25] Skin changes may be a later phenomenon caused by thrombosis of perforating vessels, and may include purple discoloration, bullae, and necrosis. Necrotizing myometritis may produce a boggy and edematous uterus with minimal tenderness caused by diminished innervation.[12] Treatment of group A streptococcal necrotizing fasciitis requires antibiotic therapy with a combination of penicillin and clindamycin (which reduces toxin production), with early surgical intervention and extensive debridement.[25] Intravenous immunoglobulin may improve outcome.[26]

#### *Septic abortion*

Sepsis following abortion is usually caused by ascending infection producing endometritis or parametritis, and may occur after spontaneous miscarriage, surgical abortion, or illegal abortion. The incidence has decreased with the legalization of abortion in many countries, but in some developing countries septic abortion may account for half of all maternal deaths.[27] Patients with advanced gestation, retained products of conception, and operative trauma are at highest risk, and a delay in seeking medical attention is responsible for most cases of severe septic shock and mortality. The clinical picture is of signs of sepsis with abdominal pain, vaginal bleeding, or sanguinopurulent discharge, with a tender uterus and often adnexal or parametrial tenderness.[12] Signs of peritonitis may suggest uterine perforation. The microbiology is polymicrobial, including vaginal, enteric, and sometimes sexually transmitted pathogens. Cervical cultures should be taken. *Clostridium perfringens* infection may produce gas gangrene and concomitant *Clostridium tetani* infection should also be considered.

Treatment requires early broad-spectrum antibiotic therapy and evacuation of the uterus. Mild endometritis may respond to oral antibiotic therapy, such as doxycycline or ciprofloxacin plus clindamycin. Ultrasound documents the presence of retained products of conception. Failure to respond to initial therapy may require more aggressive surgical intervention.

### *Nonobstetric Infections*

#### *Pyelonephritis*

Acute pyelonephritis may complicate up to 2% of pregnancies,[28] making it a common cause of sepsis in pregnancy. The most common causative organism is *Escherichia coli*. Asymptomatic bacteriuria predisposes to pyelonephritis, which occurs in about 25% to 30% of these cases. A significant proportion of pregnant patients with pyelonephritis go on to develop ARDS.[29] The mechanism is not clear, but likely relates to a degree of fluid overload and hypoalbuminemia[30] as well as to a lung primed for an inflammatory event by the pregnant state.[11] Treatment of pyelonephritis is with appropriate antibiotic therapy and supportive care, which usually produce a rapid response.

### *Bacterial pneumonia*

Pneumonia is an important cause of both maternal and fetal morbidity and mortality,[31,32] with an incidence that is likely not higher than the general population, and reported in the literature from 1 in 367 to 1 in 2388 deliveries.[31,32] However, an increasing incidence of pneumonia in pregnancy may be occurring, because of human immunodeficiency virus (HIV) infections as well as because of the increased prevalence of chronic disease in pregnant women.[31] Pregnancy seems to increase the risk of major complications of pneumonia, such as respiratory failure, empyema, and pneumothorax. Pneumonia may precipitate pregnancy complications, including preterm labor, small-for-gestational-age, and intrauterine and neonatal death.[31,32]

Pneumonia in pregnancy is usually caused by the common bacterial agents, with the microbiologic spectrum being no different to the usual organisms found in community-acquired pneumonia (see **Table 2**).[33] The diagnosis of pneumonia may be delayed in pregnancy because of inappropriate reluctance to obtain a chest radiograph, resulting from a concern about radiation exposure. If a chest radiograph is necessary, this should not be withheld because the risk to the fetus is negligible.[34] Antibacterial therapy is generally the same as treatment in the nonpregnant patient. Penicillins, cephalosporins, and macrolides are considered safe in pregnancy, but some drugs such as tetracyclines and quinolones should be avoided if possible (see **Table 3**).[33]

### *Viral infections*

Viral pneumonitis is associated with an increased mortality in pregnancy compared with the general population, likely related to the alterations in cell-mediated immunity. Influenza pandemic data show this increased maternal mortality: in the influenza pandemic of 1918 to 1919, the maternal rate of mortality was as high as 27%, and, in the epidemic of 1957, half of the fatalities among women of childbearing age occurred in pregnant women.[35] The more recent 2009 swine-origin H1N1 influenza A pandemic was associated with a high incidence of severe disease and respiratory failure in pregnant women, with significant mortality.[36] Institution of antiviral therapy within 48 hours of symptoms was associated with an improved outcome. Amantadine has previously been used in pregnancy as treatment and prophylaxis, but the 2009 pandemic strain was resistant. Oseltamivir was used extensively in pregnant women during the 2009 pandemic with good effect.[37] Although influenza vaccination is an important prophylactic option, a low uptake of vaccination was noted in pregnant patients who went on to develop severe respiratory failure.[38]

Varicella pneumonia has also been associated with worse outcomes in pregnant women. Up to 40% of pregnant women with varicella pneumonia require mechanical ventilation, and the mortality in these patients is 3% to 14% with antiviral therapy.[39] Varicella is more severe in adults and probably more so in pregnant women.[39] However, not all studies have confirmed this increased incidence or mortality of varicella pneumonia in pregnancy. However, this may be caused by early treatment with acyclovir, which reduces mortality in gravid patients.[40,41] Fetal effects of varicella include low birth weight, preterm delivery, and a congenital varicella syndrome.[39]

### *Fungal pneumonia*

Although fungal pneumonias are uncommon, it seems that coccidioidomycosis is more likely to disseminate in pregnancy, particularly during the third trimester. This condition may be caused by the impaired cell-mediated immunity as well as by a stimulatory effect of progesterone and 17-beta-estradiols on fungal proliferation.[42] Amphotericin is the accepted therapy for disseminated coccidioidomycosis and this

seems safe in pregnancy (see **Table 3**). Data for prolonged use of triazoles or for echinocandins in pregnancy are either insufficient or suggest harm to the fetus.

### *Malaria*

Because of the alterations in cell-mediated immunity, pregnant women are more susceptible to malaria infections and to severe disease with significant risk of maternal and/or fetal demise.[43] Placental malaria may occur in the absence of significant blood parasitemia, with parasites sequestered in the placenta and protected from immune mechanisms. The optimal management is prevention: avoidance of exposure to a malaria area or use of mosquito nets and DEET-containing repellants and, if necessary, chemoprophylaxis.

Drug treatment of malaria depends on the resistance patterns of malaria in the geographic area of exposure and drug safety in pregnancy, for which the evidence is sparse. Intermittent preventative treatment (2–4 doses over 6 months) with sulfadoxine-pyrimethamine has been shown to be well tolerated and effective in pregnancy.[43] Some of the prophylactic drugs are contraindicated in pregnancy. Chloroquine and mefloquine are probably safe for prophylactic use in pregnancy, avoiding mefloquine during the first trimester. Atovaquone/proguanil has been used for treatment of malaria (with adequate folic acid supplementation) and may be considered for prophylaxis in a mefloquine-resistant area. The safety of artemisinins in pregnancy is unclear.

Severe malaria in pregnancy should be treated similarly to malaria in the nonpregnant patient, but the risk to both mother and fetus is substantial. Pregnant women are at risk of developing hypoglycemia related to severe malaria, particularly when treated with quinine. A World Health Organization report supported by a systematic review suggests treating first-trimester severe malaria with quinine plus clindamycin, and second-trimester and third-trimester disease with artemisinins.[44]

### *Other conditions*

Pregnant women are at risk of HIV infection and the associated opportunistic infections. Pneumocystis pneumonia is the most common cause of AIDS-related death in pregnancy and may have a more aggressive course than in the nonpregnant patient.[45] Treatment with trimethoprim-sulfamethoxazole has the potential for adverse effects on the fetus, particularly near term, because of the risk of hyperbilirubinemia. However, in the case of pneumocystis pneumonia treatment or prophylaxis, the benefits of this treatment generally outweigh these potential risks.

Intra-abdominal sepsis in the pregnant woman may be related to similar causes as in the nonpregnant patient. Diagnosis may be complicated by the altered position of intra-abdominal organs and by reduced peritoneal signs, caused by stretching of the peritoneum. Imaging by ultrasound should be considered, but CT scan of the abdomen and pelvis may become necessary. Although these studies carry a small risk for the fetus, they may be essential and lifesaving. Percutaneous drainage and laparotomy may be complicated by the large uterus and abnormal anatomy of intra-abdominal organs.

## ANTIMICROBIAL MANAGEMENT

As in the nonpregnant patient, identification of causative organisms by culture, and early source control and antibiotic administration, are paramount. The spectrum of microbiology affecting pregnant women needs to be borne in mind, including vaginal, enteric, and sexually transmitted organisms as well as group A streptococci, *Listeria*, and others (see **Table 2**). Antibiotic choices in pregnancy are similar to those for the

nonpregnant patient, with a few exceptions. Initial broad-spectrum antibiotic therapy should be used, and de-escalated to a narrow-spectrum agent once culture results are available. Cephalosporins are often used for prophylaxis during cesarean section and subsequent infections should be treated with an alternative class of drug. Tetracyclines are avoided because of their effect of discoloring teeth. Some concern exists regarding adverse effects of fluoroquinolones causing fetal musculoskeletal abnormalities. Although this effect has been shown in animals, a meta-analysis of the use of quinolones in pregnancy showed no concerns[46] and these drugs are used in pregnancy when no other reasonable options are available (eg, oral ciprofloxacin for pseudomonas infection).

## SURGICAL INDICATIONS AND THERAPY

Management of sepsis in the pregnant patient follows a similar approach to that in the nonpregnant patient. The fetus is particularly susceptible to maternal hypotension, which should be rapidly managed with volume resuscitation. The pregnant woman should be placed in the left lateral position to reverse the supine hypotension syndrome occurring because of compression of the inferior vena cava.[47] If cardiac output is measured, the increased levels expected in pregnancy should be borne in mind. Vasopressor therapy may be required for persistent hypotension. Although many vasopressor drugs may adversely affect uterine, and therefore placental, perfusion, the benefit of correcting maternal hemodynamics outweighs this concern. Data on the fetal effects of vasopressors are limited and largely derived from older studies in animal models. It may be difficult to distinguish whether the adverse fetal effects are caused by the hypotension or the administered vasopressor. Norepinephrine, epinephrine, and dopamine have all been shown to adversely affect uterine blood flow.[48] Ephedrine and, more recently, phenylephrine have been used without problem in small bolus doses or infusion (phenylephrine) for maternal hypotension secondary to neuraxial anesthesia.[49,50]

Although the management of fever is not always considered necessary in the stable patient in an ICU, high body temperature may have adverse structural and functional effects on the fetus. Several potential fetal effects have been described, predominantly neurologic (including autism) as well as craniofacial and cardiac defects.[51] Fever should be rapidly treated with antipyretic agents and external cooling.

Electronic fetal heart rate (FHR) monitoring may be used to identify changes in fetal physiology, and is of value once the fetus is viable and delivery may potentially be beneficial. Because maternal physiology does not give the fetus priority over other organs, the presence of a stable fetus is reassuring for adequate maternal oxygen delivery. FHR monitoring may be performed intermittently (eg, once or twice a day), or continuously if staff are available to read and interpret the tracing. Equipment should be available at all times for urgent vaginal or operative delivery, as well as resuscitation equipment for a preterm neonate. The resuscitation status of the fetus should be identified after discussion between the family and neonatologists.

## OUTCOMES

Sepsis is an important cause of maternal morbidity, and may be the most common cause of direct maternal death.[14] The mortality of pregnant or postpartum women admitted to the ICU with sepsis has been reported as 14% to 39%.[52,53] The prognosis for pregnant women who develop septic shock may be better than that in the general population, likely because of the younger age group, lack of associated comorbidity, and a site of infection that is amenable to surgical intervention.[13]

## SUMMARY

- Severe sepsis and septic shock remain one of the leading causes of pregnancy-associated death around the world.
- Altered cell-mediated immunity in pregnancy may predispose to infection or increased severity of infection by certain organisms (eg, *L monocytogenes*, influenza, and malaria).
- The microbiology of obstetric sepsis includes enteric bacteria as well as vaginal and sexually transmitted organisms, and *L monocytogenes*.
- Management should take in to account the safety of the fetus, but investigations (eg, radiological) and therapy (eg, antibiotics, vasopressors) that may be life-saving for the mother should not be avoided.

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