Post partum infections: A review for the non-OBGYN

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Abstract

The epidemiology of infections in the puerperium (post partum period) is not well understood and remains underestimated because surveillance systems are often limited to the acute care setting. The most common source of persistent fever after delivery is genital tract infection for which diagnosis remains mostly clinical and antibiotic treatment empiric. This review will emphasize surgical site infections (SSIs) and endometritis. Septic thrombo-phlebitis, mastitis, urinary tract infections and rare infections will be covered in less detail. Puerperal sepsis will not be reviewed.

Keywords

Puerperal infection, endometritis, pregnancy complications, puerperal disorders

Introduction

The epidemiology of infections in the puerperium is not well understood. Infection affects 5–7% of postpartum women with higher rates for women undergoing caesarean sections, but real prevalence remains underestimated because surveillance systems are often limited to the acute care setting.¹ The Centre for Disease Control and Prevention (CDC) estimates that up to 84% of surgical site infections (SSIs) are diagnosed post-operatively in the post-acute care setting.² Community surveillance post-caesarean section has been shown to increase detection of SSIs by up to 71%.^{3,4} In addition to SSIs, post-discharge surveillance programs have documented endometritis, mastitis, urinary tract infections (UTIs), and episiotomy infections at higher rates than reported by hospital surveillance systems.^{1,5–7}

This review will emphasize SSIs and endometritis as the most common sources of persistent fever after delivery. Significant fever is defined as an oral temperature of 38.5°C or higher in the first 24 h after delivery, or 38°C or higher for at least 4 consecutive hours thereafter. Even though SSIs and endometritis can be indistinguishable as the surgical wound includes the uterus, they are discussed separately as endometritis may develop following vaginal delivery. Antibiotic recommendations will be provided based on empirical coverage, however, in all cases where collection of sterile specimens for pathogen identification is possible, those results should guide directed therapy to reduce the risk of antibiotic resistance.

Surgical site infections

In this review, SSIs are defined as infections of the surgical wound following caesarean section. In the era of universally recommended antibiotic prophylaxis prior to caesarean section, SSIs affect 3–5% of women undergoing caesarean section but prior studies had placed the rate of infection upwards of 15%.^{5,6,8,9}

Caesarean sections are considered clean-contaminated procedures as the surgical wound is in contact with vaginal flora.¹⁰ Preoperative risk factors for SSIs include diabetes, obesity, prolonged use of systemic steroids, smoking and pre-existing infections such as intra-amniotic infection or bacterial vaginosis. The length of surgical procedure and use of foreign material such as sutures further increase the risk of infections.^{10,11}

The most common organism in non-obstetric SSIs is *Staphylococcus aureus*; however, in obstetrical surgeries gram-negative rods, enterococci, group B streptococci, and anaerobes must be considered.¹⁰ Wound infections can present early or late following caesarean section, with pain often being the first sign. Deeper hematomas or seromas may drain from the surgical wound or be recognized as an area of fluctuance below the incision. Deep tissue infection may be seen

on imaging as collections of fluid or gas in the pelvis following surgery.^{10,12}

Treatment of wound complications following caesarean section involves antibiotic therapy and source control. Localized infections can be treated empirically with agents directed towards gram-positive organisms such as cefazolin and vancomycin. Systemic symptoms of any type warrant treatment with intravenous broad-spectrum antibiotics including gentamicin (5 mg/kg q24 h) and clindamycin (900 mg IV q8 h) plus or minus ampicillin (2 g then 1 g q4 h). Alternatively, a regime with ampicillin, gentamicin, and metronidazole (500 mg q8 h) or levofloxacin (500 mg q24 h) and metronidazole can be effective¹² (see Table 1).

All women undergoing caesarean section should receive antibiotic prophylaxis for prevention of wound infection and endometritis.^{12,13} A meta-analysis of randomized controlled trials ($n = 446$ women) comparing a penicillin or cephalosporin with placebo prior to elective caesarean section found a significant reduction in endometritis (RR 0.05 95% CI 0.01–0.38) and postoperative fever (RR 0.25 95% CI of 0.14–0.44).¹⁴ The recommendation for antibiotic prophylaxis before all caesarean sections is 1 or 2 g of cefazolin 15–60 min prior to skin incision (clindamycin or erythromycin may be used for penicillin allergic patients), with repeat doses if operative time >3 h or blood loss >1500 ml.¹³ Many centers are using 2 g of cefazolin for all patients regardless of patient weight. The evidence is clear that administration of antibiotic prophylaxis prior to skin incision is superior to traditional administration at time of cord clamping. It is associated with a reduction in all maternal infectious morbidity (RR 0.4 95% CI 0.18–0.87) and endometritis in particular (RR 0.2 95% CI 0.15–0.94), without increasing rates of neonatal sepsis, investigations or length of stay.^{14,15} A 2010 Cochrane review of 25 RCTS involving 6367 women looked at antibiotic prophylaxis before caesarean section comparing cephalosporins and penicillins and found no difference in their efficacy at reducing infection.¹¹ In addition to antibiotic prophylaxis, all patients should have adequate skin preparation with a chlorhexidine solution. Chlorhexidine is superior to proviodine in decreasing the risk of superficial skin and subcutaneous tissue infections (RR 0.59 95% CI 0.41–0.85).¹⁶ There is a stringent protocol for the use of high

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Table 1. Treatment recommendations by clinical entity.

Clinical Entity	IV antibiotic regimes ^{1–5}	PO antibiotic regimes
SSI limited to the skin	Cefazolin 1–2 g q8 h ^b or Vancomycin 1 g q12 h ^b	Cephalexin 500 mg QID or Amoxicillin 500 mg TID
SSI with systemic or pelvic symptoms	Clindamycin 900 mg q8 h + Gentamicin 5 mg/kg q24 h (±ampicillin 2 g then 1 g q8 h or vancomycin) ^b Or Ampicillin 2 g then 1 g q4 h + Gentamicin 5 mg/kg q24 h + Metronidazole 500 mg q8 h ^b Or Levofloxacin 500 mg q24 h + Metronidazole 500 mg q8 h ^{a,b}	Doxycycline 100 mg BID + Metronidazole 500 mg BID Or Levofloxacin 500 mg daily + Metronidazole 500 g TID Or Amoxicillin-clavulin 850 mg po BID
Endometritis	Clindamycin 900 mg q8 h + Gentamicin 5 mg/kg q24 h (±ampicillin 2 g then 1 g q8 h or vancomycin) ^{a,b} Or Ampicillin 2 g then 1 g q4 h + Gentamicin 5 mg/kg q24 h + Metronidazole 500 mg q8 h ^b	Doxycycline 100 mg BID + Metronidazole 500 mg BID Or Levofloxacin 500 mg daily + Metronidazole 500 g TID Or Amoxicillin-clavulin 850 mg po BID
Septic Pelvic Thrombophlebitis	Clindamycin 900 mg q8h + Gentamicin 5 mg/kg q24h (±ampicillin 2 g then 1 g q8h or vancomycin) Consideration for addition of heparin, although there is no evidence of benefit ⁶	Doxycycline 100 mg BID + Metronidazole 500 mg BID Or Levofloxacin 500 mg daily + metronidazole 500 g TID Or Amoxicillin-clavulin 850 mg po BID Consideration for addition of heparin

^aAdditional combinations include Aztreonam in place of Gentamicin¹; Piperacillin/Tazobactam and Imipenem/Cilastin are alternative therapies reserved for severe cases, specific local resistance patterns and issues of patient tolerance.^{1,5}

^bParenteral therapy should be continued until the patient has been afebrile for 24–48 h, has normalized leukocyte count, and their pain is improving. There is no evidence that de-escalation to oral antibiotics following parenteral treatment improves outcomes.⁷

concentration chlorhexidine solution due to its flammability and this must be strictly followed.¹⁶

SSIs remain a common source of morbidity and readmission following caesarean section despite increasing efforts to maximize prevention and, given increasing trends for caesarean deliveries worldwide, occurrence of SSIs is expected to continue to rise.

Endometritis

Endometritis is defined as an infection of the upper genital tract including endometrium, myometrium, and surrounding tissue.¹⁷ It most commonly occurs postpartum, when vaginal bacteria can access the upper genital tract.¹⁸ Reported rates of postpartum endometritis vary depending on studies and whether data included postpartum surveillance. A 2012 Cochrane review of endometritis found a 1–3% rate of endometritis after vaginal delivery and 5–10 times higher rates following caesarean section.¹⁹ More recent studies have quoted rates between 1.2–2% for vaginal deliveries and 1.5–5% for planned or emergency caesarean sections.^{5,7} Risk factors include prolonged rupture of membranes (>18 h), colonization with group A or B streptococcus, chorioamnionitis, prolonged operative time, bacterial vaginosis, internal monitoring, and multiple vaginal exams.¹⁹

Dominant pathogens are aerobic gram-positive cocci (group B streptococci, enterococci, and staphylococcal species), anaerobic gram positive cocci (peptococci and peptostreptococci species), aerobic gram-negative bacilli (*Escherichia coli*, *Klebsiella pneumoniae*, and *Proteus* species), and anaerobic gram-negative bacilli (*Bacteroides* and *Prevotella* species).¹⁷

Endometritis affects endometrium and myometrium but can progress beyond the uterus to include abscess, peritonitis, and pelvic thrombophlebitis. It is historically referred to as puerperal fever and

is divided into early (within 24–48 h) and late (>48 h) postpartum.^{17,18} Fever is often the first sign, with uterine tenderness, bleeding, and foul smelling lochia as additional signs. These can progress to include systemic inflammatory response syndrome (SIRS) and sepsis if severe or left untreated. The diagnosis remains largely clinical, as imaging modalities such as ultrasound have not been shown to improve diagnostic accuracy. A patient who presents with signs of severe systemic illness, diarrhea, and/or abdominal pain out of proportion to physical findings should raise suspicion for group A *Streptococcus* (GAS) infection which requires urgent antibiotic and surgical management given the risk of toxic-shock syndrome, necrotizing fasciitis and death.^{20,21} Outbreaks of GAS infection in postpartum women have often been linked back to colonized or infected health care providers prompting the CDC to recommend health care worker screening when two or more episodes of postpartum GAS infection cases are identified over a six-month period.^{18,20}

Late postpartum endometritis (>7 days) should raise suspicion for *Chlamydia trachomatis*, beyond the common pathogens, and testing for Chlamydia should be done when endometritis presents >7 days after delivery and in patients at high risk such as adolescents.¹⁸

Empiric combination of clindamycin (900 mg q8 h IV or 600 mg q6 h IV) and an aminoglycoside (most commonly gentamicin 5 mg/kg q24 h or 1.5 mg/kg q8 h) remains the most effective regimen to treat postpartum endometritis.²² A 2007 Cochrane review—republished in 2012—including 39 studies ($n=4221$ women) found clindamycin and gentamicin in combination to have equal or greater efficacy to other regimens including fluoroquinolones or regimens without coverage for *Bacteroides fragilis* (RR of 1.44 95% CI 1.15–1.80). There was not a significant increase in treatment failures amongst areas with a higher degree of *Bacteroides fragilis* resistance to clindamycin.²² Once daily gentamicin (5 mg/kg every 24 h) dosing has been found more efficacious with comparable toxicity to traditional q8-hour dosing.¹⁹

A more recent comparison of combination therapy with clindamycin and gentamicin (plus or minus ampicillin) against ertapenem showed similar treatment outcomes and length of stay but higher cost and higher rate of wound dehiscence for the ertapenem group.²³

Parenteral therapy should be continued until patient becomes afebrile (temperature $<37.5^{\circ}\text{C}$ for 24 h), pain improves, and the leukocyte count normalizes. Most patients respond to parenteral antibiotics within 48 h and become afebrile within 96 h. Failure to improve within this time frame or relapse of symptoms and signs usually results from abdominal wound infection in up to 50% of these patients, septic pelvic thrombophlebitis (SPT), or enterococcal superinfection, as neither cephalosporins—common caesarean prophylaxis—nor clindamycin plus gentamicin are effective against enterococcus.

The combination of clindamycin and gentamicin remains the gold standard, with the addition of ampicillin or vancomycin as a third agent if enterococcus is suspected or isolated (pure culture or heavy growth from an endometrial specimen) at efficacy rates of 90–97%.^{12,19} Stepping down to oral antibiotics after resolution of fever, clinical symptoms and leukocytosis has not been shown to decrease recurrence rates.²⁴ See Table 1 for antibiotic recommendations.

Endometritis is a potentially severe postpartum infection that most often requires hospitalization. For women with very mild disease, treatment with oral antibiotics may be considered. Late endometritis is often less severe and amenable to outpatient treatment with oral antibiotics including doxycycline or erythromycin and metronidazole.¹⁸

Septic pelvic thrombophlebitis

SPT is an infection that travels via venous plexuses in the pelvis, can involve the ovarian veins, and extend into the vena cava.¹⁷ SPT occurs in 1/500 to 1/800 caesarean sections and 1/9000 vaginal deliveries and is associated with the presence of chorioamnionitis (RR 2.74 CI: 1.34–5.46).²⁵ Incidence was much higher prior to the era of broad spectrum antibiotic therapy.^{17,25}

Patients may present with persistent pain, leukocytosis, and ongoing spikes in fever despite adequate antibiotic therapy for endometritis for more than 3–5 days. Thrombi in the pelvic veins can often be seen radiologically with CT or MRI both capable of making the diagnosis.²⁶ In multiple studies, the rates of thrombi seen by imaging patients with fever for more five days despite adequate therapy was 20%.^{25,26}

Adequate antibiotic therapy consists of intravenous IV ampicillin (2 g then 1 g q4 h), gentamicin (5 mg/kg q24 h), and clindamycin (900 mg q8 h). The addition of heparin to this regimen is controversial. Historically, heparin was used on patients with postpartum endometritis and persistent fever for more than five days on appropriate antibiotics. Deffervescence within 48 h of heparin initiation confirmed SPT.²⁵ The only randomized trial showed no benefit with the addition of heparin, and a systematic review concluded that heparin use in SPT remains controversial as there is no conclusive evidence that it provides benefit above and beyond adequate antimicrobial therapy.^{25,27} See Table 1 for therapy recommendations.

Puerperal mastitis

Mastitis can be categorized as infective or non-infective. Non-infective results from milk stasis due to incomplete emptying or malpositioned infant while infective can include cellulitis, intra-lobular tissue infection, abscess, and sepsis.

A recent study placed breast infections as the most common postpartum infection at 12% of all infections, most occurring within the first 4 weeks after discharge and a 2013 Cochrane review reported a range from 2% to 33% of all postpartum women.⁷ The incidence of breast abscess has been reported as 3% from an Australian cohort study although previous studies estimated a number closer to 20%.^{28,29}

Infection in the breast most often begins with skin flora and breakdown of the nipple and areola allowing bacterial entry.³⁰ *Staphylococcus aureus* is the most commonly implicated organism followed by coagulase negative *Staphylococcus*, and more rarely *E. coli*, or *Streptococci*.²⁹

The presentation of mastitis can range from localized to systemic. Localized signs and symptoms include discrete unilateral erythema, tenderness, and swelling which can start in a segment of the breast and spread without appropriate treatment. Abscesses can be identified by localized fluctuance, pain, and systemic symptoms including fevers and malaise.³¹ Diffuse, bilateral breast discomfort is more commonly related to engorgement or incomplete emptying. Investigations include a physical exam, ultrasound if an abscess is suspected, and possible milk cultures to look for pathologic organisms.

Guidelines for treatment recommend appropriate antibiotic therapy, regular emptying, and drainage of abscesses if present. A 2013 Cochrane review of two studies ($n = 125$ women) concluded that there is very little evidence on the effectiveness of antibiotic therapy, and more research is needed. A study comparing no treatment, breast emptying and antibiotic therapy and breast emptying alone suggested faster resolution of symptoms with antibiotic therapy (RR 1.89, 95% CI 1.45–2.47).²⁹ The second study compared two antibiotics and found no difference. The most common regimens include a penicillin, such as cloxacillin, or a cephalosporin to cover methicillin-sensitive *S. aureus* (MSSA). Inadequate clearance requires investigations and consideration of an agent against methicillin-resistant *Staphylococcus aureus* (MRSA) (first line if the patient is high risk or a known carrier).

Patient education on early signs and symptoms of mastitis, treating breast engorgement with early emptying, and seeking a medical opinion are crucial. Early treatment with an appropriate antibiotic and referral to a hospital or investigation if symptoms do not resolve after one course of antibiotic therapy is necessary to prevent complications of infection.²⁸

Urinary tract infections

Postpartum bacteriuria has been documented in 8–12% of postpartum women when using midstream urine samples but only 3.2% by suprapubic aspiration.^{32,33} Only 25% of these women will have dysuria or other UTI symptoms.³² Risk factors for postpartum bacteriuria and UTIs include caesarean section or operative delivery, bladder catheterization, epidural anaesthesia, high maternal BMI, and African American, Native American or Hispanic ethnicities.^{32–34} It is unknown if postpartum asymptomatic bacteriuria or pyuria results in increased morbidity or not. Diagnosis and therapy of acute cystitis or pyelonephritis in the postpartum population have not been studied either, and it is unknown if existing recommendations for either pregnancy or non-pregnant women should be applied. The decision to treat should be dictated by clinical assessment taking into consideration risk factors (i.e. bladder catheterization), symptoms, pyuria, and a urine culture if there is suspicion for antimicrobial resistance or a complicated infection. If voided urine cultures are sent, *E. coli* should be quantified to at least 10^2 CFU/ml to improve sensitivity. *E. coli* should not be considered a contaminant in mixed flora since its growth in voided urine usually reflects bladder growth.³⁵ The following empiric antibiotic recommendations are extrapolated from the non-pregnant female population taking into account compatibility with breastfeeding.^{36–38} First choices for acute uncomplicated cystitis remain nitrofurantoin 100 mg po BID $\times$ 5 days or Trimethoprim-sulfamethoxazole (TMP-SMX) 160/800 mg (one DS tablet) BID $\times$ 3 days. Fluoroquinolones such as ciprofloxacin 500 mg po BID $\times$ 3 days should be considered second line and oral beta-lactams should not be used on their own and should be reserved as a last line for uncomplicated cystitis.³⁶ Nitrofurantoin and TMP-SMX are considered safe for breastfeeding in the absence of G6PD deficiency. Even though fluoroquinolones (ciprofloxacin and ofloxacin) are compatible with breastfeeding as per the American Academy of Pediatrics, they should not be used as first line therapy as they reach higher levels on breastmilk than maternal serum and risk

to the neonate is unknown. When using fluoroquinolones in lactating women, pumping and disposing of breast milk at least 2 h after dosing must be considered.

Acute pyelonephritis can be treated orally with fluoroquinolones for 7 days (such as ciprofloxacin 500 mg BID) or TMP-SMX (1 DS tab) twice daily for 14 days. An initial IV dose of ceftriaxone or an aminoglycoside is recommended for severe infection or when resistance is unknown.³⁶

Perineal/episiotomy infections

Episiotomy infections are classically reported as being rare at a rate of 0.1% and increasing up to 2% if a third or fourth degree tear occurs.¹⁸ A large retrospective chart review placed the episiotomy infection rate at 0.3% following vaginal delivery.¹ While routine episiotomy is no longer performed, infections still occur and are often seen post-vaginal or operative vaginal delivery. Vaginal hematomas and midline episiotomy may be associated with higher risk of postpartum perineal infections.¹⁷

Episiotomy infections most commonly present with increasing pain, discharge, edema, and occasionally systemic symptoms of infection. Localized erythema and tenderness over the episiotomy site are the most common physical signs. Pain or systemic symptoms out of proportion to physical findings at the perineum can be a sign of deep tissue or necrotizing infection which requires systemic antibiotics and surgical exploration to remove dead tissue.¹⁸ A rectovaginal exam must be considered to rule out occult rectal injury.¹⁸

Oral antibiotics with coverage against streptococci, staphylococci, enteric, and anaerobic organisms are first line in treating perineal infections.¹⁸ Maintaining an intact perineum at vaginal delivery is a crucial step in preventing infection.³⁹ Limiting episiotomy to feto-maternal indications, recognition and proper closure of third and fourth degree tears, and the addition of prophylactic antibiotics in these tears have been recommended to reduce the rates of infection.⁴⁰ Teaching proper care of the perineum postpartum to maintain cleanliness of the wound is paramount to maintain the best environment for wound healing.

Other infections

Epidural abscess, infection of the deep para-spinal muscles, meningitis, and osteomyelitis have sporadically been reported as a complication of epidural anesthesia for labour and delivery in healthy women.^{41–44} Incidence is unknown, but likely very low (one case of epidural abscess in 506,000 deliveries reported in a retrospective study). Skin organisms (*Staphylococcus aureus*) have been the most frequently implicated, but other bacteria (*Streptococcus salivarius*) and *herpes simplex* (for meningitis) have been reported as well. Diagnosis requires a high index of suspicion, as presenting symptoms such as headache or back pain are also common complaints in the postpartum period. Most cases of epidural abscess have been diagnosed by day 5 postpartum either on the presence of focal neurological signs and/or fever with positive blood cultures. Essential aseptic precautions when providing epidural or spinal anesthesia including wearing a face-mask, sterile gloves and sterile gown, removal of wristwatch, hand washing, proper skin preparation, and a properly placed sterile drape are recommended.^{45,46} No systematic review has evaluated these recommendations, and wide variations in what is considered to be essential aseptic precautions for placement of epidural catheters for labor and delivery have been documented.^{46,47}

Pregnant women are at increased risk of influenza-related morbidity and mortality, seasonal and pandemic strains alike.^{48,49} Persisting risk of severe influenza-related illness was observed for at least two weeks postpartum during the 2009 H1N1 pandemic influenza A season, prompting CDC to extend its recommendation for treatment with antiviral medications for the first two weeks postpartum. Postpartum women, particularly in the first two weeks should be encouraged to seek early medical attention for influenza-like illness.

Conclusion

Infections in the puerperal period can pose a significant risk of morbidity to women. The true incidence is not fully understood as outpatient surveillance data are lacking. The best evidence for treatment of endometritis and SSIs is for parenteral therapy with limited data recognizing oral alternative regimes, making referral to hospital or parenteral therapy services a key in preventing severe infection. Reducing infection risks with prophylactic antibiotics, appropriate skin preparation, limiting vaginal exams, and adhering to sterile technique are paramount to decreasing infection risk. Postpartum women are particularly susceptible to infection and all health professionals caring for these women must be aware of the specific risk factors, presentations, and treatment protocols in order to limit hospitalization and infectious complications.

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Contributorship

Eliana Castillo (EC) conceptualized review and provided critical revision of the article for important intellectual content. Elise Dalton (ED) did literature review and drafted manuscript.

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