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Chorioamnionitis: Prevention and Management

Marie Hastings-Tolsma, PhD, CNM, FACNM,
Rachel Bernard, BS,
Mollie Gilbert Brody, BSN, RN,
Jennifer Hensley, EdD, CNM, WHNP, LCCE,
Kate Koschoreck, MSN, CNM, and
Elisa Patterson, MS, CNM

Abstract

Chorioamnionitis most often occurs during labor, affecting as many as 10% of laboring women. When intrapartum chorioamnionitis occurs, women are at peripartal risk for endometritis, cesarean birth, and postpartum hemorrhage; and the neonate is at significant risk for sepsis, pneumonia, respiratory distress, and death. The impact is greater for preterm infants where the incidence of chorioamnionitis is nearly 30%. When chorioamnionitis is believed to be present, antibiotics are administered, but not without potential adverse consequence to the mother/fetus, as well as significantly increased healthcare cost. A number of factors increase the risk of chorioamnionitis, including use of intrauterine pressure catheters and fetal scalp electrodes, urogenital tract infections, prolonged rupture of membranes, digital vaginal examinations, and the nature of perineal hygiene. This article presents key intrapartum factors and those nursing actions that can help to reduce rates of chorioamnionitis and improve perinatal outcomes.

Key words: Chorioamnionitis; Fetal tachycardia; Intrapartum; Maternal infection; Perineal hygiene.

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Chorioamnionitis, or intraamniotic infection, is a histopathologic finding of inflammation of the fetal membranes (the amnion and/or chorion) (Czikk, McCarthy, & Murphy, 2011) and may extend to the umbilical cord (funisitis) (Tita & Andrews, 2010). Chorioamnionitis commonly results from polymicrobial infection of the amniotic fluid, fetal membranes, placenta, and/or uterus. It is thought to affect as many as 10% of all laboring woman (Newton, 2005; Redline, 2012), though rates of treatment for presumed chorioamnionitis may be significantly higher. Anecdotal reports suggest nearly 8% of women in term labor are treated for symptoms believed to reflect chorioamnionitis. Chorioamnionitis presents significant risk for both the laboring woman and her fetus. Perinatal nurses are in a key position to mitigate these risks by influencing practices related to the development of chorioamnionitis.

Etiology

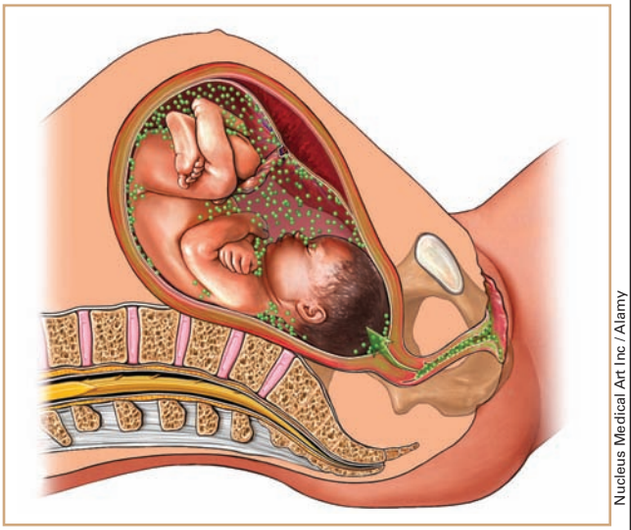
Chorioamnionitis can result from iatrogenic causes (e.g., amniocentesis) or transplacental passage from maternal blood-borne infection, though in term pregnancy it is thought to be primarily an ascending infection (see Figure 1). This pathogenic bacterial invasion may stimulate maternal and fetal inflammatory responses with the release of endotoxins, prostaglandins, and cytokines capable of triggering rupture of membranes, cervical remodeling, and/or uterine contractions (Goldenberg, Hauth, & Andrews, 2000). Other contributing factors to the development of chorioamnionitis include a compromised maternal immune system, use of internal monitoring devices, amnioinfusion, prolonged rupture of membranes (>12 hours), urogenital infections, and more than four vaginal examinations in labor (Curtin, Katzman, Florescue, & Metlay, 2012; Soper, Mayhall, & Froggatt, 1996; Tita & Andrews, 2010), though the number of cervical examinations in term labor has not been found to be an independent risk factor for intrapartum fever (Cahill et al., 2012) (see Table 1). Obesity may also be a confounding variable influencing the development of chorioamnionitis (Menon, Taylor, & Fortunato, 2010) with rising rates of obesity likely contributing to the increased incidence of chorioamnionitis (Tita & Andrews, 2010). A genetic component is also a potential risk factor as women with chorioamnionitis are at increased risk in subsequent pregnancies (Cohen-Cline, Kahn, & Hutter, 2012). Other potential contributing factors include periodontal disease, lower genital tract infections, cervicitis, and urinary tract infections (Klein & Gibbs, 2005). Finally, rates of chorioamnionitis differ by race/ethnicity (Martin & Menacker, 2007), though the reason for this is unclear. The underlying commonality with these processes is likely an exaggerated inflammatory state due to the disease and/or genetic predisposition.

A factor that has received little attention is perineal hygiene, believed to be especially important during labor. As labor progresses increased vaginal secretions are common and create a medium conducive to the growth of bacteria, easily introduced into the vagina and upper reproductive structures, especially when rupture of membranes has occurred. Where invasive procedures such as cervical digital examinations and internal monitoring devices are used, risk of introducing pathogens increases. Imseis, Trout, and Gabbe (1999) obtained bacterial cultures pre- and postdigital cervical examination in women presenting in labor 34 weeks or more gestation. Findings demonstrated a doubling of organisms following just one examination ($p < .0001$), though perineal hygiene was not examined.

A factor that may contribute to perineal hygiene practices in labor and chorioamnionitis rates is the large number of women who labor primarily in bed. The majority of women receive regional anesthesia (Martin & Menacker, 2007) and, in many institutions, large numbers of patients have internal electronic monitors inserted. With immobility common and the need for supportive care due to pain decreased, perineal hygiene measures have been anecdotally noted to be infrequent or irregular. Poor perineal hygiene, prolonged supine periods, and frequent invasive vaginal procedures may combine to promote genital tract colonization from ascending bacteria, promoting chorioamnionitis (Imseis et al., 1999).

Chorioamnionitis can result in significant peripartal sequelae that are believed to reflect an exaggerated inflammatory response beyond the proinflammatory state known to exist in normal pregnancy (Norman, Bollapragada, Yuan, & Nelson, 2007). It is not well understood how the profiles of circulating inflammatory cytokines that mediate maternal and infant responses to infection differ with chorioamnionitis. The ability to identify such profiles could contribute to interventions targeted at those who are at highest risk for poor outcomes (Smulian, Shen-Schwarz, Vintzileos, Lake, & Ananth, 1999). Maternal adverse

Figure 1. Ascending bacterial infection in the development of chorioamnionitis.



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outcomes include cesarean birth, postpartum hemorrhage, and infections (endometritis, wound, pelvic abscess, bacteremia). Septic shock, disseminated intravascular coagulation, adult respiratory distress syndrome, and maternal death can also occur, though are relatively rare where broad spectrum antibiotics and medical support are available (Newton, 2005; Tita & Andrews, 2010).

Neonatal effects may be seen at or shortly after birth and manifest as temperature instability, breathing problems, and feeding difficulties—all potential indicators of neonatal sepsis. Other possible sequelae include cerebral palsy, pneumonia, respiratory distress, and death (Tita & Andrews, 2010; Wu et al., 2003). Prematurity is the most important cause or consequence of chorioamnionitis with the incidence inversely related to gestational age (Redline, 2012, Strunk et al., 2011). Impacting nearly 30% of preterm births, early subclinical chronic infection may explain the high incidence of chorioamnionitis (Goldenberg et al., 2000).

Diagnosis

Chorioamnionitis may be clinical or subclinical. Primary clinical findings include maternal fever (≥38 °C), maternal (>100 beats per minute [bpm]) and/or fetal tachycardia (>160 bpm), maternal leukocytosis on complete blood count (>15,000 cells/mm^3), and uterine tenderness and/or purulent and/or foul-smelling amniotic fluid. Presence of fever and tachycardia are highly suggestive of chorioamnionitis with other symptoms less-sensitive indicators (Soper et al., 1996; Tita & Andrews, 2010). Fever and tachycardia, however, may be the result of other factors such as medication, maternal illness, dehydration, and high body mass index (Frölich, Esame, Zhang, Wu, & Owen, 2012; Tita & Andrews, 2010). Epidurals have also been identified as a cause of maternal intrapartum fever, though findings from prospective observational study suggest otherwise (Frölich et al., 2012). Nevertheless, these factors may contribute to overdiagnosis and treatment of chorioamnionitis (Borders, Lawton, & Martin, 2012). Table 2 identifies differential diagnoses for chorioamnionitis that should be considered in the face of associated signs and symptoms.

A definitive diagnosis of chorioamnionitis through amniotic fluid culture (Fahey, 2008) is not often performed due to time constraints. When it is done, anaerobic, aerobic, and atypical bacteria have been found with mycoplasmas, anaerobic Gram-negative bacilli, coliforms, anaerobic streptococci, and Group B streptococci most commonly isolated (Edwards, 2005). Other laboratory testing that may be helpful in the diagnosis of chorioamnionitis include amniotic fluid glucose, Gram stain, C-reactive protein, and determination of proinflammatory cytokines (interleukin-6, interleukin-1, interleukin-8, matrix metalloproteinase-8, tumor necrosis factor-alpha), though all have relatively low predictive value (Menon et al., 2010; Newton, 2005; Tita & Andrews, 2010) since results can be abnormal for reasons other than chorioamnionitis.

Subclinical chorioamnionitis does not manifest findings noted with clinical infection but should be suspected with premature prolonged rupture of membranes and preterm labor (Klein & Gibbs, 2005). A histological examination can confirm infection of the placenta and umbilical cord (Menon et al., 2010), though results may be negative even with suggestive clinical findings (Newton, 2005). With suspected chorioamnionitis, the placenta should be sent to pathology.

Table 3 details clinical and laboratory diagnosis of chorioamnionitis and the corresponding sensitivity of each item in term labor, where known. Unfortunately, most of these markers for chorioamnionitis do not discriminate well and may not be noted in subclinical chorioamnionitis. It is likely that useful future markers will mandate a combination of biomarker profiles based on varying factors including genetic, epigenetic, behavioral, psychosocial, and molecular processes (Menon et al., 2010).

Table 1. Risk Factors Associated With the Development of Chorioamnionitis

Prolonged rupture of membranes (≥12 hours)
Meconium-stained amniotic fluid (heavy or particulate)
Prolonged labor
Nulliparity
High body mass index (obesity)
Regional anesthesia
Immunocompromised status
Periodontal disease
Select race/ethnic backgrounds (e.g., African American)
Urogenital tract infections (e.g., group B streptococcus colonization, urinary tract infection, cervicitis)
Prior history of chorioamnionitis
Invasive vaginal procedures Amnioinfusion Cervical examinations (>4) Internal monitoring
Decreased frequency of perineal hygiene

Source: Cahill et al. (2012); Cohen-Cline, Kahn, and Hutter (2012); Curtin, Katzman, Florescue, and Metlay, 2012; Edwards (2005); Imseis et al. (1999); Klein and Gibbs (2005); Menon et al. (2010); Soper, Mayhall, and Froggatt (1996); Tita and Andrews (2010).

Management

There is disagreement among healthcare providers regarding the diagnostic criteria for treating chorioamnionitis. Some healthcare providers treat based solely on fever; others require use of two or three clinical findings as criteria for treatment (Newton, 2005). Confounding variables make diagnosis based upon temperature alone uncertain. For example, there is widespread belief that epidural analgesia is associated with temperature elevation in laboring women (Lieberman & O'Donoghue, 2002).

If correct, the high numbers of women receiving epidural analgesia make it likely that chorioamnionitis is overdiagnosed, contributing to unnecessary antibiotic use (Goetzl, Cohen, Frigoletto, Lang, & Lieberman, 2003). Despite the potential benefits of perinatal antibiotics, such treatment can increase cost and increase the risk of antibiotic resistance including cases of late-onset neonatal infection (Didier et al., 2012).

Despite the difficulty in diagnosing chorioamnionitis, it is important that efforts in prompt recognition be made. Timely administration of intrapartum antibiotics, as well as antipyretics, has proven to be a significant factor in reducing the incidence of postpartum maternal and neonatal complications (Newton, 2005; Smulian et al., 1999). All antibiotic treatment regimens should be broad spectrum, covering both anaerobes and aerobes (Hopkins & Smaill, 2002).

Although a diagnosis of chorioamnionitis is an indication for birth that may necessitate labor augmentation, it is not an indication for cesarean birth (Rouse et al., 2004). Patients should be observed closely for responsiveness to antibiotic therapy or signs of worsening disease (sepsis, shock). Neonatal care staff should be made aware of maternal chorioamnionitis in anticipation of newborn needs.

Prevention Strategies

Obstetrical attendants have a responsibility to safeguard the laboring patient from practices that unnecessarily increase the risk of chorioamnionitis. Nurses, as the constant attendant during labor, are in an ideal position to influence the nature and extent of many interventions that have become commonplace. Those responsibilities center on patient education, intrapartum care, and documentation (Table 4).

Patient Education

Chorioamnionitis is a theoretical risk for all laboring women. Signs and symptoms of chorioamnionitis, the importance of promptly reporting rupture of membranes, and the importance of chemoprophylaxis if positive for Group B streptococcus should be taught to all women prenatally. Antenatal education needs to emphasize laboring behaviors that minimize risks for chorioamnionitis. Discussion should include the risks of elective induction, the value of ambulation during labor, and alternatives to regional anesthesia, ensuring awareness of the increased risk of chorioamnionitis with epidural use (Smulian et al., 1999).

Intrapartum Care

Many interventions in labor that are done for the convenience of healthcare providers increase the risk of developing chorioamnionitis. Elective inductions should be discouraged as they increase the likelihood of invasive interventions. In contrast, where premature rupture of membranes has occurred, prompt induction should be considered to decrease the likelihood of developing chorioamnionitis (American College of Obstetrician

Table 2. Differential Diagnosis of Chorioamnionitis

Associated Sign/ Symptom	Possible Causes
Maternal fever	Ambient room temperature Dehydration Epidural analgesia Extrauterine infection (e.g., urinary tract infection) High body mass index Medication (e.g., prostaglandins) Prolonged labor
Maternal tachycardia	Anemia Fever Hyperthyroidism Hypovolemia Medication (e.g., β-agonists, antihistamines, ephedrine)
Fetal tachycardia	Fetal anemia Fetal hypoxia Fetal tachyarrhythmia Maternal fever Medication (e.g., β-agonists, hydroxyzine) Prematurity
Maternal leukocytosis	Diabetic ketoacidosis Eclampsia Infection Inflammatory diseases (e.g., immune disorders) Medications (e.g., prednisone, nonsteroidal anti-inflammatory drugs) Stress of labor
Uterine tenderness and/or abdominal pain	Extrauterine infection • Appendicitis • Influenza • Pneumonia • Pyelonephritis Noninfectious etiology • Colitis • Connective tissue disorders (e.g., systemic lupus erythematous) • Placental abruption • Round ligament pain • Thrombophlebitis (e.g., iliac vein thrombosis) • Uterine rupture
Foul-smelling amniotic fluid	Genital tract infection (e.g., sexually transmitted infections, vaginitis) Poor maternal hygiene

Table 3. Clinical and Laboratory Diagnosis of Chorioamnionitis

Indicators	Findings Suggestive of Chorioamnionitis	Indicator Sensitivity
Clinical		
Maternal fever	≥38 °C	42% sensitivity; 86.5% specificity
Maternal tachycardia	>100 beats/min	47.4% sensitivity; 69.7% specificity
Fetal tachycardia	>160 beats/min	36.2% sensitivity; 83.7% specificity
Maternal leukocytosis	>15,000 cells/mm^3	
Uterine tenderness and/or abdominal pain	Tenderness on palpation	
Vaginal discharge	Purulent and/or foul smell	
Laboratory		
Inflammatory Biomarkers: Proinflammatory cytokines		
• Granulocyte colony-stimulating factor	↑ in amniotic fluid, cervical/vaginal secretions, maternal serum	
• Interleukin-6	↑ in amniotic fluid, cord blood, cervical/vaginal secretions, maternal serum	81% sensitivity; 75% specificity. Poor correlation with maternal serum values; wide ethnic variation
• Interleukin-1	↑ in amniotic fluid, cervical/vaginal secretions	
• Interleukin-8	↑ in cervical/vaginal secretions	
• Matrix metalloproteinase-8	Positive	
• Tumor necrosis factor-alpha	↑ in amniotic fluid, cervical/vaginal secretions, and maternal serum	
C-reactive protein	↑ in maternal serum, amniotic fluid	Nonspecific indicator of inflammatory response; presence in amniotic fluid better indicator
Fetal fibronectin	↑ in cervical/vaginal secretions	
Leukocyte esterase	Positive (trace or greater on dipstick)	85–91% sensitivity; 95–100% specificity
Amniotic fluid culture	Positive, typically polymicrobial anaerobic and aerobic organisms but also fungi and mycoplasma (e.g., *Ureaplasma urealyticum and Mycoplasma hominis*)	Diagnostic gold standard
Amniotic fluid glucose	Low, <15 mg/dl	57% sensitivity, 74% specificity. Values influenced by maternal glycemic levels.
Amniotic fluid Gram stain	Positive (bacteria present) or if white cell count is >30 cells/mm^3	24% sensitivity, 99% specificity
Histologic examination: • Placenta • Umbilical cord	Evidence of immunocyte infiltration and neutrophil accumulation	83–100% sensitivity, 23–52% specificity

Source: Curtin et al. (2012); Edwards (2005); Goldenberg, Hauth, and Andrews (2000); Klein and Gibbs (2005); Menon et al. (2010); Newton (2005); Redline (2011); Smulian et al. (1999); Tita and Andrews (2010).

Gynecologists Committee on Practice Bulletins-Obstetrics, 2007), though an individualized approach is reasonable since clear evidence for immediate induction is lacking (Marowitz & Jordan, 2007).

Invasive procedures such as artificial rupture of membranes, digital cervical examinations, and internal monitoring devices need to be avoided without clear indication as they increase the potential for inoculation of amniotic

fluid with pathogenic organisms (Soper et al., 1996), increasing chorioamnionitis rates (Smulian et al., 1999). Cervical examinations are of particular concern and are likely excessive, particularly in academic teaching facilities (Borders et al., 2012). Where premature rupture of membranes has occurred, minimizing digital cervical examinations and other vaginal procedures and augmenting labor are important for reducing the risk of chorioamnionitis (Marowitz & Jordan, 2007).

Vaginal irrigation with chlorhexidine as a means of preventing chorioamnionitis and perinatal mortality is not recommended (Berghella, Baxter, & Chauhan, 2008). However, more frequent perineal hygiene likely decreases the risk of developing chorioamnionitis in term women though an optimum protocol has not been determined. Perineal care is generally relegated to labor nurses and evidence-based protocols are needed to ensure care that promotes best outcomes.

Documentation

Accurate documentation of care during labor is crucial to determine those practices most effective in the prevention, diagnosis, and treatment of chorioamnionitis. Much of what is known about chorioamnionitis is based on information in the patient record and much of that is documented by the nurse. The actual number of invasive interventions is of particular importance. Anecdotal reports suggest that the number of invasive procedures performed during labor may be greater than that recorded by nurses in the electronic record. For example, cervical examinations, artificial rupture of membranes, and placement of internal monitors often involve multiple attempts, though the nurse typically documents only one event. Multiple attempts may be due to the need to verify student or other healthcare provider findings, or difficulty in performing the intervention (e.g., placement of a scalp electrode). Most studies related to chorioamnionitis have involved retrospective study of medical record data, which would likely benefit from verification through prospective study.

Nursing documentation should also record the extent of patient ambulation, labor and birthing positions, and the frequency and nature of perineal hygiene. Current documentation systems likely fail to capture important intrapartum data. Collaboration between informatics colleagues and perinatal nurses to create documentation fields that demonstrate the value of intrapartum care by nurses and those measures that improve patient outcomes would be beneficial. Required fields documenting the Bishops score for labor inductions, clinical diagnosis when intrapartum antibiotics are administered, real-time quantification of patient activities throughout labor and birth, and fuller description of nursing care (e.g., nature of perineal hygiene) are examples of the types of documentation needed. Improvement in data quality will render the patient record a valid source for clinical research (Weiskopf & Weng, 2013).

Table 4. Suggested Clinical Implications

Nurses who work with pregnant and laboring women:
• Discuss prevention strategies with women who have a history of chorioamnionitis in a prior pregnancy, including weight loss, treatment of periodontal disease, treatment of genitourinary tract infection, and the importance of Group B streptococcus chemoprophylaxis.
• Encourage prompt reporting when rupture of membranes is suspected.
• Detail risks from elective induction.
• Discuss with women the value of ambulation and laboring upright.
• Discourage the use of early regional anesthesia.
• Minimize invasive vaginal procedures such as the use of intrauterine pressure catheters, electronic fetal scalp electrodes, and intracervical foley bulbs.
• Carefully document the actual number of invasive vaginal procedures, including, the total number of cervical examinations.
• Discourage unnecessary artificial rupture of membranes.
• Provide regular, standardized perineal hygiene, particularly for women unable to ambulate.
• Record the rationale for antibiotic administration, noting when given for presumed chorioamnionitis.

Chorioamnionitis affects approximately 10% of laboring women and carries significant risk for mother and baby.

Summary

Many factors that contribute to the development of chorioamnionitis and its potentially devastating consequences can be minimized or eliminated. However, research is needed to clarify diagnostic criteria and risk factors, detailing those that may be modifiable. Investigation of intrapartum care practices is also needed with emphasis on the impact of invasive vaginal procedures when accurately recorded, and the influence of perineal hygiene protocols. Interventions that are known to interfere with normal birth processes such as epidural administration and internal monitoring are in particular need of rigorous examination.

The WHO has long held that in normal birth there should be a valid reason for interfering with the natural process (WHO, 1997). Nurses and midwives are the standard bearers for care that promotes safe and normal birth. Patients should be safeguarded from practices that reduce the likelihood of normal birth and create risk for adverse outcomes such as chorioamnionitis. ✣

Marie Hastings-Tolsma, PhD, CNM, FACNM, University of Colorado Denver College of Nursing Division of Women, Children & Family Health. She can be reached via e-mail at Marie.Hastings-Tolsma@ucdenver.edu.

Rachel Bernard, BS, University of Colorado Denver College of Nursing.

Mollie Gilbert Brody, BSN, RN, University of Michigan Health System.

Jennifer Hensley, EdD, CNM, WHNP, LCCE, University of Colorado Denver College of Nursing.

Kate Koschoreck, MSN, CNM, University of Colorado Denver College of Nursing.

Elisa Patterson, MS, CNM, University of New Mexico College of Nursing.

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