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Group B Streptococcus and Pregnancy

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ABSTRACT:
Group B streptococcus, or Streptococcus agalactiae, is a gram-positive bacterium commonly found in the genital and gastrointestinal tracts of humans and less frequently in the upper respiratory tracts of children and adults, which poses significant health risks, particularly to neonates, young infants, pregnant women, and those with certain medical conditions. Group B streptococcus can colonize the gastrointestinal and vaginal tracts of up to one-third of pregnant individuals and can cause various infections, including asymptomatic bacteriuria, urinary tract infections, chorioamnionitis, postpartum endometritis, pneumonia, puerperal sepsis, and bacteremia. Although group B streptococcus accounts for a small percentage of urinary tract infections and peripartum bacteremia during pregnancy, it can occasionally lead to meningitis and endocarditis. However, vertical transmission of group B streptococcus during vaginal birth can result in early-onset group B streptococcus disease in newborns, posing the most significant risks to the neonate, including bacteremia, sepsis, and death. Universal third-trimester screening and intrapartum antibiotic prophylaxis have significantly reduced neonatal group B streptococcus infections, although these measures may have unintended consequences for mothers and infants. Effective prevention of neonatal group B streptococcus disease depends on proper screening, timely antibiotic administration, and coordination with pediatric care clinicians. Despite CDC recommendations for routine group B streptococcus screening and intrapartum antibiotics, group B streptococcus remains a leading cause of early-onset neonatal sepsis in the India. Up to 40% of individuals who test positive for group B streptococcus during prenatal care may test negative at delivery, leading to significant overtreatment and increasing antibiotic resistance. Therefore, new therapeutics to prevent group B streptococcus colonization at delivery are still being investigated. This activity for healthcare professionals is designed to enhance the learner's competence in recognizing the significance of group B streptococcus in pregnancy, performing the recommended evaluation, and implementing appropriate interprofessional preventive management approaches to improve patient outcomes.

Objectives: •Screen for group B streptococcal infection in pregnancy sing established protocols. •Implement intrapartum antibiotic prophylaxis (IAP) guidelines based on GBS screening results and patient risk factors •Select the appropriate treatment for group B streptococcal infection in pregnancy. •Implement interprofessional team strategies to improve care coordination and outcomes in patients with group B streptococcal infection during pregnancy.

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INTRODUCTION

Group B streptococcus (GBS), or Streptococcus agalactiae, is a gram-positive bacterium commonly found in the genital and gastrointestinal tracts of humans and less frequently in the upper respiratory tracts of children and adults. This bacterium poses significant health risks, particularly to neonates, young infants, pregnant women, and those with certain medical conditions.[1] GBS can colonize the gastrointestinal and vaginal tracts of up to one-third of pregnant individuals, resulting in various infections, including asymptomatic bacteriuria, urinary tract infections, chorioamnionitis, postpartum endometritis, pneumonia, puerperal sepsis, and bacteremia.[2] Although GBS accounts for a small percentage of urinary tract infections and peripartum bacteremia during pregnancy, it can occasionally lead to more severe maternal sequelae, such as meningitis and endocarditis.[3] However, invasive maternal GBS infections pose the most significant risks to the neonate, including bacteremia, sepsis, and death.[4]

Vertical transmission of GBS during vaginal birth can result in early-onset GBS disease (GBS-EOD) in newborns. Maternal colonization of GBS in the gastrointestinal tract and vagina is the primary risk factor for GBS-EOD in neonates. According to the Centers for Disease Control and Prevention (CDC), approximately 0.23 cases per 1000 live births are diagnosed with early-onset GBS in the India.[4] Preventive measures include correct specimen collection, nucleic acid amplification testing (NAAT) for GBS identification, and specific regimens for mothers with premature rupture of membranes, preterm labor, or penicillin allergy, along with coordination between obstetrics and pediatrics.

The American College of Obstetricians and Gynecologists (ACOG) recommends universal GBS screening at 36 to 37 6/7 weeks of gestation, with positive cases receiving appropriate intrapartum antibiotics. Penicillin is the preferred antibiotic, with alternatives available for individuals allergic to penicillin. Effective prevention of neonatal GBS disease depends on proper screening, timely antibiotic administration, and coordination with pediatric care clinicians.[5] Before the widespread use of maternal intrapartum chemoprophylaxis, the incidence of early-onset GBS was much higherEverywhere, neonatal GBS-EOD rates have declined markedly due to guidelines for maternal GBS screening and intrapartum antibiotic prophylaxis (IAP). Universal third-trimester screening and IAP have significantly reduced neonatal GBS infections, although these measures may have unintended consequences for mothers and infants. Currently, an estimated 31% of individuals in the United States are administered antibiotics for intrapartum GBS prophylaxis.[2]

Despite CDC recommendations for routine GBS screening and intrapartum antibiotics, GBS remains a leading cause of early-onset neonatal sepsis in the India. Furthermore, up to 40% of individuals who test positive for GBS during prenatal care may test negative at delivery, leading to significant overtreatment and increasing antibiotic resistance. Therefore, evolving therapeutics to prevent GBS colonization at delivery are still being investigated, including probiotic interventions and maternal vaccines.[6][7]

Etiology

GBS is the primary cause of GBS-EOD in newborns; maternal GBS colonization is the primary risk factor for newborn GBS-EOD, as maternal-neonatal transmission occurs in approximately half of the women colonized by GBS without IAP.[8] According to studies, 20% to 30% of the women in the United States are colonized with GBS. Transmission from mother to infant typically occurs during labor or delivery when GBS bacteria from the maternal gastrointestinal and genitourinary tract ascend into the uterine cavity, infecting the fetus through direct colonization or the aspiration of infected amniotic fluid. This transmission is common during labor in term infants, whereas preterm infants may be infected earlier, potentially causing premature rupture of membranes and preterm labor.[4] Rarely, GBS-EOD can develop before labor if bacteria traverse intact membranes.

GBS disease diagnosed between 7 and 89 days of age is termed late-onset GBS disease and is a much rarer manifestation of neonatal GBS infection. However, unlike GBS-EOD, transmission is believed to occur not only from maternal colonization but also from transmission by other caregivers. Consequently, GBS IAP has not reduced the incidence of late-onset GBS.

Maternal Group B Streptococcus Colonization and Neonatal Early-Onset Disease Risk Factors

Factors that increase the risk of maternal GBS colonization and GBS-EOD in newborns include:

- Gestational age <37 weeks
- Young maternal age <20 years
- Low neonatal birth weight
- Prolonged rupture of membranes
- Maternal fever during labor ≥100.4 °F (38 °C)
- Previous infant with GBS-EOD
- Heavy maternal vaginal–rectal GBS colonization or GBS bacteriuria[8]

Some obstetric interventions, such as frequent vaginal examinations, invasive fetal monitoring, and membrane sweeping, may increase the risk of GBS-EOD; however, the data are not conclusive.[4]

Epidemiology

The prevalence of GBS colonization in pregnant women in India is estimated to be between 10% and 30%.[9][7] The estimated prevalence of maternal GBS colonization worldwide ranged from 11% to 35%. The incidence of GBS-EOD in newborns among women with GBS colonization is approximately 29 times higher compared to that of neonates without maternal GBS colonization.[10] Over the last 20 years, advancements in screening for GBS colonization and IAP have significantly decreased the incidence of GBS-EOD from approximately 1.8 cases of GBS-EOD per 1000 live births to approximately 0.23 per 1000 live births.[8] In addition, early risk assessment has reduced GBS-EOD incidence by 60% to 70%.[4]

Pathophysiology

GBS primarily colonizes the female rectovaginal tract asymptomatically, using mechanisms such as binding to host surfaces and evading the immune system. These pathophysiological mechanisms also help GBS spread and cause damage, especially in newborns. GBS colonization can also be intermittent or persistent.[11] Some GBS strains are more virulent due to specific factors that enhance their ability to spread, evade maternal immunity, and cause tissue damage. Consequently, GBS's regulation of virulence factors and specialized adhesins makes it a significant pathogen, particularly in newborns.[12]

At the cellular level, GBS regulates its virulence factors through signal transduction systems, allowing it to adapt and survive in various host environments beyond the lower genital tract. The most studied system is CovR/S, which regulates many virulence genes. GBS also controls virulence using one-component transcriptional regulators and a serine/threonine kinase system.[12]

GBS adheres to epithelial cells using surface-associated adhesins such as fibrinogen-binding proteins (Fbs), hypervirulent GBS adhesin (HvgA), and others. These adhesins help in colonization and facilitate invasion and immune evasion. Certain strains, such as the highly virulent clonal complex 17, express adhesins with higher binding affinity, aiding in dissemination and pathogenicity.[12]

Key adhesins include:

- Ssr1/Ssr2: Ssr2, found in clonal complex 17 strains, has a greater binding affinity to fibrinogen and can bind plasminogen, aiding in dissemination.
- HvgA: Found in clonal complex 17 strains, HvgA enhances adherence to intestinal and brain cells, promoting colonization and crossing of barriers.
- FbsA, FbsB, FbsC: These fibrinogen-binding proteins are crucial for adherence, biofilm formation, and dissemination
- Lmb: Binds laminin, aiding in tissue invasion and neurotropism.
- C5a peptidase: Facilitates immune evasion and adherence
- Pili: Cell wall–anchored structures that assist in adherence and resistance to immune mechanisms.
- PbsP: Promotes vaginal colonization and invasive infection.
- SfbA: Enhances invasion of various cells and contributes to brain colonization.
- BibA: A multifunctional protein involved in colonization and immune evasion.[12]

GBS also produces a hemolytic pigment regulated by the CovR/S system, which aids in colonization, tissue damage, and immune evasion. Other virulence factors include superoxide dismutase for resisting reactive oxygen species, hyaluronidase for tissue invasion, and a sialic acid–rich capsular polysaccharide for immune

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evasion by mimicking maternal protein structures or molecular mimicry.[12]

History and Physical

Clinical manifestations of maternal GBS infection include intra-amniotic infection, urinary tract infection, endometritis, bacteremia, and, less frequently, meningitis and endocarditis.[13][14][15][16] Intra-amniotic infection, or chorioamnionitis, is an infection of the amniotic fluid and membranes, placenta, or umbilical cord. Urinary tract infections include pyelonephritis, cystitis, and asymptomatic bacteriuria.[17] Asymptomatic bacteriuria, identified through routine urine cultures performed during prenatal care, is treated when a bacteria concentration of at least 100,000 CFU/mL is reached, as recommended by the Infectious Diseases Society of America.[18] However, asymptomatic bacteriuria of any concentration or a urinary tract infection with GBS isolates identified is evidence of maternal GBS colonization, and IAP is indicated.

Clinical features of chorioamnionitis, endometritis, or urinary tract infections secondary to GBS include maternal fever (temperature 100.4 °F (38 °C) or higher), uterine tenderness, maternal and fetal tachycardia, purulent amniotic fluid, dysuria, costovertebral tenderness, and maternal leukocytosis. Maternal GBS colonization is also associated with a higher risk of premature rupture of membranes, preterm labor, and neonatal infections, such as sepsis.[17] Signs and symptoms of intra-amniotic infection also include maternal leukocytosis and purulent cervical drainage.[19] Furthermore, clinical features that are suspicious for intra-amniotic infection include preterm prelabor rupture of membranes, term rupture of membranes for ≥18 hours, and intrapartum fever.[8]

Evaluation

Group B Streptococcus Screening in Pregnancy

Universal GBS screening during pregnancy is the primary approach utilized to evaluate patients for GBS colonization in globally, testing is only performed on symptomatic patients, such as those with vaginal discharge and premature rupture of membranes.[11] This screening approach varies between countries due to the debate regarding decreasing neonatal sepsis with the risk of causing increased antimicrobial resistance.[11]

Studies show that the negative predictive value of the GBS culture is highest (95% to 99%) in the first 5 weeks after collection. However, the predictive accuracy diminishes significantly when the interval between culture collection and birth exceeds 5 weeks, which may be secondary to some women found to have GBS colonization intermittently.[8][4][11] The 2010 CDC guidelines recommended universal prenatal GBS screening starting at 35 weeks of gestation. Currently, the ACOG advises universal GBS screening between 36 and 37 6/7 weeks of gestation. This adjustment is based on the recommendation for antibiotic prophylaxis for women with unknown GBS status who give birth before 37 weeks and that screening within this time frame ensures that culture results are valid for up to 5 weeks, covering births up to at least 41 weeks of gestation. Furthermore, the purpose of these updated screening recommendations was to decrease the incidence of inconsistent antepartum culture results and GBS colonization at birth. For women who do not give birth within this 5-week window and have a negative initial GBS screening, repeat screening is advisable to guide management beyond 41 weeks of gestation.[8][4]

Screening is also advised for women presenting in preterm labor or with preterm premature rupture of membranes, such as before 37 weeks, to guide future IAP in patients where delivery does not occur. In these patients, a positive GBS screen is an indication for IAP at any point labor resumes. However, in patients with preterm labor or rupture of membranes with a negative GBS screen, IAP is not indicated if labor resumes within the 5-week screening range. In patients that remain pregnant for longer than 5 weeks following GBS screening, the screening should be repeated if preterm labor begins again or at 36 to 37 6/7 weeks of gestation. In addition, if GBS colonization is identified through urine culture, reconfirmation with a vaginal-rectal culture is unnecessary in any patient. GBS screening may use culture methods or NAAT.[8][4]

Group B streptococcus culture: For GBS culture collection, a single swab is used to sample both the lower vagina and the rectum without a speculum. This method significantly increases culture yield compared to sampling the cervix alone or the vagina without rectal culture. Incorrect specimen collection, typically vaginal cultures without rectal sampling, is the most common GBS prenatal screening error among clinicians. Studies

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have also demonstrated that patients trained to collect their own vaginal-rectal samples can achieve culture yields comparable to those collected by clinicians. Swabs should be placed in a nonnutritive transport medium. GBS culture specimens are typically viable in these media for several days at room temperature, but their recovery declines within 1 to 4 days, especially at higher temperatures, potentially leading to false-negative results.[8]

Specimens should clearly indicate that the GBS samples are from a pregnant woman and whether the woman is allergic to penicillin to ensure appropriate testing protocol. For women at high risk of anaphylaxis to penicillin, susceptibility testing for clindamycin should be requested, as 20% to 30% of GBS isolates are found to be clindamycin resistant.[11] Laboratories process GBS culture swabs by incubating them in selective enrichment broth to enhance the sensitivity of subsequent cultures. After enrichment, subcultures are made onto blood agar plates, and bacterial colonies are identified as GBS using latex agglutination with group B antisera, chromogenic agars, DNA probes, or NAAT. Inducible resistance to clindamycin is detected using the D-zone test, which indicates resistance due to macrolide-induced enzyme activity that can lead to clindamycin treatment failure.[8]

Group B streptococcus nucleic acid amplification: Although culture-based testing remains the standard for maternal antepartum GBS screening, NAAT offers a potentially more sensitive alternative. However, NAAT does not isolate the organism for antibiotic susceptibility testing, which is crucial for women with penicillin allergies. Therefore, if NAAT is used and the result is positive in a penicillin-allergic woman, an additional culture and susceptibility test should be performed.[8]

NAAT can also be used for rapid intrapartum testing but has limitations, including variable sensitivities and a failure rate of approximately 7% to 10%. Rapid testing requires 24-hour laboratory infrastructure, which limits its widespread use. Consequently, routine prenatal screening at 36 to 37 6/7 weeks remains the primary recommendation.[8] Some facilities use NAAT for point-of-care screening. However, NAAT is not the primary approach due to variable sensitivity and the inability to determine antibiotic susceptibility in penicillin-allergic women.[4]

Management

GBS colonization in pregnancy is primarily treated with IAP to decrease the transmission of GBS and the incidence of neonatal sepsis by reducing the maternal GBS burden within the genitourinary tract and eliminating GBS bacteria in the fetus.[8][4] However, IAP guidelines are not consistently implemented; therefore, GBS remains a leading cause of early-onset neonatal sepsis in India. Furthermore, up to 40% of individuals who test positive for GBS during prenatal care may test negative at delivery, leading to significant overtreatment and increasing antibiotic resistance. Consequently, studies continue to investigate alternative management approaches for GBS in pregnancy, including probiotic interventions and maternal vaccines.[6][7]

Intrapartum Antibiotic Prophylaxis Indications

IAP is recommended to prevent neonatal GBS-EOD in women with identified GBS colonization or with risk factors for GBS colonization.[8][4] Indications for intrapartum GBS prophylaxis include:

- GBS colonization identified by antenatal culture
- GBS bacteriuria detected during pregnancy
- History of a previous infant with GBS disease
- Unknown GBS status and preterm labor or preterm premature rupture of membranes (<37 0/7 weeks) [8][4]
- Unknown GBS status with any of the following risk factors at ≥37 0/7 weeks of gestation
- Maternal fever ≥100.4 °F (38 °C)
- Prolonged rupture of membranes (≥18 hours)
- Positive point-of-care NAAT for GBS
- History of GBS colonization in a previous pregnancy
- Negative intrapartum NAAT, but risk factors develop during labor, such as maternal fever and prolonged rupture of membranes [8][4]
- Suspected intra-amniotic infection (broad-spectrum antibiotics, including GBS coverage, should be given)[8][4]

However, IAP is not indicated in patients with a negative GBS culture ≥36 weeks of gestation, women undergoing a cesarean birth without labor onset or rupture of membranes, or those with an unknown GBS status at a

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 1064-1072 | ISSN:2251-6727

term gestation with a negative NAAT result and no development of risk factors during labor.[8][4]

Intrapartum Antibiotic Prophylaxis Recommendations

ACOG recommends penicillin as the first-line antibiotic for GBS prophylaxis due to its narrow antimicrobial activity, which decreases the risk of resulting antibiotic resistance.[11] A loading dose of 5 million units followed by 2.5 to 3 million units intravenously (IV) every 4 hours until delivery should be used to attain the serum drug levels needed to inhibit GBS bacteria in the fetus.[8] Using this dosage, penicillin achieves a peak concentration in cord blood within 1 hour.[4] Ampicillin 1 g IV every 4 hours following a 2 g loading dose may also be used; however, because it does not have as targeted of an antimicrobial effect as penicillin, ampicillin is less preferred.[8] IAP should be given at least 4 hours before delivery to be most effective, although a time interval from administration to delivery of 2 hours has been found to have some effectiveness. Due to studies demonstrating that the optimal time to administer IAP is ≥4 hours, clinicians should strive to adhere to this recommendation. However, urgent obstetrical interventions or delivery should not be delayed to ensure IAP within this time frame.[8] Moreover, clinically indicated obstetrical procedures, including cervical ripening, membrane sweeping, artificial rupture of membranes, intrauterine monitoring, vaginal examinations, and water births, are not contraindicated in women with GBS colonization.

Alternative Management for Penicillin Allergies

For women with penicillin allergies, the choice of IAP antibiotic depends on the allergy history and, if available, clindamycin susceptibility results from GBS cultures. Given that up to 90% of individuals with a reported penicillin history do not have a genuine allergic reaction due to desensitization over time or historical inaccuracy, it is important to assess the patient's risk for a severe penicillin reaction. Patients with a high risk for reaction are those who report symptoms including anaphylaxis, pruritic rash, hives, flushing, hypotension, angioedema, or Stevens-Johnson syndrome.[8]

In patients with a low risk of an allergic reaction, first-generation cephalosporins are recommended. Cephalosporins maintain a high susceptibility to GBS with a low incidence of severe allergic reactions in individuals with penicillin allergies. Typically, cefazolin with a 2 g loading dose followed by a maintenance regimen of 1 g IV every 8 hours until delivery is used. Penicillin allergy testing may also be considered. Clindamycin 900 mg IV every 8 hours until delivery is recommended in patients with a high risk for a severe allergic reaction to penicillin. However, clindamycin should be used only if the GBS isolate is susceptible to clindamycin.[8]

In women with a high-risk penicillin allergy without GBS susceptibility to clindamycin, vancomycin 20 mg/kg intravenously every 8 hours, with a maximum of 2 g per single dose, is recommended for IAP. However, the use of vancomycin should be carefully considered due to potential adverse effects and the development of resistance.[8]

Preterm Labor Group B Streptococcus Management

In patients with preterm labor or preterm premature rupture of membranes, the following recommendations for GBS prophylaxis have been established by ACOG:

- IAP should be started during the initial management of preterm labor and continued if labor progresses.
- If preterm labor does not progress to delivery, IAP can be stopped and managed based on culture results.
- If preterm labor reoccurs, IAP should be restarted based on previous or new GBS culture results.[8][4]

Group B Streptococcus Management for Planned Cesarean Birth

Women planning a cesarean birth should still undergo prenatal GBS culture as labor or rupture of membranes might occur before the scheduled delivery. If labor or premature rupture of membranes occurs before the cesarean delivery, a single dose of an antibiotic providing both GBS prophylaxis and presurgical coverage is appropriate. However, IAP is not necessary for women undergoing a cesarean birth without labor onset or rupture of membranes, even if GBS is positive.[8][4]

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 1064-1072 | ISSN:2251-6727

Group B Streptococcus Therapies Under Investigation

Due to known adverse effects of IAP, including allergic reactions, neonatal thrush, and antibiotic resistance, other therapeutic avenues have been researched to treat GBS colonization. Maternal GBS vaccines have shown promise in preventing GBS-EOD; however, further studies are required to provide evidence of the effectiveness and full scope of risks with these vaccines before being utilized for this indication. In addition, maternal ingestion of probiotics during pregnancy was ineffective in eliminating maternal GBS colonization; however, the authors of this study reported that their findings showed a potential reduction of persistent GBS colonization, requiring further investigation.[6][20]

Differential Diagnosis

Other conditions that should be considered when managing GBS in pregnancy include:

- Bacterial pneumonia
- Cellulitis
- Urinary tract infection in females
- Dermatologic manifestations of necrotizing fasciitis
- Diskitis
- Endometritis
- Epidural abscess
- Infective endocarditis
- Meningitis
- Osteomyelitis in emergency medicine
- Septic arthritis
- Urinary tract infection in pregnancy
- Wound infection

Prognosis

The severity and incidence of GBS infection can vary based on host factors and GBS serotypes. For instance, preterm infants have 3 times the risk of GBS-EOD compared to term infants.[11] However, since the initiation of universal screening for GBS colonization and IAP, the incidence of GBS-EOD has decreased from approximately 1.8 cases per 1000 live births to approximately 0.23 per 1000 live births.[8] In addition, early risk assessment has reduced GBS-EOD incidence by 60% to 70%.[4]

Complications

Maternal GBS colonization is associated with 10% percent of pyelonephritis cases and 5% to 10% of bacteremia cases during pregnancy.[21][17] In addition, maternal GBS is also responsible for causing endometritis, cesarean delivery, postoperative wound infections, and other ascending infections resulting in maternal sepsis and preterm births.[22][23][24][25] Maternal mastitis and breast abscesses have also been associated with GBS infection during pregnancy.[26][27]

In neonates with GBS-EOD, symptoms include tachycardia, tachypnea, or lethargy to severe outcomes such as cardiorespiratory failure, persistent pulmonary hypertension of the newborn, and perinatal encephalopathy. Complications associated with GBS disease include meningitis, neurologic impairment, hearing loss, seizure disorders, and cerebrovascular disease. Globally, GBS disease has caused approximately 150,000 fetal and neonatal deaths.[4]

Pearls and Other Issues

Universal screening for GBS colonization combined with IAP has significantly reduced the incidence of GBS-EOD by approximately 80%, with prophylaxis efficacy estimated between 86% and 89%.[9] Despite the success of these measures, it is crucial to recognize that GBS culture screening during prenatal care may not identify all women who are colonized at the time of labor due to the transient nature of genital tract colonization. Approximately 60% of early-onset GBS infections occur in neonates born to mothers who tested negative for GBS between 35 and 37 weeks of gestation.

Enhancing Healthcare Team Outcomes

Preventing GBS infection during pregnancy requires a collaborative interprofessional team effort, involving physicians, advanced practitioners, midwives, nurses, pharmacists, laboratory clinicians, and other health professionals. Screening for GBS is a fundamental task that should be undertaken by all healthcare practitioners caring for pregnant women. To enhance patient-centered care, outcomes, patient safety, and team performance, each interprofessional team member must be well-versed in their specific roles and responsibilities.

Obstetric clinicians should ensure timely and accurate GBS screening and the appropriate administration of

Journal of Chemical Health Risks
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JCHR (2025) 15(5), 1064-1072 | ISSN:2251-6727

IAP. Clinical microbiology laboratory team members are critical in performing appropriate GBS culture and sensitivity testing, guiding the management approach. Nurses are essential in monitoring pregnant women for signs of labor and infection, administering antibiotics, and providing education and support to patients and their families. Pharmacists are responsible for confirming the appropriate selection, dosing, and administration of antibiotics and monitoring for potential drug interactions and adverse effects.

Effective interprofessional communication is crucial for seamless care coordination to ensure that all necessary steps are taken to prevent GBS disease. Regular team meetings and standardized communication tools, such as SBAR (Situation-Background-Assessment-Recommendation), can help ensure that all team members are informed about each patient's GBS status and management plan. In addition, electronic health records should be used to document and share information regarding GBS screening results, antibiotic administration, and any changes in the patient's condition.

Furthermore, timely referral and consultation with specialized clinicians, such as infectious disease experts, are important when needed. Clear communication with patients about the importance of GBS screening and antibiotic prophylaxis, comprehensive discharge planning that includes follow-up care, and education on signs of neonatal infection are essential to optimize management. By working collaboratively and maintaining open lines of communication, healthcare professionals can effectively reduce the risk of GBS infection, thereby improving maternal and neonatal outcomes and overall patient safety.

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