



Case Report

Early-Onset Group B Streptococcal Infection in Bichorionic/Biamniotic Twins Case Study: Is It Time for Changes in Laboratory Diagnosis and Prevention?

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Abstract

Early-onset infection caused by *Streptococcus agalactiae* (Group B Streptococcus, GBS) may occur during gestation or delivery and can lead to severe neonatal sepsis, meningitis, or pneumonia. Discordant GBS infections in twin gestations are rare. We report a fatal case of early-onset GBS infection in dichorionic–diamniotic twins conceived via IVF and delivered by caesarean section at 32 weeks' gestation due to discordant fetal growth and abnormal Doppler indices in Twin A (Umbilical Artery PI = 1.4; Middle Cerebral Artery PI = 1.5). Twin A had Apgar scores of 3, 5, and 5 and rapidly developed tachycardia, respiratory distress, and systemic infection, while Twin B, with Apgar scores of 7, 8, and 9, remained clinically stable. Both infants were admitted to the NICU and underwent routine blood, urine, and cerebrospinal fluid testing. Despite the prompt initiation of parenteral ceftriaxone and respiratory support, Twin A deteriorated rapidly and died within 28 h. GBS was isolated from Twin A's blood culture, and maternal placental tissue and high vaginal samples collected before antibiotic administration also grew GBS, with all isolates demonstrating identical antimicrobial resistance profiles. Molecular analysis revealed matching *rib1* and *alp2/3* gene patterns in isolates from the mother and Twin A. Maternal anovaginal immunochromatography at delivery was positive, whereas screening cultures obtained at 29 weeks' gestation were negative. This case highlights the limitations of culture-based GBS screening in high-risk pregnancies and preterm deliveries and underscores the potential value of molecular assays and point-of-care testing to improve detection of *S. agalactiae* throughout pregnancy and the peripartum period. Emerging preventive strategies, including modulation of the genital microbiome and maternal vaccination aligned with WHO recommendations, may further reduce the burden of neonatal GBS disease.

Keywords: *Streptococcus agalactiae* (group B Streptococcus, GBS); prevention; early onset; discordant twins



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1. Introduction

Streptococcus agalactiae (Group B Streptococcus, GBS) is a leading cause of neonatal meningitis and sepsis. Although epidemiological data from Greece remain limited, available studies consistently report comparatively low maternal colonization rates and a low incidence of neonatal disease relative to many other countries. Earlier national data estimated maternal colonization at approximately 6–7% and neonatal colonization at 2–3% [1–3]. More recent findings from Crete suggest even lower maternal colonization (1.7%), although this may reflect regional screening practices [1,2].

Despite these low colonization rates, invasive neonatal GBS disease remains clinically significant. A 22-year multicenter cohort study from Crete reported an overall incidence of 0.17 per 1000 live births—among the lowest globally—with early-onset disease (EOD) accounting for 0.10 per 1000 and late-onset disease (LOD) for 0.06 per 1000 live births [3]. Case fatality reached 8%, and neurological sequelae were documented in approximately 40% of meningitis cases, underscoring the substantial morbidity associated with invasive disease even in low-incidence settings. Serotype distribution in Greece aligns with broader European patterns, with serotypes II, III, Ia, Ib, and V being most prevalent [1].

GBS infections are classified as early-onset (birth to day 6) or late-onset (day 7 to 3 months). Early-onset disease is typically vertically transmitted from mother to infant, whereas late-onset disease may arise from either vertical or horizontal transmission routes [4–8]. Rare cases involving twins—both monochorionic/bi-amniotic and dichorionic/bi-amniotic—have been described, often demonstrating distinct clinical trajectories [8–10]. Factors contributing to severe or fatal neonatal GBS infection, including host genetic susceptibility, bacterial virulence determinants, and maternal or fetal comorbidities, remain incompletely understood [11].

Current CDC guidelines recommend universal culture-based screening of pregnant women at 33–37 weeks of gestation, with earlier testing advised when preterm delivery is suspected. However, the limited sensitivity of vaginal and rectal cultures may result in false-negative results, thereby failing to identify colonized mothers and leaving infants at risk for invasive disease [12].

This report describes a rare fatal case of GBS infection in bi-chorionic, bi-amniotic twins conceived via IVF and discusses implications for improving detection, intrapartum chemoprophylaxis, and emerging diagnostic approaches for early-onset GBS disease.

2. Case Presentation

A 32-year-old woman with a dichorionic–diamniotic twin pregnancy conceived via in vitro fertilization (IVF) was scheduled for an elective caesarean section (CS) at her request, given the couple's long-standing unexplained infertility and repeated unsuccessful IVF attempts. First-trimester screening confirmed two healthy dichorionic–diamniotic embryos. Throughout pregnancy, she underwent monthly blood and urine tests and ultrasonographic evaluations. At 29 weeks' gestation, discordant fetal growth was noted without additional pathological findings. Group B Streptococcus (GBS) screening was performed according to ACOG guidelines and was negative.

At 32 weeks' gestation, the patient presented with mild fatigue and a body temperature of 37.1 °C but no clinical signs of infection. Due to an increased umbilical artery pulsatility index (UA-PI 1.4) and elevated middle cerebral artery pulsatility index (MCA-PI 1.5) in Twin A, an urgent caesarean section was performed. As per routine practice in our clinic, the mother received 2 g of intravenous cefoxitin approximately 30 min before surgery. Maternal blood cultures were not obtained, as she exhibited neither fever nor signs of infection. Placental samples were collected immediately after delivery for microbiological culture and histopathological examination, following the protocol for IVF twin pregnancies.

Both infants were admitted to the neonatal intensive care unit (NICU) immediately after birth. Neonatal management followed standardized hospital protocols: Twin A met criteria for the symptomatic-neonate protocol, while Twin B was managed under the protocol for asymptomatic neonates born before 32 weeks' gestation, which does not include empirical antibiotic therapy. Twin A weighed 1420 g and had Apgar scores of 3, 5, and 5 at 1, 5, and 10 min, respectively. Twin B weighed 1930 g with Apgar scores of 7, 8, and 9 and remained clinically stable; therefore, only the course of Twin A is described in detail.

Shortly after admission, Twin A developed fever (38.1 °C), followed by hypothermia, peripheral cyanosis, respiratory distress, bradycardia, lethargy, and pallor, consistent with infant respiratory distress syndrome (IRDS). Oxygen saturation remained unstable (<90–94%), and lung auscultation revealed no inspiratory crackles or expiratory rhonchi. Initial laboratory evaluation showed white blood cells 18,800/ μ L, platelets 480,000/ μ L, blood glucose 70 mg/dL, C-reactive protein 12.0 mg/dL, total bilirubin 11.2 mg/dL, and γ -GT 181 IU/L. Cerebrospinal fluid (CSF) analysis demonstrated normal cell counts and glucose levels.

Blood culture became positive for GBS within 18 h, confirming systemic infection; urine and CSF cultures remained negative. Parenteral ceftriaxone (80 mg/kg/day) was initiated, along with supportive care and oxygen therapy, although the precise timing and mode of antibiotic administration were not fully documented. Despite treatment, the infant's condition deteriorated rapidly, progressing to pneumonia and severe bradycardia, and death occurred less than 28 h after birth.

Maternal high vaginal culture obtained before antibiotic administration in the operating theatre was positive for GBS. Placental cultures confirmed GBS in the placenta of Twin A but not Twin B, suggesting ascending maternal infection with selective fetal transmission. Antibiotic susceptibility testing (Kirby–Bauer method) demonstrated identical resistance profiles across isolates from the infant, placenta, and maternal vaginal sample. All isolates were susceptible to β -lactams and quinolones but resistant to erythromycin and clindamycin, confirmed by the D-test for inducible clindamycin resistance.

Capsular serotyping was performed by an external accredited laboratory using PCR-based methods and identified the isolate as serotype III. As part of an ongoing research protocol, a pilot PCR assay targeting four GBS-associated gene regions (*cfb*, *rib1*, *alp2/alp3*) was performed. Genomic DNA was extracted from neonatal tissue using a commercial kit, and PCR amplification was carried out under standardized conditions. All isolates carried *rib1* and *alp2/3* genes, while *cfb* was not detected. PCR products were visualized by agarose gel electrophoresis. Routine NICU surveillance cultures (rectal swabs obtained from day 1 to day 7) showed no GBS colonization in other neonates.

Twin B remained clinically stable. On day 6, a blood culture grew *Staphylococcus haemolyticus*, representing transient bacteremia without clinical symptoms. After 5 days of intravenous ampicillin, blood cultures became negative, and the infant remained healthy. NICU surveillance confirmed that no other neonates were colonized with GBS.

3. Discussion

The clinical presentation of Twin A was consistent with infant respiratory distress syndrome (IRDS) complicated by fulminant early-onset GBS septicemia [12–16]. IRDS is a leading cause of NICU admission among infants born before 34 weeks' gestation and is primarily attributed to pulmonary immaturity, although infection may precipitate or exacerbate respiratory failure. In this case, the rapid deterioration of Twin A suggests that GBS infection significantly aggravated the underlying prematurity-related respiratory vulnerability.

To our knowledge, this represents one of the few documented cases of early-onset GBS infection in dichorionic–diamniotic twins with discordant outcomes. Approximately 95% of neonates with early-onset GBS disease develop symptoms within the first 48 h of life, supporting vertical transmission as the predominant route [4,7]. Only a small number of comparable discordant twin cases have been reported worldwide since the 1980s [8–10,17–19], along with one case involving discordant infection with GBS and *Citrobacter koseri*, and one additional case of female twins with survival of one infant, underscoring the rarity of selective involvement of one fetus in twin gestations.

All GBS isolates obtained at delivery in this case carried the *rib1* gene and exhibited identical antimicrobial resistance profiles, supporting a common maternal source. The *rib1* gene, located within GBS pathogenicity islands, has been associated with increased virulence and severe neonatal disease in experimental models and has been proposed as a potential vaccine antigen [12]. The presence of *rib1* in all isolates may have contributed to the unusually aggressive clinical course observed in Twin A.

Maternal colonization remains the principal risk factor for early-onset neonatal GBS infection [16]. In this case, maternal screening cultures obtained at 29 weeks' gestation were negative, in accordance with ASM recommendations [20]. However, the three-week interval between screening and preterm delivery, combined with the known limitations of culture sensitivity, likely contributed to a false-negative result. Culture-based screening has been reported to miss up to 30–47% of true carriers when compared with nucleic acid amplification tests (NAATs), reflecting methodological rather than antibiotic-related limitations. The negative screening result may have contributed to underestimation of infectious risk, despite ultrasonographic evidence of discordant fetal development.

In twin pregnancies, discordant infection most commonly affects the gestational sac adjacent to the cervix—typically corresponding to Twin A—supporting ascending maternal infection as the most plausible mechanism [16]. Hematogenous transmission has been proposed in rare cases, but the selective placental involvement observed here strongly favors an ascending route [18,19]. The combination of prematurity, IRDS, and exposure to a virulent serotype III strain likely contributed to the fatal outcome in Twin A, whereas Twin B remained unaffected.

Universal screening for maternal GBS colonization between 35 and 37 weeks' gestation was first recommended by ACOG in 1996 and later incorporated into CDC guidelines [21]. Implementation of universal screening and intrapartum antibiotic prophylaxis has reduced the incidence of early-onset disease from 1–2 cases per 1000 live births to substantially lower levels [12,21,22]. Since 2019, stewardship of GBS prevention guidelines has been shared among ACOG, the American Academy of Pediatrics (AAP), and the American Society for Microbiology (ASM), reflecting the need for coordinated clinical and laboratory standards [20].

Molecular assays, including PCR-based NAATs, are increasingly used as adjuncts or alternatives to culture for antepartum or intrapartum screening. Multiplex real-time PCR platforms demonstrate sensitivities exceeding 96% [23–25], whereas culture sensitivity may be as low as 53–70% when compared with NAATs [26,27]. These findings highlight the potential of NAATs to reduce false-negative screening results, particularly in preterm labor or when screening is performed weeks before delivery. However, NAATs cannot replace culture for antimicrobial susceptibility testing, and sequence variability within target regions may lead to false-negative results, as previously observed in assays targeting the *cfb* gene [24]. Modern platforms incorporating multiple gene targets have achieved sensitivities approaching 100% [28–31].

In our institution, culture-based screening remains the standard for twin pregnancies, including discordant gestations or those with abnormal ultrasonographic findings. NAAT

following enrichment is not yet implemented, as current ASM guidelines consider both approaches acceptable [32]. Nevertheless, evaluation of commercial molecular assays is ongoing to improve diagnostic performance.

Point-of-care (POC) molecular testing during labor offers particular value in cases without prior screening, suspected changes in vaginal flora, or emergency delivery. These assays can guide intrapartum antibiotic prophylaxis within clinically relevant timeframes (<4 h). ACOG currently supports universal antepartum screening with intrapartum testing when indicated [27,33]. Although rapid intrapartum NAATs demonstrate moderate to high sensitivity, larger studies are needed before routine implementation. ASM guidelines currently do not recommend direct-from-specimen intrapartum NAAT due to insufficient sensitivity and negative predictive value [32].

Additional emerging strategies include antigen-based immunochromatographic assays following broth enrichment, although direct testing of anovaginal specimens remains insufficiently sensitive. Ongoing clinical trials aim to clarify their cost-effectiveness and diagnostic utility [14,30–32,34,35].

Future prevention efforts may also involve modulation of the vaginal microbiome and maternal immunization. Several *Lactobacillus* species (e.g., *L. rhamnosus*, *L. plantarum*, *L. gasseri*, *L. salivarius*, *L. fermentum*) exhibit in vitro inhibitory activity against GBS and may contribute to colonization control [34,35]. The World Health Organization has identified maternal GBS vaccination as a potentially cost-effective global intervention, with the goal of preventing stillbirth and invasive neonatal disease. WHO guidelines emphasize the need for vaccines providing at least 80% protection, with safety profiles comparable to existing maternal vaccines and applicability across diverse healthcare settings [31,34,35].

4. Conclusions

We report a rare case of early-onset GBS infection in dichorionic–diamniotic twins following IVF, characterized by selective placental involvement and a fulminant, fatal course in one twin despite adherence to current screening guidelines. This case underscores the ongoing challenges in early detection and prevention of GBS disease, particularly in preterm deliveries, where the interval between screening and birth may exceed the diagnostic window of culture-based methods. Although culture remains the reference standard, its limited sensitivity can lead to false-negative results and missed opportunities for timely prophylaxis.

In high-risk pregnancies—including IVF gestations, discordant twins, and cases with abnormal fetal Doppler findings—enhanced surveillance or combined testing strategies may be warranted. NAAT-based assays offer improved sensitivity and faster turnaround times, but their use in antepartum and intrapartum settings requires careful selection, validation, and interpretation to ensure diagnostic accuracy. Emerging preventive approaches, including modulation of the vaginal microbiome and maternal GBS vaccination, hold promise for reducing the global burden of neonatal GBS disease. Continued refinement of diagnostic tools and preventive strategies remains essential to improving outcomes for vulnerable mothers and infants.

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