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Case Report

A case report on *Clostridium baratti* in placenta: pathogen or commensal

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ABSTRACT

Clostridium baratti is a rarely reported anaerobic bacterium, primarily isolated from immunocompromised patients. We report a case of *C. baratti* isolated from placental tissue in a 32-week pre-rupture of membranes patient with acute chorioamnionitis. The mother presented with fever, elevated inflammatory markers (CRP 42 mg/l, WBC 16,500/µl), and fetal tachycardia. Placental culture revealed *C. baratti* (anaerobic culture) and non-pathogenic organisms (aerobic culture). The neonate developed early-onset sepsis with leukopenia, thrombocytopenia, elevated CRP, and coagulopathy. Neonatal blood culture grew commensals; anaerobic cultures were not obtained on day 0. Four lines of evidence- isolation during active maternal infection, polymicrobial ascending flora, growth in infected tissue, temporal relationship to neonatal sepsis, coverage by empiric metronidazole, and isolation from protected placental tissue, may suggest *C. baratti* as pathogenic. This case suggests *C. baratti* as a pathogen in obstetric-neonatal infections and highlights the need for routine anaerobic culture of placental tissue and anaerobic blood cultures from chorioamnionitis-exposed neonates.

Keywords: Anaerobic bacteria, *Clostridium baratti*, Antimicrobial susceptibility, Anaerobic culture, Placenta culture, Rare

INTRODUCTION

For decades, the human placenta was considered a sterile environment; however, recent advances in molecular microbiology have fundamentally challenged this theory. Research utilizing high-throughput 16S rRNA gene sequencing has revealed the presence of diverse microbial communities within placental tissue, despite having significantly lower microbial biomass compared to other body sites. The placental microbiota is predominantly composed of bacteria from the phyla Firmicutes, Bacteroidetes, Proteobacteria, and Fusobacteria species.[1]

Under physiological conditions, the intrauterine microbiota coexists with the host in immunological tolerance. Disruption of this balance, termed as dysbiosis is characterised by overgrowth of pathogenic anaerobes such as *Mycoplasma hominis, Gardnerella vaginalis, Fusobacterium nucleatum* and *Clostridium* species which triggers excessive inflammation at the feto-maternal interface, resulting in chorioamnionitis, preterm labour, and neonatal infection; with broader associations extending to gestational hypertension, fetal growth restriction, and gestational diabetes mellitus. Premature rupture of membrane (PROM) frequently follows ascending bacterial colonisation of the chorioamniotic membranes, exposing the normally protected intrauterine cavity to polymicrobial flora from the lower genital tract. While *Bacteroides, Prevotella* and *Peptostreptococcus* species are well-recognised culprits in such infections, rarer *Clostridium* species remain poorly characterised.[1,2]

Clostridium baratti is a gram-positive, anaerobic, spore-forming bacillus that has been isolated sporadically from clinical specimens including soft tissue infections, wound infections, and bacteremia, primarily in

immunocompromised hosts.[3] However, reports of *C. baratti* in obstetric or neonatal infections are exceptionally rare, and the organism's role as a pathogen in pregnancy-related complications remains undefined. We report the first documented isolation of *C. baratti* isolated from placental tissue in association with PROM and subsequent neonatal sepsis. This case highlights an emerging anaerobic pathogen with potential significance in obstetric-neonatal medicine and demonstrates the need for enhanced microbiological surveillance and species-level identification of Clostridium isolates in pregnancy-related infections.

CASE REPORT

Maternal course

A 28-year-old primigravida at 32 weeks of gestation presented to the labour ward with complaints of vaginal fluid leakage for 2 days. She denied fever, abdominal pain, or vaginal bleeding. Obstetric examination confirmed rupture of fetal membranes (positive nitrazine test). Maternal vital signs were within normal limits at presentation; however, over the subsequent 48 hours, she developed low-grade fever (38.2°C) and increased vaginal discharge. Cardiotocography revealed baseline fetal heart rate tachycardia (170 bpm). Laboratory investigations showed elevated maternal serum C-reactive protein (CRP: 42 mg/l) and leukocytosis (WBC: 16,500/µl). Given clinical evidence of chorioamnionitis (maternal fever, elevated inflammatory markers, and fetal tachycardia), labor was induced and vaginal delivery was accomplished 12 hours later.

Placental examination and microbiological investigations

Placental tissue was sent for aerobic and anaerobic culture, along with histopathological examination. The clinical specimen received in Robertson cooked meat (RCM) medium was incubated at 37 °C for 24 hours. On day 2, a preliminary Gram stain was performed from the RCM broth, followed by subculture in triplicate manner; on 5% sheep blood agar for aerobic culture; on chocolate agar incubated in CO2 incubator or candle jar for microaerophilic organism and on to neomycin blood agar with a 5 µg metronidazole disc and 30 µg gentamicin disc, and onto selective media (Bacteroides bile esculin agar; Lactobacillus agar; Veillonella agar) for anaerobic organisms. All anaerobic culture plates were sealed with parafilm and incubated anaerobically for 48-72 hours using a GasPak anaerobic jar with *B. fragilis* ATCC 25285 and *Pseudomonas aeruginosa* ATCC 35032 as quality control.

On day 3, aerobic and CO2 incubated plates are noted and on day 4 after completion of anaerobic incubation, colonies were examined and the anaerobic suspected colony (5 µg metronidazole sensitive and 30 µg Gentamicin resistant) were subjected to preliminary identification tests, including Aerotolerence test, Gram staining, modified ZN staining, spot indole test, and 15% catalase test and nitrate disc test, three-disc potency test, 20% bile susceptibility along with esculin hydrolysis test. A second subculture was performed to ensure purity. Aerotolerance was assessed by subculturing the isolate onto 5% sheep blood agar incubated in parallel under aerobic and anaerobic conditions; growth restricted to anaerobic incubation confirmed obligate anaerobic growth. Presumptive identification was further supported by triple-disc testing (colistin 10 µg, kanamycin 1000 µg, and vancomycin 5 µg).

On day 5, Gram staining was repeated from the purity plate, and the suspected purified colonies identification was performed using the VITEK® ANC system (bioMérieux, Marcy-l'Étoile, France). On same day, antimicrobial susceptibility testing was carried out using E-test strips (HiMedia Laboratories Pvt. Ltd., Mumbai, India) on 5% sheep blood agar with lawn culture according to CLSI M11 9th edition.[4] Minimum inhibitory concentrations (MICs) were interpreted and reported as per CLSI M100 35th edition guidelines.[5,6] Antimicrobial susceptibility testing of *C. baratti* (broth microdilution method per EUCAST guidelines) showed susceptibility to penicillin (MIC: 0.5 µg/ml), clindamycin (MIC: 1 µg/ml), metronidazole (MIC: 2 µg/mL), and meropenem (MIC: ≤0.25µg/ml).

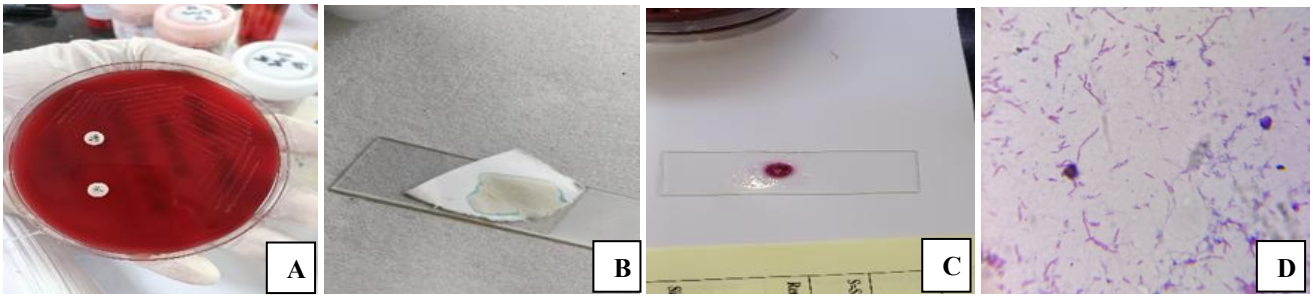

Figure 1: (A-D) Growth in neomycin blood agar; indole test-negative, nitrate test- negative, gram stain- subterminal oval spore.

Neonatal presentation and clinical course

A female neonate weighing 1.8 kg was born with Apgar scores of 5 and 7 at 1 and 5 minutes, respectively. Resuscitation was required with bag-mask ventilation. The infant required admission to the neonatal intensive care unit. Within 4 hours of delivery, she developed clinical signs consistent with sepsis: fever (38.1°C), poor feeding, irritability, and generalized hypotonia. Laboratory investigations at 4 hours of life revealed leukopenia (WBC: 3,200/µl), thrombocytopenia (platelet count: 85,000/µl), elevated serum creatinine (1.8 mg/dl), and prolonged prothrombin time (INR: 1.8). C-reactive protein was markedly elevated at 68 mg/l. Blood was received on day 1 for aerobic culture which yielded skin commensals and was reported on day 4 and repeat blood culture were not sent. Anaerobic blood culture was requested from the infant after day 6 (when placenta isolated *C. baratti* on day 5), however no growth in anaerobic blood culture.

Empiric antimicrobial therapy was initiated on day 0 with intravenous ampicillin (50 mg/kg/dose every 12 hours), gentamicin (7.5 mg/kg/dose every 18 hours), and metronidazole (7.5 mg/kg/dose every 12 hours). Lumbar puncture was performed to exclude meningitis; cerebrospinal fluid was sterile. The neonate's clinical condition improved over 48 hours with defervescence, improved vital signs, and progressive improvement in laboratory parameters (WBC normalized by day 5, CRP declined to <10 mg/l by day 8). Antimicrobial therapy was continued for 7 days. The infant was discharged home on day 14 of life in stable condition with normal physical examination.

Clostridium baratti is a saccharolytic, gram-positive, anaerobic, spore-forming bacillus that has been sporadically isolated from clinical specimens over the past several decades. The organism was first described in 1981 and subsequently identified in wound infections, soft tissue infections, intra-abdominal infections, and bacteremia, predominantly in immunocompromised patients or those with underlying malignancy.[3,7-12] While *C. baratti* is occasionally recovered from environmental samples and human stool as a commensal organism, its pathogenic potential in obstetric and neonatal infections has rarely been documented. In this case, *C. baratti* was isolated from placental tissue during active maternal chorioamnionitis.

Premature rupture of membranes (PROM) is a well-established risk factor for ascending bacterial infection of the intrauterine cavity. The intact chorion and amnion, together with cervical mucus and antimicrobial secretions, provide formidable barriers to colonization by vaginal and cervical flora. When these membranes rupture, this mechanical barrier is compromised, allowing bacterial ascent from the lower genital tract into the amniotic cavity and placental tissue. The duration of rupture without delivery is a critical determinant of infection risk; studies demonstrate that the incidence of chorioamnionitis increases with prolonging PROM duration, with significant risk manifest after 12-24 hours. In this case, the patient experienced PROM at 32 weeks of gestation with a 48-hour interval before delivery without active labor, creating an extended window for bacterial proliferation and placental invasion. The temporal sequence of PROM followed by development of maternal fever, inflammatory marker elevation, and fetal compromise within 48 hours is consistent with typical ascending bacterial infection and chorioamnionitis pathophysiology.

Clostridium baratii functions as a pathogenic organism rather than commensal colonizer, supported by four interconnected lines of evidence. First, isolation during active maternal chorioamnionitis (fever 38.2°C, CRP 42 mg/l, WBC 16,500/µl) represents systemic inflammatory response to bacterial infection, not asymptomatic colonization. Second, successful culture from inflamed placental tissue indicates the obligate anaerobe thrived in the hypoxic, anaerobic microenvironment created by inflammatory infiltration and vascular compromise. Third, *C. baratii* is temporally linked to neonatal early-onset sepsis within 4 hours of delivery, with neonatal manifestations of sepsis (leukopenia, thrombocytopenia, elevated CRP); and placental culture yielded *C. baratii* while neonatal blood culture showed commensals only, establishing selective antepartum placental invasion. Fourth, empiric metronidazole initiated on day 0 was essential to outcome, the neonate showed rapid clinical improvement (defervescence by 48 hours, normalized markers by day 5-8), demonstrating that antimicrobial coverage of *C. baratii* was clinically critical.

Neonatal anaerobic blood cultures were not obtained on day 0; cultures sent on day 6 (after metronidazole initiation) were negative. This represents a missed diagnostic opportunity. Maternal chorioamnionitis is frequently polymicrobial, and anaerobic organisms have different antimicrobial susceptibilities than aerobic organisms; their identification guides optimal therapy. Because metronidazole was initiated on day 0, anaerobic organisms present in early blood may have been suppressed or eliminated by day 6, making those cultures unreliable.

Standard anaerobic culture protocols require collection before antimicrobial therapy. However, the absence of day 0 anaerobic blood cultures prevents microbiological confirmation of anaerobic neonatal bacteremia. For all cases of maternal chorioamnionitis with neonatal early-onset sepsis, anaerobic blood cultures obtained on day 0, prior to antimicrobial therapy are essential to characterize organisms transmitted in utero and definitively document anaerobic neonatal sepsis when present.

Table 1: Case reports of *Clostridium baratti* in published literature

Year & location	No. Of cases	Clinical presentation	Risk factor/context
1985, Mexico[7]	1	Infant botulism	BoNT/F-producing strain; neonatal exposure
2007, Italy[8]	1	Bacteremia/sepsis	Kawasaki syndrome; immunocompromise
2008, Taiwan[9]	1	Lung abscess	Concurrent invasive pulmonary aspergillosis
2012, Taiwan[10]	1	Liver abscess/sepsis	Emphysematous cholecystitis; immunocompromise
2014, USA[11]	2	Adult botulism	Elderly patients; foodborne exposure
2015, France[12]	3	Botulism	Contaminated Bolognese sauce; community cluster
2016, Brazil[3]	1	Pneumonia/sepsis	Advanced Alzheimer's disease; immunocompromise

Table 1 depicts the published case reports of *C. baratti*, spanning over three decades from 1985 to 2016. Notably, the majority of documented cases involve either botulinum toxin production (infant or adult botulism) or infection in profoundly immunocompromised hosts. To our knowledge, this is the first documented case of *C. baratti* isolated from placental tissue in association with maternal chorioamnionitis. The addition of this obstetric-neonatal case expands the clinical spectrum of *C. baratti* infection and suggests that the organism may have greater pathogenic potential in maternal-fetal medicine than previously recognized.

Clostridium baratii's rarity in clinical literature reflects identification gaps and host factors rather than true rarity: most laboratories do not perform routine species-level anaerobic identification, reporting it as "Clostridium sp." or dismissing it as contaminant, and anaerobic culture of obstetric specimens is not standard practice. The organism also shows predilection for immunocompromised hosts and lower inherent virulence than toxigenic Clostridium species, limiting detection in immunocompetent populations.

Limitations

This case has several limitations. This is a single-patient case report lacking comparative controls. Anaerobic blood cultures were not obtained from the neonate on day 0; cultures drawn on day 6 (post-metronidazole initiation) were negative, preventing confirmation of anaerobic neonatal bacteremia. Quantitative bacterial burden (CFU/g) from placental culture was not measured, limiting assessment of bacterial load. Histopathological examination of placental tissue was not performed, precluding direct visualization of bacterial invasion and inflammatory changes. Molecular identification (16S rRNA, MALDI-TOF mass spectrometry, or PCR) was not performed to confirm species identity, though conventional culture and biochemical identification were used. These gaps restrict the strength of microbiological evidence, though the strong temporal and clinical association between maternal chorioamnionitis, neonatal sepsis, and response to targeted therapy provides compelling circumstantial evidence of pathogenicity.

CONCLUSION

This case establishes *Clostridium baratii* as a potential pathogen in obstetric infections, isolated from placental tissue during maternal chorioamnionitis with possible cause of neonatal early-onset sepsis. The organism's rarity in published literature reflects diagnostic gaps rather than true uncommonness. To improve recognition and management of anaerobic pathogens in maternal-fetal infections, we recommend: routine anaerobic culture of placental and amniotic specimens in cases of suspected chorioamnionitis; species-level identification of all Clostridium isolates rather than nonspecific reporting; anaerobic blood cultures from neonates exposed to maternal chorioamnionitis, obtained at delivery before antimicrobial initiation; and empiric anaerobic coverage (metronidazole or clindamycin) for all exposed neonates.

Enhanced microbiological investigation of obstetric infections can unmask *C. baratii* and other underrecognized anaerobes, improving both detection rates and clinical outcomes in maternal-fetal medicine.

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