

Letter to the Editor

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An unexpected pathogen in the amniotic fluid of a patient with chorioamnionitis

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Sir,

Chorioamnionitis can be defined as inflammation of the fetal membranes, usually as a result of infection of the chorioamnion and amniotic fluid [1]. Microbiological confirmation of chorioamnionitis is performed by culturing an amniotic fluid sample, which is the gold standard and most specific test for detecting intraamniotic infection [2].

Microorganisms can reach the amniotic fluid through various pathways. Chorioamnionitis is often caused by bacteria from the lower genital tract that can infect the amniotic fluid through an ascending mechanism [3,4].

Although rare pathogens have been reported to cause chorioamnionitis, little is known about the infection when caused by unusual bacteria. This study aims to provide new evidence on the clinical management and outcomes of chorioamnionitis caused by a pathogen that has not been previously reported in the literature as a causative agent of intraamniotic infection.

The following case involves a pregnant woman who presented to the emergency unit with increased vaginal discharge and hypogastric pain. The patient had a significant past medical history of three previous miscarriages. At the time of admission, she was afebrile and neither metrorrhagia nor amenorrhea were present. The patient was admitted to the gynecology unit where urine and vaginal discharge samples were collected. Both urinary tract infections and sexually transmitted infections (STIs) were ruled out. Further exploration led to a diagnosis of threatened preterm labor due to a prolapsed amniotic sac. As a result, the patient received a first cycle of atosiban, combined with magnesium sulfate, betamethasone, and prophylactic antibiotic therapy consisting of gentamicin (240 mg/24h for 6 days), ampicillin (1 g/6h for 7 days), and azithromycin (500 mg/12h for 6 days).

Shortly after, the patient presented amenorrhea and was diagnosed with preterm premature rupture of membranes. Blood analysis results showed leukocytosis but no other hematologic alterations were observed. An amniocentesis was performed due to suspicion of chorioamnionitis. The amniotic fluid sample was tested for sexually transmitted microorganisms (Chlamydia trachomatis, Mycoplasma genitalium, Neisseria gonorrhoeae and Trichomonas vaginalis) and genital mycoplasmas (Mycoplasma hominis, Ureplasma urealyticum and Ureplasma parvum) using a real-time polymerase chain reaction (AllplexTM STI Essential Assay, Seegene®, Seoul, Republic of Korea). The results were negative for all tested microorganisms and mycoplasmas. Amniotic fluid was cultured in both aerobic and anaerobic media. Aerobic media included blood agar with ram blood and chocolate agar from Biomerieux, USA, and was incubated for 5 days at 37°C with 5% CO₂. Anaerobic media included anaerobic blood agar and anaerobic phenylethyl alcohol agar and was incubated for 5 days at 37°C in an anaerobic atmosphere. After 24 hours of incubation at 37°C with 5% CO₂, small colonies with abundant and pure growth were observed in blood agar. The microorganism was identified as Lactobacillus rhamnosus using MALDI-TOF (Bruker Daltonik GmbH, Bremen, Germany) with a score of 2.14. Antibiotic prophylaxis was administered again using the same combination of antimicrobials and dosage as previously mentioned.

Despite these results, labor was not induced, and a clinical approach of watchful waiting was decided. Finally, two weeks after the amniocentesis, at 30 weeks of gestation, the patient went into labor. After delivery, microscopic examination of the placenta and umbilical cord confirmed acute histologic chorioamnionitis and funisitis. To the best of our knowledge, this is the first reported case of chorioamnionitis caused by *L. rhamnosus* in the literature.

While *Lactobacillus* species dominate healthy vaginal microbiota [5], they are seldom reported as causative agents of chorioamnionitis [1,6,7]. In a study conducted by Lannon SMR

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et al., *Lactobacillus* species were detected in the chorioamnion of pregnant women without chorioamnionitis or funisitis, suggesting that these microorganisms could be part of the normal microbial community that develops in the chorioamnion during labor [8].

Multiple studies have identified several risk factors for chorioamnionitis, including longer duration of membrane rupture (including premature rupture of membranes), prolonged labor, nulliparity, multiple vaginal exams, and colonization with group B *Streptococcus* or genital mycoplasmas [1,9]. In this case, the only reported risk factor was premature rupture of membranes, which may have allowed the *L. rhamnosus* to progress through an ascending mechanism from the genital microbiota to the amniotic fluid.

According to the literature, clinical management of chorioamnionitis should involve giving birth and administering broad-spectrum parenteral antibiotics, such as a combination of ampicillin and gentamicin [1,2,10]. In this case, delivery was not induced in an attempt to prolong gestation and maximize fetal development. Regarding antimicrobial treatment, the patient was administered ampicillin and gentamicin. Additionally, azithromycin was prescribed to cover genital mycoplasmas as soon as preterm premature rupture of membranes was diagnosed.

In conclusion, chorioamnionitis is a common infection during pregnancy and a significant risk factor for adverse maternal and perinatal outcomes. Although *Lactobacillus* species dominate the vaginal microbiota, they are rarely reported as causative agents of chorioamnionitis. This study provides new evidence on the clinical management and outcomes of chorioamnionitis caused by these bacteria. However, further studies are needed.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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