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Therapeutic Challenges of Co-Infections with Chlamydia Trachomatis and Urogenital Mycoplasmas during Pregnancy and Obstetric Consequences: A Report of Three Cases

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Abstract

Introduction: *Urogenital mycoplasmas* and *Chlamydia trachomatis* are common pathogens in women of reproductive age and may act synergistically during pregnancy. Their management is challenging, because the most effective antibiotics, such as tetracyclines and fluoroquinolones, are contraindicated due to fetal toxicity, while resistance to safer alternatives like azithromycin is increasing. These infections are associated with miscarriage, preterm delivery, and neonatal respiratory complications. Case presentation: We report three cases of pregnant women in Cameroon co-infected with *Chlamydia trachomatis* and *Urogenital mycoplasmas*. The first patient, presenting with threatened miscarriage at 21 weeks, had *Ureaplasma urealyticum* resistant to azithromycin but sensitive to tetracyclines; azithromycin was nevertheless prescribed to target *Chlamydia trachomatis*, with good maternal and neonatal outcomes. The second patient, at 28 weeks, presented with threatened preterm labor due to polymicrobial infection (*Chlamydia trachomatis*, *Mycoplasma hominis*, and *Ureaplasma urealyticum*) and improved under azithromycin therapy, delivering at term a healthy neonate. The third patient, diagnosed at 9 weeks with *Chlamydia trachomatis*, *Mycoplasma hominis*, *Ureaplasma urealyticum*, and candidiasis, later developed threatened miscarriage and underwent emergency cesarean delivery at 35 weeks for fetal distress. The newborn developed respiratory distress and neonatal infection, illustrating adverse perinatal consequences. In all cases, sexual partners were empirically treated to prevent reinfection. Discussion: These cases highlight the therapeutic dilemmas posed by co-infections during pregnancy. Vaginal dysbiosis (type III-IV Doderlein flora) was

consistently present, favoring pathogen proliferation. The link between maternal colonization and neonatal morbidity underscores the need for improved detection and management. Conclusion: Targeted screening and timely treatment of urogenital mycoplasmas and Chlamydia trachomatis during pregnancy, though controversial, may prevent severe obstetric and neonatal complications in high-burden settings.

Keywords

Chlamydia Trachomatis, Urogenital Mycoplasmas, Pregnancy, Neonatal Infection, Case Report, Cameroon

1. Introduction

Urogenital mycoplasmas are the smallest organisms capable of multiplying outside of a living cell [1]. Four species of mycoplasmas have been implicated in female urogenital pathology: Mycoplasma hominis, Ureaplasma urealyticum, Ureaplasma parvum, and Mycoplasma genitalium [2]. Mycoplasmas are present in a commensal state, notably Ureaplasma urealyticum, with a prevalence ranging from 40% to 80% among asymptomatic women [3]. A study in China revealed a prevalence of 48.81% for Ureaplasma urealyticum among patients attending gynecology or obstetrics consultations [4]. The colonization of vaginal and cervical mucosa by Mycoplasma hominis is around 20% to 30% globally [5]. The global prevalence of Mycoplasma genitalium is lower, approximately 0.3% to 1%, but in the West region of Cameroon, it is higher at 5.2% [6] [7]. Colonization of the genital tract by genital mycoplasmas is strongly influenced by age, black race, low socioeconomic status, risky sexual behaviors, hormonal changes, contraceptive use, and it increases during pregnancy [8]-[10]. Several studies document the significant frequency of co-infections with Chlamydia trachomatis and urogenital mycoplasmas in pregnant women. A study in Japan revealed that 9.2% of women had co-infections of *C. trachomatis* and Ureaplasma (mainly *U. parvum*), suggesting a possible synergy or shared habitat within the genital mucosa [11]. In general, bacterial resistance to antibiotics is a major threat to reducing infections in medical practice worldwide. In the case of Chlamydia trachomatis and urogenital mycoplasmas, an increase in antibiotic resistance has been observed in recent years. The difficulty in treating certain mycoplasma strains poses a real problem during pregnancy, as many compounds are harmful to the fetus. This highlights a pathogenic complexity to consider, particularly in screening strategies and the management of these infections during pregnancy.

2. Case Presentation

2.1. Case No. 1

A 29-year-old patient, G4P1021, with a history of two consecutive spontaneous

miscarriages at 8 weeks of gestation, and the first child's birth weight was 3800 g. She was seen in consultation for intermittent pelvic pain resembling uterine contractions that had been present for a week, with no other symptoms noted, during a pregnancy of 21 weeks and 3 days of amenorrhea. No medication had been initiated. Upon physical examination, uterine activity was observed at a rate of two moderate-intensity uterine contractions in 10 minutes. There was no per vaginal discharge from the endocervix, the cervix appeared bluish, and there were no pathological leukorrhea. A vaginal examination revealed pain upon cervical mobility, sensitivity of the Douglas pouches, and a clean glove.

The diagnosis was made of a threatened late miscarriage. The biological workup revealed a type IV Doderlein flora (characterized by an almost complete depletion of protective lactobacilli, reflecting a profound dysbiosis), an active infection of Chlamydia trachomatis was diagnosed based on positive IgM serology; and the presence of urogenital mycoplasmas, specifically Ureaplasma urealyticum, which was sensitive to minocycline, gatifloxacin, sparfloxacin, and josamycin. The rest of the prenatal workup was normal. In-hospital management included administering an antispasmodic (phluroglucinol 50 mg, three times a day) and azithromycin 1500mg per week for three weeks (empirical treatment was also administered to the partner as part of comprehensive management to prevent reinfection). The treatment resulted in the disappearance of pelvic pain. The patient delivered at 41 weeks and 1 day with oxytocin stimulation, without any complications, a male infant weighing 2900g, with APGAR scores of 9-10-10 and without complications. The postpartum course was normal, with no neonatal infection. It should be noted that the antibiotics to which the Ureaplasma urealyticum strain was sensitive were incompatible with pregnancy, and treatment was deferred until after delivery.

2.2. Case No. 2

A 30-year-old patient, G2P2002, with no significant medical history, presented to consultation for intermittent pelvic pain resembling uterine contractions that had been occurring for three days, with no other associated symptoms, during a pregnancy of 28 weeks and 5 days of amenorrhea. Moderate uterine activity was observed. There was no per vaginal discharge from the endocervix, the cervix appeared bluish, and there were no leukorrhea lining the vaginal walls. On vaginal examination, the cervix was long, posterior, and closed, with intermediate consistency, pain upon mobilization, sensitivity of the Douglas pouches, and a glove contaminated with thick, yellowish, abundant, and foul-smelling leukorrhea. Given this clinical presentation, a diagnosis of a mild threat of preterm delivery was made. A biological workup indicated the destruction of the vaginal flora and a polymicrobial infection. with Chlamydia trachomatis (positive IgM serology confirmed), Mycoplasma hominis, and Ureaplasma urealyticum, all sensitive to minocycline, doxycycline, gatifloxacin, ofloxacin, josamycin, roxithromycin, and azithromycin. The prenatal workup carried out a month prior to the consultation was normal.

Management involved outpatient treatment of urogenital mycoplasma and Chlamydia trachomatis infections with azithromycin at 500 mg/day for three consecutive days over three weeks (the partner was also treated) and an antispasmodic to alleviate pelvic pain. The patient's symptoms resolved, and she gave birth at 38 weeks and 2 days to a female infant weighing 3570 g, with APGAR scores of 8-9-10, with no neonatal infection reported.

2.3. Case No. 3

A 27-year-old patient with AS hemoglobin electrophoresis, without other significant medical history, G1P0101, presented for consultation due to abundant, itchy leukorrhea at 9 weeks and 3 days of gestational amenorrhea. During the gynecological exam, inspection of the vulva revealed lesions from desquamation and scratching, a long posterior cervix with a rigid consistency that was closed, and vaginal walls lined with abundant, clumpy, non-foul-smelling leukorrhea. Vaginal swabs indicated a type III Doderlein flora imbalance (corresponds to a depleted flora dominated by anaerobic bacteria), and the presence of Candida albicans sensitive to nystatin and amphotericin B. The search for urogenital mycoplasmas revealed Mycoplasma hominis and Ureaplasma urealyticum sensitive to josamycin, levofloxacin, azithromycin, roxithromycin, and clarithromycin. Chlamydia was diagnosed based on positive IgM. We initiated treatment with azithromycin (the partner was also treated) and nystatin.

A week later, at 10 weeks and 3 days, the patient was seen in the emergency department for sudden-onset per vaginal bleeding without other associated signs. A vaginal examination revealed abundant non-foul-smelling leukorrhea with blood streaks. An emergency ultrasound showed an intrauterine monoembryonic pregnancy that was evolving, with the ultrasound age matching the theoretical age, but with a trophoblastic detachment. We concluded a diagnosis of threatened early miscarriage, followed by hospitalization with strict rest, 200 mg of progesterone twice daily intravaginally, nystatin, and 500 mg of azithromycin daily for three consecutive days over three weeks. The treatment was satisfactory with an improvement of symptoms.

At 35 weeks of gestation, the patient was admitted to the emergency department again for moderate per vaginal fluid leakage associated with pelvic pain resembling uterine contractions. On physical examination, the Valsalva test was negative, and the cervix was posterior, 2 cm dilated, with an intermediate consistency and intact membranes. Paraclinical tests for malaria and urinary infection were negative, and an ultrasound showed an evolving monofetal pregnancy at an ultrasound gestational age of 35 weeks and 5 days, with an estimated fetal weight of 2776 g, oligohydramnios, and no nuchal cord. Three hours after admission, fetal tachycardia of 170-184 bpm was reported, and an emergency cesarean section was performed, resulting in the delivery of a female infant with a birth weight of 2560 g and APGAR scores of 7-8-10. The newborn was admitted to neonatology and developed a neonatal infection with respiratory distress 24 hours later.

3. Discussion

Mycoplasma hominis and Ureaplasma urealyticum are commensals of the lower urogenital tract. They can be transmitted from mother to fetus through several pathways: in utero, either transplacentally or via intrauterine ascent by colonizing the amniotic fluid (even with intact membranes), with transmission rates up to 55% in preterm infants. During delivery, as the infant passes through the birth canal, resulting in neonatal colonization of around 45% of full-term newborns, regardless of the mode of delivery. After birth, through postnatal or nosocomial transmission. Colonization is particularly frequent among preterm infants with low birth weight, up to 89% in newborns weighing less than 1000g [3] [12] [13].

A 2020 Israeli study conducted on pregnant women revealed significant colonization rates in newborns, particularly in cases of premature membrane rupture [14]. In China, a large hospital-based neonatal cohort showed that 7.7% of newborns were infected with Ureaplasma urealyticum, with a significantly higher prevalence among preterm infants and those born vaginally [15]. Additionally, a German study conducted on infants less than 32 weeks of gestational age reported respiratory colonization by *Ureaplasma* spp. in 25.8% of them. This colonization was strongly associated with neonatal respiratory complications, especially bronchopulmonary dysplasia, and its likelihood was increased by 7.8 times in children of mothers who were themselves colonized [16]. In our third case, the neonatal infection and subsequent respiratory distress are consistent with this evidence, supporting the hypothesis that maternal colonization with mycoplasmas and Chlamydia trachomatis directly contributed to the adverse neonatal outcome. This demonstrates the significant ongoing perinatal risk associated with urogenital mycoplasmas and, importantly, that co-infections with Chlamydia trachomatis can have a synergistic effect, worsening fetal complications.

The treatment of these infections in pregnant women remains particularly complex. Indeed, most of the antibiotics most effective against these germs, such as tetracyclines (doxycycline, minocycline) and fluoroquinolones (levofloxacin, gatifloxacin, sparfloxacin), are contraindicated during pregnancy due to their fetal toxicity [17]. Macrolides, such as erythromycin or azithromycin, are the most frequently used options as they pose less risk to the fetus. However, recent studies have reported increasing resistance to these drugs, limiting their therapeutic effectiveness in a significant number of cases. A 2023 Chinese study reported an azithromycin resistance rate of 9.9% among pregnant women, with clinical consequences including miscarriages and preterm deliveries [18]. Alternatives like josamycin or pristinamycin show a good in vitro activity profile, but their use in pregnant women is less documented and sometimes difficult to access depending on the context [19]. In our first case, azithromycin was administered despite the identified resistance of *Ureaplasma urealyticum*, specifically to target the concomitant Chlamydia trachomatis infection, for which azithromycin remains the recommended first-line therapy during pregnancy. This choice reflects the therapeutic compromises often necessary in such co-infections.

The increase in bacterial vaginal infections and the physiological changes associated with pregnancy may be caused by weakened cervical defenses. In our patients, the vaginal flora was classified as type III (depleted flora dominated by anaerobic bacteria) or type IV (almost complete absence of protective lactobacilli, indicating severe imbalance). Such dysbiosis is known to favor the proliferation of opportunistic pathogens including urogenital mycoplasmas. In a study in Mali, Guimdo *et al.* demonstrated that the imbalance of flora is proportional to the proliferation of urogenital mycoplasmas [20]. This could be explained by multiple sexual partners and insufficient or erroneous knowledge about good intimate hygiene practices.

Partner treatment was systematically undertaken in our cases, as part of comprehensive management aimed at preventing reinfection. This measure is crucial, since untreated partners constitute an important reservoir for recurrent infections.

Managing co-infections with Chlamydia trachomatis and urogenital mycoplasmas during the prenatal period requires an approach based on clinical risk and the microbiological context. While systematic screening could expose to the risk of overtreating asymptomatic carriers whose clinical significance remains debated, in our setting where genital infections are a major driver of obstetric complications the benefits of targeted screening and timely treatment appear to outweigh these concerns. Developing accessible diagnostic tools and conducting clinical trials to evaluate safe therapeutic alternatives during pregnancy are essential to improve management and prevent fetal complications.

4. Conclusion

Urogenital mycoplasmas, long considered mere commensals of the female genital tract, are now revealing their potential role in certain obstetric and neonatal complications. Diagnosing these infections during pregnancy remains outdated in resource-limited countries. They are not routinely tested for during pregnancy because, according to World Health Organization recommendations, testing for mycoplasmas is not included in the mandatory examinations for pregnant women. Additionally, these challenges are exacerbated by the asymptomatic nature of these infections, the high cost of tests, and their limited or almost nonexistent availability in healthcare facilities at the operational level, where patient flow is highest. Therapeutically, options are limited by the contraindications of the most effective antibiotics in pregnant women, and the growing resistance to available treatments further complicates management. Faced with these challenges, especially in our context, it would be wise to integrate testing for urogenital mycoplasmas into prenatal check-ups to screen as many cases as possible. It is also imperative to strengthen research on safe antibiotic alternatives during the prenatal period to prevent associated fetal and neonatal complications.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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