Case Report

Pregnancy Complicated by Tuberculous Lymphadenitis and PPROM Following Contraceptive Failure: A Case Report

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Abstract
Introduction: Tuberculous lymphadenitis during pregnancy is associated with increased risks of adverse maternal and perinatal outcomes. Rifampin-based anti-tuberculosis therapy may reduce the effectiveness of hormonal contraception and lead to unintended pregnancy.
Case presentation: A 25 – 26-week pregnant woman (G2P1A0) was diagnosed with preterm premature rupture of membranes (PPROM) and threatened preterm labor. The pregnancy occurred despite levonorgestrel implant use during rifampicin therapy for tuberculous lymphadenitis. Conservative management with antibiotics, tocolytics, corticosteroids, and continuation of anti-tuberculosis therapy was initiated. However, the patient delivered an extremely preterm infant (820 g) through vaginal birth after cesarean (VBAC). The neonate died on day 14 due to complications of prematurity, while the mother recovered well after completing anti-tuberculosis treatment.
Conclusion: This case highlights contraceptive implant failure during rifampin therapy, resulting in unintended pregnancy and severe obstetric complications, including preterm birth. Tuberculosis screening, appropriate contraceptive counseling, and multidisciplinary management are essential to optimize maternal and neonatal outcomes.

Keywords: Contraceptive implant failure; pregnancy; preterm prelabor rupture of membranes; rifampicin; tuberculous lymphadenitis

Kehamilan yang Komplikasi oleh Limfadenitis Tuberkulosis dan PPROM setelah Kegagalan Kontrasepsi: Laporan Kasus

Abstrak
Pendahuluan: Limfadenitis tuberkulosis pada kehamilan berhubungan dengan peningkatan risiko komplikasi maternal dan perinatal. Terapi berbasis rifampisin dapat menurunkan efektivitas kontrasepsi hormonal dan menyebabkan kehamilan yang tidak direncanakan.
Laporan kasus: Seorang wanita hamil 25 – 26 minggu (G2P1A0) didiagnosis dengan ketuban pecah dini preterm dan partus prematurus imminens. Kehamilan terjadi meskipun menggunakan implan levonorgestrel selama terapi rifampisin untuk limfadenitis tuberkulosis. Penatalaksanaan konservatif berupa antibiotik, tokolitik, kortikosteroid, dan terapi anti-tuberkulosis diberikan. Namun, pasien melahirkan bayi prematur ekstrem (820 g) secara VBAC. Bayi meninggal pada hari ke-14 akibat komplikasi prematuritas, sedangkan kondisi ibu membaik setelah menyelesaikan terapi.
Kesimpulan: Kasus ini menunjukkan kegagalan implan kontrasepsi selama terapi rifampisin yang menyebabkan kehamilan tidak direncanakan dan persalinan prematur. Skrining tuberkulosis, konseling kontrasepsi, dan perawatan multidisiplin sangat penting.

Kata kunci: Kehamilan; kegagalan implan kontrasepsi; ketuban pecah dini preterm; limfadenitis tuberkulosis; rifampisin

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Introduction

Tuberculosis (TB) remains the leading infectious cause of mortality worldwide and continues to pose significant risks during pregnancy. Although the incidence of active TB is relatively low in some countries, such as the United States, its presence during pregnancy is associated with markedly increased adverse outcomes, including higher maternal morbidity, miscarriage, preterm birth, low birth weight, and perinatal mortality. A substantial proportion of the global population carries latent TB infection, with only a minority progressing to active disease; however, predicting this progression remains challenging. Therefore, screening high-risk individuals, particularly during antenatal care, is essential for early detection and prevention of complications. Pregnant women, especially those from high-burden regions or with known risk factors, are a key population for targeted TB screening and management.[1,2]

Globally, TB during pregnancy remains underrecognized, with significant gaps in epidemiological data. Evidence consistently shows that active TB during pregnancy is linked to worse maternal and neonatal outcomes, particularly when diagnosis is delayed or treatment is inadequate. Importantly, the risks of untreated TB far outweigh those of pharmacological therapy, underscoring the need for timely management. Additionally, although rare, congenital TB can occur via transplacental transmission or exposure at birth and carries a high mortality rate. These findings underscore the importance of early diagnosis, appropriate treatment, and multidisciplinary care to improve outcomes for both the mother and the fetus.[1]

In many TB-endemic settings, women of reproductive age rely on progestin-only contraceptive implants after childbirth. Rifampin, a key component of first-line anti-TB therapy, potently induces cytochrome P450 3A4 and drug transporters, markedly reducing systemic exposure to hormonal contraceptives. Pharmacokinetic and pharmacodynamic data from implant users show that short-course rifampin substantially lowers serum progestin concentrations and is associated with increased luteal activity and ovulation in a subset of women.[3] Previous clinical case reports have described contraceptive implant failure in women treated with rifampin for TB, underscoring that the combination of enzyme-inducing therapy and long-term implant use can result in unintended pregnancy.[4]

Furthermore, TB may contribute to the development of preterm premature rupture of membranes (PPROM) through its role as a chronic systemic inflammatory disease. Evidence shows that PPROM is strongly associated with inflammatory pathways, particularly involving cytokines such as interleukin-6 (IL-6), which are elevated in both maternal serum and amniotic fluid in cases of membrane rupture.[5] Similarly, TB infection induces a persistent inflammatory response characterized by increased pro-inflammatory cytokines, including IL-6 and tumor necrosis factor-alpha (TNF-α). This sustained inflammatory state can weaken the structural integrity of fetal membranes by promoting collagen degradation and extracellular matrix breakdown. Molecular studies further support that inflammation and immune activation disrupt the amniotic membranes and contribute directly to PPROM pathogenesis.[6] Therefore, TB-related systemic inflammation provides a biologically plausible mechanism linking the infection to membrane fragility and rupture.

In addition to systemic inflammation, TB may indirectly increase the risk of PPROM by altering maternal immune function and predisposing the patient to secondary infections. PPROM has been closely linked to intrauterine infection, cervical microbiome imbalance, and fetal inflammatory response

syndrome.[7] Chronic infectious conditions such as TB can impair host immunity, thereby increasing susceptibility to ascending genital tract infections, well-established triggers of PPROM. Supporting evidence indicates that infection-driven inflammatory responses, including elevated IL-6 and IL-8 levels, play a central role in weakening fetal membranes.[8] Epidemiological data further emphasize that maternal systemic diseases and infections are important contributors to PPROM.[9] Taken together, although TB is not traditionally classified as a direct cause of PPROM, current evidence supports its role as an indirect risk factor through mechanisms of chronic inflammation, immune dysregulation, and increased vulnerability to intrauterine infection.

Several studies from Indonesia and other high-burden settings have reported that maternal TB is associated with an increased risk of PPROM and preterm labor, conditions that substantially contribute to neonatal morbidity and mortality.[10] Data from tertiary centers in Indonesia, including Dr. Soetomo Hospital, indicate that TB during pregnancy is often accompanied by obstetric complications, leading to higher perinatal morbidity and mortality. These findings underscore the need for early TB detection and integrated obstetric–pulmonology management to improve outcomes for both mother and fetus.[11]

This case report describes the clinical course of a pregnant woman with tuberculous lymphadenitis undergoing rifampin-based anti-TB treatment who experienced contraceptive implant failure. This failure led to an unintended pregnancy that was further complicated by PPROM and imminent preterm labor (*partus prematurus imminens*), ultimately resulting in an extremely preterm delivery and an adverse perinatal outcome. This case highlights the critical importance of TB screening in pregnancy, careful contraceptive counseling for women receiving rifampin, and multidisciplinary management to reduce maternal and perinatal morbidity and mortality.[12,13]

Case Presentation

A 25–26-week pregnant woman (G2P1A0) presented to the emergency department with a four-hour history of intermittent uterine contractions and two hours of vaginal fluid leakage. Fetal movements were intact. She also reported a one-week history of greenish vaginal discharge with pruritus but without a foul odor. She denied fever, dysuria, or abdominal trauma. Notably, the patient conceived while actively using a levonorgestrel contraceptive implant (two rods, 75 mg). The device was removed after pregnancy was confirmed in September 2024, representing a definitive contraceptive failure.

The patient's medical history was significant for tuberculous lymphadenitis, diagnosed in August 2024 by fine-needle aspiration biopsy (FNAB). The biopsy showed chronic granulomatous lymphadenitis consistent with tuberculosis, with no evidence of malignancy. At presentation, she was in the continuation phase of anti-tuberculosis treatment, receiving a fixed-dose combination regimen (2FDC: rifampicin 150 mg and isoniazid 75 mg, three tablets daily). Her obstetric history was notable for a prior cesarean section performed two years earlier secondary to premature rupture of membranes. Consequently, the current pregnancy was classified as high-risk due to both her prior cesarean delivery and ongoing tuberculosis treatment.

Upon admission to the labor ward, the patient was in good general condition with stable vital signs. Obstetric examination showed a fundal height at the umbilicus, a fetal heart rate of 142 beats per minute, and uterine contractions every ten minutes. A vaginal examination noted 2 cm of cervical

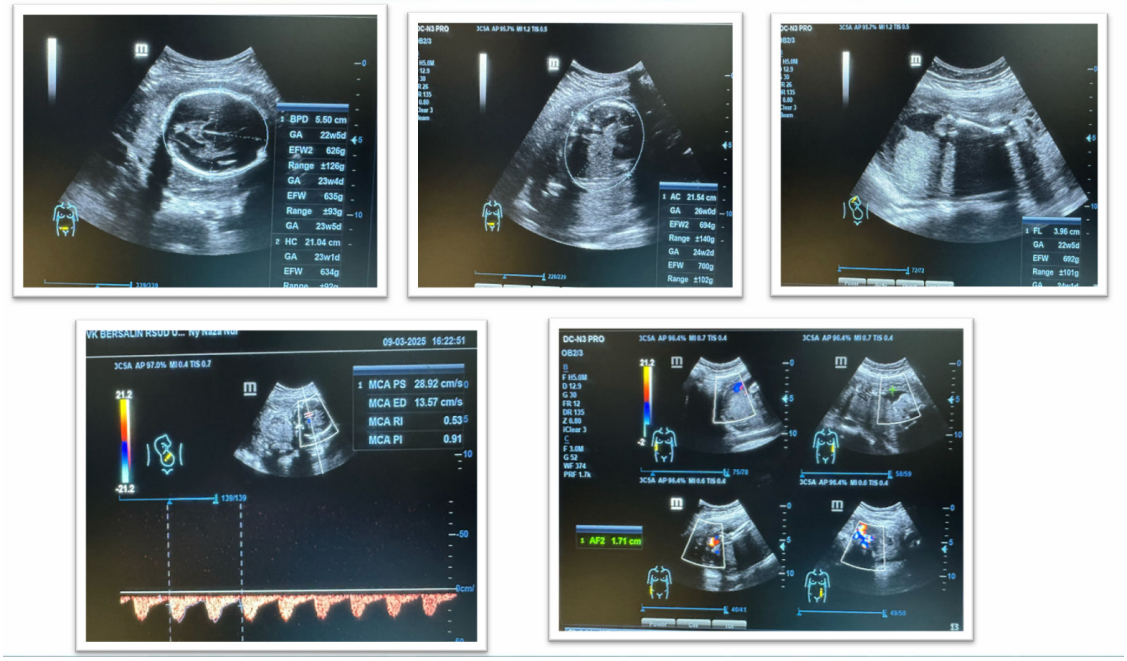


Figure 1 Obstetric Ultrasound

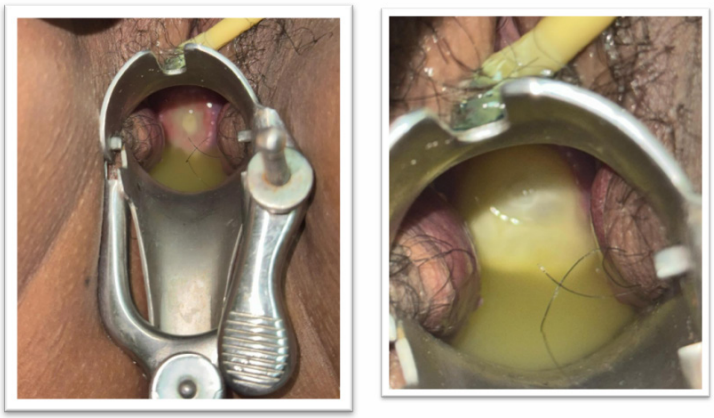


Figure 2 Pooling Test (+)

dilation and 25% effacement. A sterile speculum examination showed pooling of amniotic fluid in the posterior fornix and a positive nitrazine test, confirming preterm premature rupture of membranes (PPROM). Cardiotocography (CTG) was reassuring (Category I). Obstetric ultrasound revealed a single live intrauterine fetus in cephalic presentation without major congenital anomalies. The estimated fetal weight was 600–670 grams, and severe oligohydramnios was noted, with an amniotic fluid index of 1.6–1.7 cm.

Laboratory investigations revealed mild anemia, with a hemoglobin level of 10.6 g/dL and a hematocrit of 30.5%, likely secondary to the physiological hemodilution of pregnancy combined with chronic illness. The leukocyte count was elevated (15,100/ µL), indicative of an inflammatory or infectious process. This leukocytosis was likely multifactorial in etiology, driven by both PPROM and the underlying tuberculous lymphadenitis. Despite this elevated white

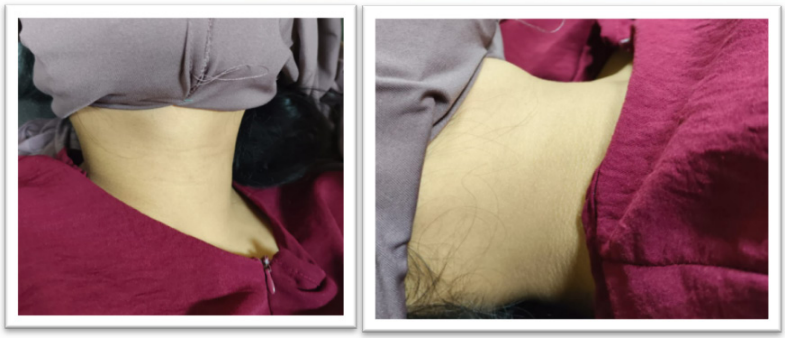


Figure 3 Enlargement of Cervical Lymph Nodes

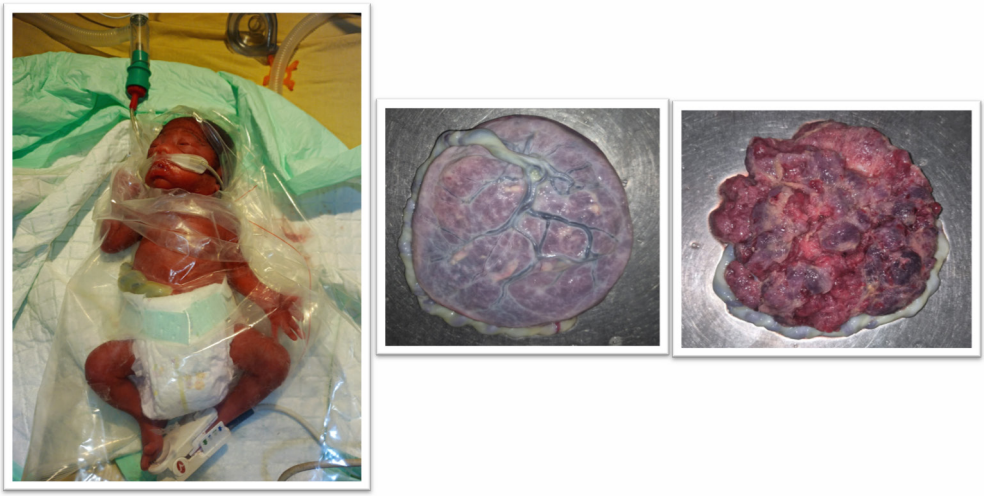


Figure 4 Newborn at Birth and Placenta

blood cell count, the absence of fever and stable hemodynamics suggested no overt systemic infection or sepsis at presentation. Platelet count and coagulation parameters were within normal limits, demonstrating preserved hemostatic function. Random blood glucose levels were normal, and urinalysis showed no significant abnormalities, effectively ruling out a urinary tract infection as a trigger for preterm labor.

The patient was formally diagnosed with a 25-week intrauterine pregnancy complicated by PPROM (less than 12 hours duration) and imminent preterm labor, alongside comorbid tuberculous lymphadenitis on rifampicin-based therapy and a history of a previous cesarean section.

Conservative management was initiated immediately to prolong the pregnancy and optimize fetal outcomes. Maternal rehydration was achieved with intravenous fluids (1000 mL/day). To mitigate the risk of ascending intrauterine infection associated with PPROM, empirical intravenous ceftriaxone (1 g every 12 hours) was administered. For fetal lung maturation, intramuscular dexamethasone (6 mg every 12 hours for two days) was administered. Tocolytic therapy with a stepwise nifedipine regimen was initiated to suppress uterine contractions (30 mg-30 mg-20 mg on day one; 20 mg-20 mg-20 mg on day two; tapered to 20 mg-20 mg-0 mg on day three). Her anti-tuberculosis therapy was continued per pulmonology recommendations, alongside routine antenatal supplementation (calcium, iron, and vitamin D). Maternal vital signs, particularly temperature, were closely

Table 1 Clinical Timeline of the Case

Time / Gestational Age	Clinical Events & Findings	Diagnostic Evaluation	Intervention / Management
Aug 2024 (pre-pregnancy)	Enlarged cervical lymph nodes	FNAB → chronic granulomatous lymphadenitis consistent with TB	Initiated anti-TB therapy (Rifampicin + Isoniazid, 2FDC)
Sep 2024 (Early pregnancy)	Pregnancy confirmed despite contraceptive implant	Positive pregnancy test	Removal of levonorgestrel implant (contraceptive failure suspected)
25–26 weeks GA (Admission)	Uterine contractions (4 hours), fluid leakage (2 hours), vaginal discharge	Speculum: pooling (+), Nitrazine (+); CTG Category I; USG: oligohydramnios, EFW 600–670 g	Diagnosis: PPROM + threatened preterm labor
Day 1 (Hospitalization)	Stable maternal condition	Hb 10.6 g/dL, leukocytes 15,100/µL	IV fluids, ceftriaxone 1 g q12h, nifedipine (tocolysis), dexamethasone (6 mg q12h)
Day 2–3	Persistent contractions	Clinical monitoring	Continuation of tocolysis, antibiotics, corticosteroids, anti-TB therapy
Day 3	Increased contractions, bloody show → active labor	Cervical dilation progressed to full	Spontaneous vaginal delivery (VBAC)
Birth	Female neonate, BW 820 g, extreme prematurity	APGAR 5/6/7	NICU admission (ventilator, surfactant, antibiotics)
NICU Day 14	Neonatal deterioration	Clinical course consistent with prematurity complications	Neonatal death
Postpartum follow-up (May 2025)	Resolution of lymphadenopathy	Clinical evaluation normal	Anti-TB therapy completed, patient recovered

monitored every four hours.

Supportive therapy was continued with routine antenatal supplementation: 500 mg of calcium twice daily, 1 g of iron once daily, and 5000 IU of vitamin D once daily. Maternal temperature was closely monitored every four hours to detect early signs of infection. This multimodal management strategy was implemented to optimize maternal condition, reduce the risk of infection, and improve neonatal outcomes in the setting of preterm premature rupture of membranes.

The differential diagnosis of preterm labor in this case includes Braxton–Hicks contractions, non-obstetric causes of abdominal pain, and placental abruption. Braxton–Hicks contractions are typically irregular and infrequent, without progressive cervical dilatation, making them less likely in this patient, who had increasing contraction frequency with cervical changes. Non-obstetric causes of abdominal pain, such as gastroenteritis or urinary tract infection, may present with abdominal discomfort; however,

these conditions are usually accompanied by gastrointestinal or urinary symptoms and were not supported by the clinical and laboratory findings in this case. Placental abruption should also be considered, as it may present with abdominal pain and uterine activity, but it is commonly associated with vaginal bleeding, uterine tenderness, and fetal distress, none of which were evident in this patient. Therefore, based on the clinical presentation and supporting findings, these conditions were considered less likely than preterm labor associated with preterm premature rupture of membranes.

Despite conservative measures, the patient developed increasing uterine contractions and bloody show on the third day of hospitalization. She entered the active phase of labor, rapidly progressing from 4 cm to full cervical dilation. A spontaneous vaginal birth after cesarean (VBAC) was achieved. She delivered a female neonate weighing 820 grams and measuring 32 cm in length, with APGAR scores of 5, 6, and 7 at 1, 5, and 10 minutes, respectively.

The neonate was admitted to the neonatal intensive care unit (NICU) for extreme prematurity, extremely low birth weight, and respiratory distress syndrome (suspected hyaline membrane disease). Neonatal management included mechanical ventilation, surfactant administration, and broad-spectrum antibiotics. Despite optimal management, the neonate died after 14 days in the NICU.

During a routine pulmonology follow-up on May 15, 2025, after completing her anti-tuberculosis regimen, the patient reported complete resolution of bilateral submandibular lymphadenopathy. She denied pain, fever, or further weight loss, though she noted residual mild nausea and decreased appetite. Physical examination confirmed good general condition and an interval weight gain to 57.3 kg (up from previous recordings of 54–56 kg). No residual cervical lymphadenopathy was palpable. The tuberculous lymphadenitis was deemed fully resolved. Her anti-tuberculosis therapy was discontinued, and she was prescribed a daily Curcuma supplement with instructions for routine follow-up.

Discussion

This report discusses the case of a patient with tuberculous lymphadenitis undergoing regular anti-tuberculosis therapy with a fixed-dose combination regimen (2FDC) containing rifampicin. Concurrently, the patient was using a levonorgestrel contraceptive implant (two rods, 75 mg). However, the effectiveness of this contraceptive method was compromised by a well-documented pharmacokinetic interaction. Rifampicin, a potent inducer of hepatic cytochrome P450 enzymes (specifically CYP3A4), accelerates the metabolism of levonorgestrel, thereby reducing its serum concentration and negating its contraceptive efficacy.

As a result, the patient experienced contraceptive failure, leading to an unintended pregnancy. This pregnancy was subsequently classified as high-risk due to multiple converging factors: an ongoing tuberculosis infection, exposure to anti-tuberculosis therapy, and a prior cesarean section. These factors significantly increased the risk of adverse maternal and fetal outcomes, clinically manifesting as PPROM and *partus prematurus imminens*, which ultimately culminated in extreme prematurity and neonatal death. This case highlights the critical importance of counseling women of reproductive age about potential drug–contraceptive interactions, particularly in the context of rifampicin-based therapy.

Women with systemic conditions such as TB, which inherently increase health risks during pregnancy, have a definitive need to avoid unintended conceptions. However, pharmacokinetic studies consistently

demonstrate that rifampicin significantly reduces systemic exposure to progestins by accelerating hepatic metabolism. While the exact threshold for contraceptive failure varies individually, these reductions are clinically significant and frequently impair ovulation suppression. By contrast, other rifamycins, such as rifabutin, exhibit milder interactions, and rifaximin has minimal effects due to its lack of systemic absorption.[3,14]

The literature supports these pharmacokinetic shifts. A two-week course of rifampin in etonogestrel (ENG) implant users results in profound pharmacodynamic changes, reducing serum ENG levels below the threshold required for ovulation suppression. This is often accompanied by increased endogenous estradiol and progesterone levels, indicating reduced suppression of the hypothalamic–pituitary–ovarian axis. These findings parallel studies of combined oral contraceptives (COCs) co-administered with rifampin, which also report decreased hormone levels, breakthrough bleeding, and occasional breakthrough ovulation. Therefore, patients using hormonal implants who require rifampin therapy must be counseled to use alternative or supplementary non-hormonal contraceptive methods.[3,14]

Beyond contraceptive failure, maternal TB, even in extrapulmonary forms like lymphadenitis, is strongly associated with increased risks of preterm birth, low birth weight, and perinatal mortality, particularly when the disease is active during pregnancy.[15–17] In this patient, tuberculous lymphadenitis was diagnosed prior to conception and appropriately managed; nevertheless, she developed PPROM and imminent preterm labor, conditions that significantly amplified neonatal morbidity and mortality.[18,19]

Pregnancy-related physiological and immunological adaptations may favor progression or reactivation of TB. TB has been associated with placental dysfunction, chronic fetal hypoxia, fetal growth restriction, and preterm birth.[15,16] Several studies from high-burden countries indicate that TB in pregnancy is linked to higher rates of PPROM, chorioamnionitis, and spontaneous preterm labor.[10,11]

These regional findings are echoed globally. A retrospective study in the United States found that pregnant women with TB had higher rates of preterm birth and maternal complications, including chorioamnionitis, postpartum anemia, and acute respiratory distress syndrome, than non-TB cohorts.[20,21] Furthermore, systematic reviews consistently show that active TB during pregnancy is associated with nearly threefold higher maternal mortality, fourfold higher perinatal mortality, a 1.7-fold higher risk of preterm birth, and up to a ninefold higher risk of miscarriage. These risks are exacerbated in cases of multidrug-resistant TB (MDR-TB).[20,22,23] Appropriate and timely anti-TB therapy remains the most effective intervention to improve maternal and fetal outcomes, underscoring the need for early diagnosis and integrated care.

Regarding the obstetric complications observed in this case, the pathogenesis of PPROM is multifactorial, involving inflammation, infection, mechanical stress, and biochemical weakening of the fetal membranes. Subclinical intrauterine or lower genital tract infections, such as bacterial vaginosis, are strongly associated with PPROM and spontaneous preterm labor. In this patient, a history of greenish, pruritic vaginal discharge suggests a localized infectious or inflammatory component. Although there were no overt signs of clinical chorioamnionitis or systemic sepsis at presentation, the early onset of PPROM at 25–26 weeks of gestation and its rapid progression to active labor suggest an underlying inflammatory mechanism that may not have been fully captured by routine laboratory parameters.[24,25]

Clinical Implications

This case underscores several key clinical lessons for both obstetricians and infectious disease clinicians:

1. Pre-treatment contraceptive counseling. Counseling is essential for women of reproductive age initiating rifampin-based TB regimens. Patients must be explicitly informed that rifampin reduces the effectiveness of progestin-only implants and other hormonal contraceptives. Alternative or additional methods should be offered.
2. For women who already use a progestin implant and are about to start rifampin, clinicians should consider:
   a Switching to a non-hormonal method (e.g., copper IUD) or
   b Adding consistent barrier contraception if the implant is continued, particularly when the device is nearing the end of its approved duration, as in this case.
3. In pregnant women with TB who have a history of hormonal contraception, clinicians should actively inquire about drug–drug interactions to identify potential causes of contraceptive failure and to guide postpartum contraceptive planning.
4. Finally, in TB-complicated pregnancies with PPROM and threatened preterm labor, multidisciplinary care and adherence to evidence-based protocols (antibiotics, corticosteroids, magnesium sulfate, and individualized timing of delivery) are crucial, but even optimal management cannot fully mitigate the high risks of extreme prematurity.[10–12,15]

Conclusion

This case demonstrates that rifampin-based treatment for tuberculous lymphadenitis can cause contraceptive implant failure, leading to an unintended pregnancy complicated by PPROM, threatened preterm labor, and extreme prematurity. It underscores the need for rigorous TB screening, careful contraceptive counseling regarding rifampin–hormonal interactions, and multidisciplinary management to optimize maternal and perinatal outcomes.

Artificial Intelligence Use Statement

Generative AI tools were used solely for language editing and improving the clarity of the manuscript. All content was reviewed and verified by the authors, who take full responsibility for the final manuscript.

Conflict of Interest

The authors declare no conflict of interest.

Author Contribution Statement

IA: Conceptualization, data collection, literature review, and manuscript preparation.
RA: Obstetric supervision, critical review, and manuscript revision.
IN: Pulmonology supervision, critical review, and manuscript revision. All authors reviewed and approved the final manuscript.

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