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CORIOAMNIONITIS Y TUBERCULOSIS PERITONEAL EN EL EMBARAZO: DIAGNÓSTICO INESPERADO Y DESENLACE OBSTÉTRICO ADVERSO. REPORTE DE CASO

CHORIOAMNIONITIS AND PERITONEAL TUBERCULOSIS IN PREGNANCY: AN UNEXPECTED DIAGNOSIS AND ADVERSE OBSTETRIC OUTCOME—A CASE REPORT

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Resumen

Antecedentes: La infección intraamniótica es una causa importante de morbilidad materna y desenlaces perinatales adversos. La tuberculosis durante el embarazo, en particular sus formas extrapulmonares, puede manifestarse con síntomas inespecíficos y retrasar el diagnóstico. La coexistencia de infección intraamniótica y tuberculosis peritoneal es excepcional y plantea un reto diagnóstico cuando la fiebre persiste pese al tratamiento antimicrobiano y al control del foco obstétrico. Presentación del caso: Una gestante con edad gestacional estimada de 21,8 semanas ingresó por fiebre, dolor abdominal tipo contracción, sangrado vaginal y pérdida de líquido amniótico. Presentaba taquicardia materna, temperatura de 38,6 °C, leucocitosis con neutrofilia y rotura prolongada de membranas. La amniocentesis obtuvo líquido turbio y amarillento, con marcada respuesta neutrofílica, glucosa no detectable y abundantes cocos grampositivos, hallazgos compatibles con infección intraamniótica. Ante hallazgos ecográficos fetales preocupantes y la persistencia de la infección, se indicó la interrupción médica del embarazo, que culminó con la expulsión de un feto masculino de aproximadamente 370 g, sin signos vitales. Posteriormente persistieron la fiebre elevada, la leucocitosis y el dolor abdominal intenso. La laparotomía exploratoria evidenció engrosamiento peritoneal, múltiples lesiones blanquecino-amarillentas, adherencias fibroadhesivas y líquido libre de aspecto purulento. La biopsia peritoneal mostró inflamación granulomatosa necrosante con células gigantes tipo Langhans, y el cultivo del líquido peritoneal aisló Mycobacterium tuberculosis, lo que confirmó el diagnóstico de tuberculosis peritoneal. Conclusión: La persistencia de fiebre tras el tratamiento de una aparente infección obstétrica debe motivar una reevaluación diagnóstica. La tuberculosis peritoneal puede coexistir con una infección intraamniótica y simular o prolongar un cuadro de sepsis obstétrica. Debido a que no se documentó M. tuberculosis en la placenta, las membranas ni el líquido amniótico, no puede establecerse una relación causal directa entre la tuberculosis y la corioamnionitis en esta paciente.

Palabras claves: corioamnionitis; infección intraamniótica; tuberculosis peritoneal; embarazo; rotura prematura pretérmino de membranas; Mycobacterium tuberculosis.

Abstract

Background: Intra-amniotic infection is an important cause of maternal morbidity and adverse perinatal outcomes. Tuberculosis during pregnancy, particularly extrapulmonary disease, may have nonspecific manifestations and consequently be diagnosed late. The coexistence of intra-amniotic infection and peritoneal tuberculosis is rare and poses a diagnostic challenge when fever persists despite antimicrobial therapy and obstetric source control. Case presentation: A pregnant patient at an estimated gestational age of 21.8 weeks was admitted with fever, contraction-like abdominal pain, vaginal bleeding, and leakage of amniotic fluid. Maternal tachycardia, a temperature of 38.6 °C, neutrophilic leukocytosis, and prolonged rupture of membranes were documented. Amniocentesis yielded turbid, yellow fluid with a marked neutrophilic response, undetectable glucose, and abundant gram-positive cocci, supporting the diagnosis of intra-amniotic infection. Because of concerning fetal ultrasonographic findings and ongoing infection,

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the pregnancy was terminated for medical indications, resulting in delivery of a male fetus weighing approximately 370 g, with no signs of life. High-grade fever, leukocytosis, and severe abdominal pain persisted thereafter. Exploratory laparotomy revealed diffuse peritoneal thickening, multiple whitish-yellow lesions, extensive fibroadhesive changes, and free intraperitoneal fluid with a purulent appearance. Peritoneal biopsy demonstrated necrotizing granulomatous inflammation with Langhans-type giant cells, and peritoneal-fluid culture grew Mycobacterium tuberculosis, confirming peritoneal tuberculosis. Conclusion: Persistent fever after treatment of an apparent obstetric infection should prompt diagnostic reassessment. Peritoneal tuberculosis may coexist with intra-amniotic infection and mimic or prolong obstetric sepsis. Because M. tuberculosis was not documented in the placenta, membranes, or amniotic fluid, a direct causal relationship between tuberculosis and chorioamnionitis cannot be established in this patient.

Keywords: chorioamnionitis; intra-amniotic infection; peritoneal tuberculosis; pregnancy; preterm prelabor rupture of membranes; Mycobacterium tuberculosis.

1. Introduction

Tuberculosis (TB) remains a leading cause of death from a single infectious agent. The World Health Organization estimated 10.7 million incident TB cases and 1.23 million TB-related deaths in 2024, including 3.7 million cases among women [1]. Diagnosis during pregnancy may be particularly challenging because constitutional manifestations such as fatigue, weight changes, dyspnea, and sweating can overlap with physiologic changes of gestation, while extrapulmonary disease may occur without respiratory symptoms that would otherwise raise clinical suspicion [2].

Intra-amniotic infection, historically referred to in clinical practice as chorioamnionitis, encompasses a spectrum in which clinical findings, amniotic-fluid results, and histopathology do not always coincide. Contemporary recommendations emphasize integrating maternal fever and associated clinical findings with microbiologic or inflammatory evidence, particularly in cases involving preterm prelabor rupture of membranes and preterm birth [3,4].

Peritoneal TB is an uncommon form of extrapulmonary tuberculosis that may present with persistent fever, abdominal pain, ascites, peritoneal thickening, or adhesions. These findings overlap with those of several surgical, infectious, gynecologic, and obstetric conditions [5]. During pregnancy, placental TB, intrauterine infection caused by M. tuberculosis, and acute membranitis have been described. However, establishing that clinical chorioamnionitis is directly caused by M. tuberculosis requires organism-specific evidence

from the placenta, membranes, amniotic fluid, or fetal tissues [6-8].

We report the case of a patient in the second trimester of pregnancy who was initially managed for intra-amniotic infection in the setting of prolonged membrane rupture. Persistent fever followed by signs of peritoneal irritation prompted exploratory laparotomy and led to the unexpected diagnosis of peritoneal TB. This report identifies clinical findings that should trigger diagnostic reassessment and critically examines the possible, but unproven, association between the two infectious processes.

2. Case Presentation

A pregnant patient at an estimated gestational age of 21.8 weeks by last menstrual period presented with contraction-like abdominal pain of approximately three hours' duration. The pain was initially mild and later became moderate in intensity, and it was accompanied by a moderate amount of bright-red vaginal bleeding. A previous ultrasound examination had reportedly shown complete placenta previa. The patient also reported seven days of documented fever above 38.5 °C and intermittent contraction-like abdominal pain. She had self-administered an anti-inflammatory medication but could not recall the active ingredient. She additionally described leakage of clear, non-malodorous vaginal fluid; a crystallization test was positive for amniotic fluid, prompting referral to a hospital.

On admission, moderate-intensity contractions and leakage of clear fluid persisted. No relevant medical, allergy, or surgical history was documented in the available clinical record. Vital signs were as follows: heart rate, 118 beats/min; blood pressure, 110/70 mmHg; respiratory rate, 19 breaths/min; oxygen saturation, 94% on room air; and axillary temperature, 38.6 °C. The patient was alert and oriented.

Cardiopulmonary examination was unremarkable. The abdomen was enlarged by the gravid uterus, with a uterine fundal height of 21 cm. Fetal heart rate was 140 beats/min, and uterine activity consisted of two contractions within 10 minutes, each lasting 15-20 seconds. The cervix was posterior and closed, without

effacement. Vaginal bleeding and leakage of clear fluid were observed. The clinical record documented membrane rupture approximately six days before examination.

Initial Diagnostic Assessment

Complete blood count showed a leukocyte count of 12.3 × 10^3/µL, a neutrophil count of 10.2 × 10^3/µL (85.6%), hemoglobin of 10.2 g/dL, hematocrit of 29.7%, and a platelet count of 232 × 10^3/µL. C-reactive protein was 10 mg/L. Initial examination of the amniotic fluid revealed abundant inflammatory cells, bacteria (2+), and some gram-positive cocci in pairs. Subsequent amniocentesis yielded turbid, yellow fluid with a predominance of polymorphonuclear cells (93%), undetectable glucose, and abundant leukocytes and gram-positive cocci on Gram stain. Together with maternal fever, tachycardia, prolonged rupture of membranes, and uterine symptoms, these findings supported the diagnosis of intra-amniotic infection.

Institutional obstetric ultrasonography estimated a fetal weight of 342 g and documented a fetal heart rate of 150 beats/min, an amniotic fluid index of 12.6 cm, an anterior placenta, breech presentation, and male fetal sex. No fetal movements were observed. The institutional description of an anterior placenta differed from the prior external ultrasound report of complete placenta previa. Because the complete earlier study was unavailable, this discrepancy could not be resolved retrospectively.

Therapeutic Intervention and Obstetric Outcome

During hospitalization, febrile peaks above 38 °C persisted. Intravenous paracetamol, isotonic saline, and broad-spectrum antimicrobial therapy were administered. The available record identifies ceftriaxone 1 g intravenously every 12 hours and documents a second antimicrobial agent administered at 900 mg intravenously every 8 hours, but does not identify that agent. Given the concerning fetal ultrasonographic findings and ongoing infection, the treating team decided to terminate the pregnancy for medical indications. The procedure resulted in delivery of a male fetus weighing approximately 370 g, with no signs of life.

Despite termination of the pregnancy and antimicrobial therapy, the febrile syndrome did not resolve. Approximately 10 days later, temperatures above 38.9 °C persisted, with a leukocyte count of 15.4 × 10^3/µL and marked neutrophilia. The patient developed severe hypogastric pain on superficial and deep palpation, signs of peritoneal irritation, and a visual analog pain score of 9/10. This clinical evolution prompted reassessment of the initial diagnosis and investigation for an uncontrolled intra-abdominal focus.

Unexpected Diagnosis of Peritoneal Tuberculosis

Exploratory laparotomy was performed. Intraoperatively, diffuse peritoneal thickening, multiple whitish-yellow lesions involving the visceral surfaces, extensive fibroadhesive changes, and free intraperitoneal fluid with a purulent appearance were identified. Granulomatous tissue adherent to the parietal peritoneum was sampled for histopathologic examination, and the peritoneal fluid was submitted for culture.

Histopathologic examination showed granulomatous inflammation with central necrosis, epithelioid cells, lymphocytes, and Langhans-type giant cells. Culture of the peritoneal fluid grew Mycobacterium tuberculosis. The concordance between the necrotizing granulomatous morphology and microbiologic confirmation established the diagnosis of peritoneal tuberculosis. The available source material did not document molecular drug-resistance testing, examination of the placenta or membranes for M. tuberculosis, TB-specific fetal evaluation, the subsequent antituberculous regimen, or maternal follow-up; these data were therefore neither inferred nor reconstructed.

Clinical Timeline

Clinical time point	Key findings and decisions
~7 days before admission	Fever >38.5 °C, contraction-like pain, and leakage of clear fluid.
Admission, ~21.8 weeks	Vaginal bleeding, fluid leakage, temperature 38.6 °C, heart rate 118/min, membranes ruptured for several days, cervix closed.

Clinical time point	Key findings and decisions
Initial evaluation	Leukocytosis/neutrophilia; inflammatory amniotic fluid. Amniocentesis: turbid fluid, 93% polymorphonuclear cells, undetectable glucose, and abundant gram-positive cocci.
During hospitalization	Antipyretics, hydration, and broad-spectrum antibiotics. Ultrasound showed abnormal fetal findings and absence of fetal movements.
Obstetric outcome	Medically indicated pregnancy termination; male fetus without signs of life, approximately 370 g.
~10 days later	Persistent fever >38.9 °C, leukocytosis/neutrophilia, severe hypogastric pain, and signs of peritoneal irritation.
Laparotomy	Thickened peritoneum, diffuse whitish-yellow lesions, fibroadhesive changes, and free fluid with a purulent appearance.
Etiologic confirmation	Biopsy with necrotizing granulomas and Langhans-type giant cells; peritoneal-fluid culture positive for *M. tuberculosis*.

Surgical and Histopathologic Findings

Figure 1. *Intraoperative findings during exploratory laparotomy. The peritoneal surfaces show diffuse inflammatory changes, whitish-yellow lesions, and an adhesive pattern. The image is reproduced from the original clinical material without diagnostic markings or other alterations.*

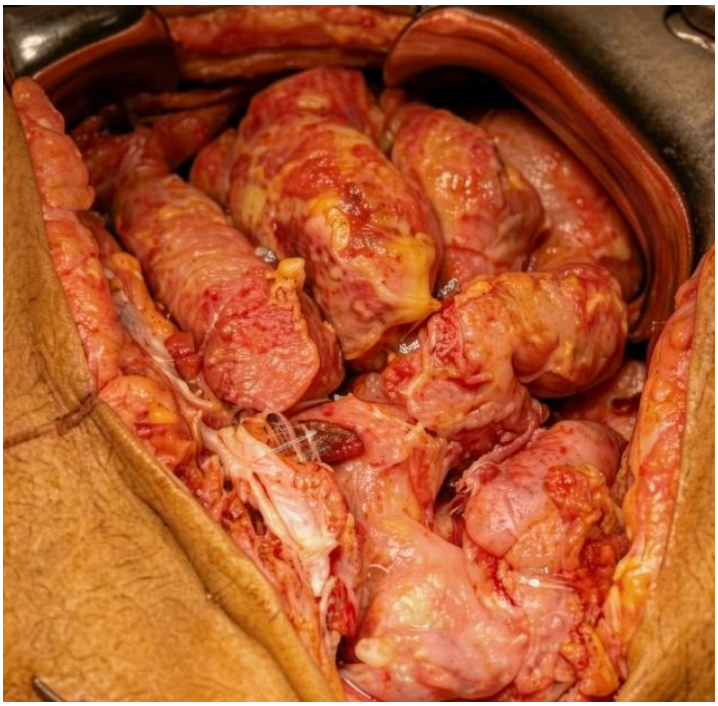
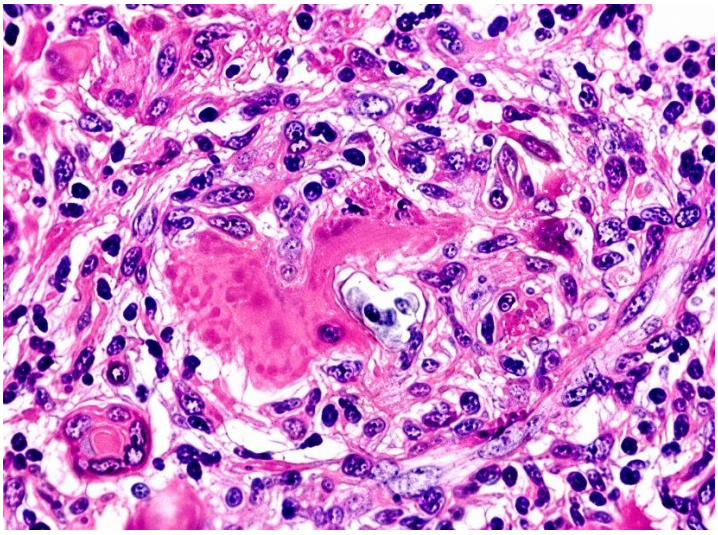


Figure 2. *Micrograph of peritoneal tissue. Histopathologic examination showed granulomatous inflammation with central necrosis, epithelioid cells, lymphocytes, and Langhans-type giant cells, findings suggestive of a tuberculous etiology. Magnification was not documented in the source record.*



3. Discussion

1. From the Initial Obstetric Diagnosis to the Need for Reassessment

The initial diagnostic framework was clinically consistent with intra-amniotic infection in a pregnancy at a previable gestational age complicated by prolonged rupture of membranes. Maternal fever of 38.6 °C accompanied by tachycardia, uterine symptoms, and fluid leakage required immediate evaluation. Amniocentesis provided direct evidence of intra-amniotic inflammation: turbid fluid, neutrophil predominance, undetectable glucose, and abundant gram-positive cocci. Current recommendations from the American College of Obstetricians and Gynecologists and recent microbiologic reviews emphasize integrating clinical suspicion with amniotic-fluid data when available, because fever alone identifies neither the underlying etiology nor the affected compartment [3,4].

The critical issue in this case was therefore not the plausibility of the initial diagnosis, but its inability to explain the entire subsequent clinical course. After pregnancy termination and broad-spectrum antimicrobial therapy, persistent high-grade fever, increasing leukocytosis, and the development of abdominal pain with signs of peritoneal irritation suggested an additional disease

process or an uncontrolled focus. Continuing to attribute the entire course to chorioamnionitis alone would have reflected diagnostic anchoring. The change in clinical trajectory appropriately prompted reassessment of the differential diagnosis and surgical exploration.

2. Peritoneal Tuberculosis: Why It May Be Missed During Pregnancy

Peritoneal TB is an extrapulmonary form of tuberculosis characterized by nonspecific manifestations. Fever, abdominal pain, anorexia, weight loss, ascites, and peritoneal thickening may evolve subacutely and be mistaken for inflammatory or infectious processes of another etiology. During pregnancy, this diagnostic difficulty is amplified because constitutional symptoms may overlap with physiologic changes of gestation, and extrapulmonary disease may occur without prominent cough or other respiratory manifestations [2,5].

Laparotomy in this patient revealed a pattern highly suggestive of abdominal TB: a thickened peritoneum, whitish-yellow lesions, fibroadhesive changes, and free peritoneal fluid. Nevertheless, the macroscopic appearance is not pathognomonic and may mimic carcinomatosis, inflammatory disease, fungal infection, or other granulomatous peritonitides. The diagnostic strength of this case lies in the concordance between pathology and microbiology: necrotizing granulomas with Langhans-type giant cells supported a mycobacterial etiology, while culture of the peritoneal fluid confirmed M. tuberculosis.

In patients with ascites, ascitic-fluid adenosine deaminase may support suspicion of peritoneal TB. An updated meta-analysis reported pooled sensitivity and specificity estimates of approximately 0.90 and 0.94, respectively, although the overall certainty of evidence was very low [9]. Molecular assays such as Xpert MTB/RIF may provide rapid confirmation and an initial assessment of resistance, but their sensitivity for abdominal TB is limited, and a negative result does not exclude disease [5,10]. Therefore, when the peritoneum is accessible, tissue acquisition for histopathology, culture, and molecular testing represents a high-value diagnostic strategy.

3. Can Tuberculosis Explain the Chorioamnionitis?

The literature demonstrates that M. tuberculosis can involve the placenta and, in rare cases, cause intrauterine infection with acute inflammation of the membranes. Taweevisit et al. described intrauterine tuberculosis presenting as acute chorioamnionitis, while more recent pathologic series have documented acute fetal membranitis, placental necrosis, and M. tuberculosis positivity by staining and molecular methods [6,7]. A particularly relevant report published online in 2025 described placental TB secondary to tuberculous peritonitis, with molecular confirmation in obstetric tissues and fluids [8]. These observations make it biologically plausible that maternal extrapulmonary TB could contribute to placental inflammation or intrauterine infection.

Biologic plausibility, however, does not establish causality in the present case. The amniotic fluid contained abundant gram-positive cocci, whereas M. tuberculosis was confirmed in the peritoneal compartment. No culture, polymerase chain reaction, or staining for M. tuberculosis was available from the amniotic fluid, placenta, membranes, or fetal tissues, and placental histopathology was not documented. Thus, the most rigorous interpretation is that two processes were demonstrated—intra-amniotic infection and peritoneal TB—with a potential pathophysiologic relationship that cannot be proven retrospectively. Direct tuberculous causation of chorioamnionitis therefore remains unconfirmed by compartment-specific microbiologic or histopathologic evidence.

4. Maternal-Fetal Implications

Tuberculosis during pregnancy is associated with a higher risk of maternal and perinatal complications, particularly when diagnosis is delayed or extrapulmonary or disseminated disease is present. Contemporary reviews describe associations with preterm birth, fetal growth restriction, low birth weight, pregnancy loss, and maternal morbidity [2,11]. In a series of 19 placentas affected by TB, seven pregnancies ended in intrauterine fetal death; prematurity

and cases of congenital TB were also documented among surviving neonates [7].

In this patient, the adverse obstetric outcome occurred in the setting of prolonged membrane rupture, objectively documented intra-amniotic infection, and previously unrecognized peritoneal TB. The relative contribution of each process to the fetal outcome cannot be quantified. Intra-amniotic infection alone can trigger a fetal inflammatory response and is associated with fetal deterioration, preterm birth, and perinatal mortality; active maternal TB may also increase obstetric risk [2,4]. The coexistence of both conditions increased the clinical complexity but does not justify unsupported causal attribution.

5. What Would Have Strengthened Etiologic Attribution?

When maternal TB is suspected in a similar obstetric presentation, examination of the placenta and membranes—and, when clinically indicated and feasible, amniotic fluid or fetal samples—may be decisive. Combining histopathology, acid-fast bacillus staining, culture, and molecular assays increases the likelihood of demonstrating gestational involvement by M. tuberculosis [7]. In the present case, the absence of these specimens limits the ability to distinguish concomitant ascending bacterial chorioamnionitis from tuberculous intrauterine infection.

The clinical course also illustrates the value of maintaining a low threshold for investigating extrapulmonary TB in patients with persistent fever, abdominal pain, and an evolution not fully explained by the initial diagnosis, particularly in settings where TB remains epidemiologically relevant. Current guidelines recommend rapid microbiologic and molecular testing whenever possible and sampling of the affected site; culture remains important for confirmation and drug-susceptibility testing [12,13].

6. Treatment and Pregnancy: Scope of the Available Information

Active TB during pregnancy requires treatment and therapy should not be postponed once the diagnosis is established; the maternal and fetal risks of untreated disease outweigh those associated with recommended first-line regimens when used in

accordance with international guidance [13,14]. However, the available record does not document the antituberculous regimen administered after confirmation, the drug-susceptibility profile of the isolate, or the subsequent maternal outcome. For methodological rigor, these elements were neither reconstructed nor assumed in this manuscript.

4. Conclusions

This case demonstrates that a well-supported initial obstetric diagnosis should not preclude reconsideration when the clinical pattern changes. Intra-amniotic infection was supported by maternal fever, prolonged rupture of membranes, and markedly inflammatory amniotic fluid containing abundant gram-positive cocci. Persistent fever and subsequent signs of peritoneal irritation revealed a second process: peritoneal TB confirmed by necrotizing granulomas and a positive culture for M. tuberculosis. The central lesson is that peritoneal TB and intra-amniotic infection may coexist and create a diagnostically misleading clinical picture. In similar scenarios, failure to improve as expected should prompt active investigation for extrapulmonary TB. When tuberculous involvement of pregnancy is suspected, the placenta, membranes, and relevant obstetric specimens should be examined using histopathology, culture, and molecular methods.

Declarations

Informed consent for publication: Written informed consent for publication of the case and accompanying images was obtained and is retained by the authors.

Conflicts of interest: The authors declare no conflicts of interest.

Data availability: The clinical data underlying this report are restricted because of patient confidentiality and may be handled only in accordance with institutional policies and the consent obtained.

References

1. World Health Organization. Global tuberculosis report 2025. Geneva: WHO; 2025.

2. Longo C, Pontes KFM, Faíco MMdM, Melo MM, Callado GY, Barbosa CdB, et al.

Tuberculosis in Pregnancy: An Updated Narrative Review. Diagnostics (Basel). 2026;16(11):1576. doi:10.3390/diagnostics16111576.

3. American College of Obstetricians and Gynecologists. ACOG Clinical Practice Update: Update on Criteria for Suspected Diagnosis of Intraamniotic Infection. Obstet Gynecol. 2024;144(1):e17-e19. doi:10.1097/AOG.0000000000005593.

4. Pongchaikul P, Warintaksa P, Jenjaroenpun P, Opasawatchai A, Settacomkul R, Vivithanaporn P, et al. Intra-amniotic infection: diagnosis, nomenclature, clinical significance, management, and microbiologic tools used for the diagnosis. Clin Microbiol Rev. 2026;Online First:e00070-26. doi:10.1128/cmr.00070-26.

5. Jha DK, Pathiyil MM, Sharma V. Evidence-based approach to diagnosis and management of abdominal tuberculosis. Indian J Gastroenterol. 2023;42(1):17-31. doi:10.1007/s12664-023-01343-x.

6. Taweevisit M, Nisagornsen C, Thorner PS. Intrauterine Tuberculosis Manifesting as Acute Chorioamnionitis: A Case Report and Review of the Literature. Pediatr Dev Pathol. 2015;18(4):335-338. doi:10.2350/15-02-1607-CR.1.

7. Hu Z, Zeng D, Yang Y, Liu H, Wang A, Li D, et al. Pathomorphological characteristics of tuberculous placenta and its clinical implication. Diagn Pathol. 2023;18(1):128. doi:10.1186/s13000-023-01419-4.

8. Ohnishi Y, Kono C, Suyama Y. Placental tuberculosis secondary to tuberculous peritonitis. QJM. Published online September 26, 2025:hcaf223. doi:10.1093/qjmed/hcaf223.

9. Mahajan M, Prasad ML, Kumar P, Kumar A, Chatterjee N, Singh S, et al. An Updated Systematic Review and Meta-Analysis for the Diagnostic Test Accuracy of Ascitic Fluid Adenosine Deaminase in Tuberculous Peritonitis. Infect Chemother. 2023;55(2):264-277. doi:10.3947/ic.2023.0014.

10. Sharma V, Soni H, Kumar-M P, Dawra S, Mishra S, Mandavdhare HS, et al. Diagnostic accuracy of the Xpert MTB/RIF assay for abdominal tuberculosis: a systematic review and meta-analysis. Expert Rev Anti Infect Ther. 2021;19(2):253-

265. doi:10.1080/14787210.2020.1816169.

11. Su Y, Kong X, Chen Z, Wang X, Lu S. Adverse pregnancy outcomes and complications of tuberculosis in pregnant women. Front Cell Infect Microbiol. 2025;15:1550430. doi:10.3389/fcimb.2025.1550430.

12. World Health Organization. WHO consolidated guidelines on tuberculosis: Module 3: diagnosis. Geneva: WHO; 2025.

13. World Health Organization. WHO consolidated guidelines on tuberculosis: Module 4: treatment and care. Geneva: WHO; 2025.

14. Centers for Disease Control and Prevention. Tuberculosis Clinical Care and Treatment During Pregnancy. Atlanta: CDC; updated April 17, 2025.

15. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. BMJ Case Rep. 2013;2013:bcr2013201554. doi:10.1136/bcr-2013-201554.