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Case Report

Pregnancy with pleural effusion – challenges in diagnosis and management

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Abstract

Tuberculosis (TB) remains a major public health threat in Bangladesh. Pregnant women face a heightened risk of contracting TB, which can lead to a more severe clinical trajectory and severely compromise both maternal and fetal outcomes. Approximately two-thirds of pregnant women with TB are asymptomatic, largely due to latent infection. Diagnosis is difficult because constitutional TB symptoms mimic normal physiological changes during pregnancy, radio-imaging options are restricted, and invasive diagnostic procedures are often delayed. Here we report a case of tuberculosisin a pregnant woman who presented with a short history of breathlessness and cough with low-grade fever, and progressive breathlessness that swiftly evolved into severe respiratory distress. A chest X-ray confirmed a right-sided pleural effusion. Although sputum smears were negative for Acid-Fast Bacilli (AFB) and GeneXpert, pleural fluid thoracocentesis revealed marked lymphocytosis (98%) and elevated adenosine deaminase (ADA) levels. This critical finding secured the diagnosis of tuberculous pleural effusion (TPE) on the 6th day of the puerperium. Tuberculous pleuritis during pregnancy is a rare, life-threatening condition that presents significant diagnostic hurdles due to its non-specific and delayed presentation. However, in regions with a high TB burden, clinicians must maintain a high index of clinical suspicion for tuberculosis in any pregnant woman presenting with unexplained pleural effusion.

Introduction

Tuberculosis (TB) is a public health concern in our country. Bangladesh faces a high tuberculosis burden, ranking among the top 30 countries globally, with an estimated incidence rate of 221 per 100,000 population with roughly 78,000 cases estimated to be undetected as of 2023 [1]. Notably, out of the 10.7 million new TB cases reported in 2024, nearly 35% occurred in women of reproductive age [2]. Consequently, pregnant women represent a highly vulnerable demographic. Tuberculosis in pregnant state is described as a "double edged sword", as it adversely affects maternal and fetal condition while pregnancy also has worsening effects on the progression of tuberculosis [3]. Screening and diagnosis are further complicated by physiological and immunological adaptations during pregnancy, leading to frequent diagnostic delays [2]. The gestational suppression of the T-helper 1 (Th1) proinflammatory response – essential for maintaining maternal-fetal tolerance – simultaneously heightens susceptibility to new TB infections and latent reactivation. This immunological shift is also responsible for masking symptoms, making TB diagnosis delayed and more

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difficult [4]. Consequently, a large proportion of pregnant women remain asymptomatic or present with subtle symptoms (such as fatigue, night sweats, and dyspnea) that are easily mistaken for typical pregnancy-related fatigue and shortness of breath [5].

Diagnosing TB becomes even more challenging when the disease presents as Extrapulmonary Tuberculosis (EPTB). EPTB frequently manifests with atypical features, creating critical delays in intervention. Tuberculous pleural effusion (TPE) is the second most common form of extrapulmonary tuberculosis, accounting for approximately 15-25% of all EPTB cases [6,7]. It is even more difficult to diagnose TPE due to the low count of tubercle bacilli in pleural fluid. While pleural biopsy remains the definitive test for confirmation, it is invasive and conventional microbiological fluid analyses fail to yield positive results in the vast majority of cases [8]. In contrast, measuring pleural fluid Adenosine Deaminase (ADA) offers high sensitivity and specificity for early TPE detection, even when bacterial density is negligible. Today, pleural fluid ADA is a essential frontline tool in evaluating unexplained effusions in TB-endemic regions [6].

Here, we report a rare, diagnostically elusive case of tuberculous pleural effusion in a pregnant woman who presented with non-specific symptoms and a negative AFB smear, where elevated pleural fluid ADA served as the sole definitive key to the diagnosis.

Case presentation

A 27-year-old housewife, para 1, gravida 2^{nd}, presented at the Obstetrics & Gynaecology outpatient department of BIRDEM General Hospital at her 36^{+5} weeks of gestation with the complaints of a 2-day history of progressive breathlessness, cough, and intermittent fever with an evening rise, without chills or rigors. She had no prior history of asthma, tuberculosis, or known exposure to active TB cases. On examination she was mildly anaemic, non-icteric, with no oedema and no groups of (including supraclavicular) lymph nodes were palpable. Her temperature was 100°F, pulse 90 b/min and blood pressure 110/70 mmHg. SPO_2 was 91% in room air in lying position. She had tachypnoea (respiratory rate 24 breaths/min), chest expansion was restricted on right side with dullness on percussion, and decreased breath sounds over the right lung field. Three hours post-admission, the patient developed lower abdominal pain accompanied by per-vaginal watery discharge. Obstetric examination revealed active fetal movements, 2 uterine contractions per 10 minutes, and a normal baseline fetal heart rate (FHR) of 145 bpm. Vaginal examination showed a soft cervix with a closed os, station -2, and the presence of bloody show was present. A preliminary diagnosis of 2nd gravida at 36^{+5} weeks in early labour complicated by right-sided pleural effusion was established. Initial laboratory workup revealed ESR 80 in first 10 min, Hb% 9.5 gm/dl, WBC 9,800 cmm/mL with normal differential count. Over the next 5 hours, labour progression stalled while the mother's respiratory distress rapidly intensified. Concurrently, cardiotocography (CTG) demonstrated a non-reassuring trace with fetal tachycardia (180–186 bpm) and decreased fetal movement. Due to acute fetal distress, an emergency Lower Uterine Cesarean Section (LUCS) was performed. The amniotic fluid was found to be deeply meconium-stained. A male infant weighing 2.5 kg was delivered with APGAR scores of 4 at 1 minute and 6 at 5 minutes, requiring immediate admission to the Neonatal Intensive Care Unit (NICU).

On the first postoperative day, the patient suffered severe acute respiratory distress. Clinical examination showed a soft abdomen, a well-contracted uterus at the umbilical level, present bowel sounds, and normal lochia. A bedside chest X-ray confirmed a significant right-sided pleural effusion.

A pulmonology consultation was obtained, and therapeutic/diagnostic thoracocentesis was performed, yielding approximately 1,000 mL of fluid. Protein content was 2.0 g/dL (normal 1-2 g/dL), lactate dehydrogenase (LDH) 120 IU/L (<50 IU/L), adenosine deaminase (ADA) 50 IU/L (40 IU/L) with mild neutrophilia (55%). Sputum was negative for acid fast bacilli (AFB) and Gene Xpert. Despite fluid drainage, the patient's condition worsened, with SpO_2 dropping even with supplemental oxygen via a high-flow mask (8 L/min), necessitating transfer to the Intensive Care Unit (ICU). Thoracocentesis was repeated on 5^{th} post operative day and the tests were repeated. On repeat analysis, all the parameters were significantly raised; protein content was raised to 3.6 g/dL, LDH 240 IU/L, ADA 72 IU/L, WBC 11,000 cmm/mL with

marked lymphocytosis (98%). Sputum and second sample of pleural fluid were still negative for AFB and Gene Xpert on repeated smear. Due to her financial constraints advanced diagnostic tests such as QuantiFERON TB Gold, Interferon-Gamma Release Assays (IGRA), or a thoracic CT scan could not be performed. Relying on mycobacterial cultures was impractical given their long turnaround time. However, a pleural fluid ADA of >40 IU/L with lymphocyte predominance gives a strong suspicion of tuberculous aetiology and an indication to start anti-TB therapy [7]. Following pulmonology guidelines, standard anti-tubercular therapy (ATT) was initiated: a 2-month intensive phase of Rifampicin, Isoniazid, Ethambutol, and Pyrazinamide, followed by a 4-month continuation phase of Rifampicin and Isoniazid [9].

As patient was sputum negative, strict airborne isolation was not required. However, comprehensive counseling was provided regarding medication adherence, hand hygiene, cough etiquette, and mask usage while handling the baby. The patient exhibited a dramatic recovery. Within 2 weeks of ATT initiation, she became completely afebrile, her dyspnea resolved, and her general condition improved significantly. At her 3-week follow-up, breastfeeding was successfully initiated, and both mother and infant were in good health.

Discussion

Tuberculous pleural effusion (TPE) is a paucibacillary (low bacterial count) mycobacterial infection within the pleural space followed by a T-cell mediated hypersensitivity reaction [10]. Patients commonly present with nonspecific symptoms, such as fever, non-productive cough, pleuritic chest pain, and dyspnea [6]. A recent study including 304 patients of TPE showed fever (76%) cough (75%, with expectoration in 40%), chest pain (61%) and respiratory distress (49%), fatigue (41%) and night sweats (38%) as the presenting symptoms [11]. Our patient also had fever, cough and dyspnea, but had no chest pain.

Pathophysiologically, TPE begins with a rapid, transient neutrophilic influx, followed by a sustained lymphocytic reaction, pleural granuloma formation, and localized ADA release. This might be responsible for decreased positive pleural fluid culture with time, as the effusion becomes lymphocyte predominant, and viable mycobacteria are sequestered [8]. Onyenekwu et al. reported that most patients with TB pleural effusions present without an elevation in the peripheral white blood cell count during the acute febrile illness [12].

Pleural fluid in our patient was negative for acid-fast bacilli (AFB) in repeated samples. The diagnosis of TPE and distinguishing it from other causes of pleural effusion is challenging and often delayed, especially if clinicians still depend primarily on the demonstration of Mycobacterium tuberculosis (MTB) on Ziehl Neelsen (ZN) staining or positive culture in pleural tissue or fluid samples for the definite diagnosis [13]. Pleural fluid examination with ZN stain requires an AFB density of >10,000/ml to be positive. So, ZN staining has very poor sensitivity due to the low bacterial count in pleural fluid in TPE and culture is time-consuming [14]. AFB smears from pleural fluid are rarely positive, with reported positivity rates of less than 5-15% [6,15]. Though MTB culture requires much less viable material, it is time consuming (2 weeks in liquid media compared to 6 weeks for traditional solid media) and is also poorly sensitive, with positivity rates of as low as <20% [6,8]. In a case reported byAhuja et al., the pleural fluid culture tested positive for AFB, but it took 4 weeks [16]. Only then anti-TB therapy was started and she made a rapid recovery with liberation from mechanical ventilation in the intensive care unit.

On X-ray chest, this patient had right sided pleural effusion without any parenchymal involvement. So, it was difficult to assume the cause of the effusion from the radiograph. Actually, there are no specific or distinguished pleural radiologic findings that can confirm TPE [7]. Rate of parenchymal involvement in TPE is quite low, reported to be 30-38% in a number of previous studies [7,17]. Tuberculous pleural effusions are unilateral in most of the cases, as seen in this case, with no predilection for either the left or right hemithorax and usually occupies less than two-thirds of the hemithorax in 80% of cases [18]. Thoracic ultrasound is better in characterizing the nature of the effusion and also helps in guided thoracocentesis and closed pleural biopsies [8]. Ahuja et al. reported a similar case where early use of bedside lung ultrasonography (USG) was instrumental in the successful management of their patient who was admitted in critical care unit with TPE and was in shock [16]. Computed tomographic (CT) scan of the chest is

currently the best imaging modality to visualize both pleura and lung parenchyma in TB pleural effusions [8].Parenchymal involvement rates with TPE are consistently higher when CT chest studies are performed [7]. We did not have the facility of bedside USG and CT scan was not considered due to financial constraints of the patient.

Alternative methods which have better sensitivity and need much less time include pleural biopsy via thoracoscopy, pleural fluid adenosine deaminase assay (ADA) and tuberculosis antibody (TB-antibody) test [19]. While pleural biopsy has higher diagnostic sensitivity, it is invasive and not always feasible. In this context, high adenosine deaminase (ADA) measurement in pleural fluid has emerged as a valuable diagnostic tool in recent years [6].

Pleural fluid analysis for biochemical parameters is very important in determining the probable cause. The effusion is uniformly exudative having protein concentrations invariably >5.0, and >3.0 g/L in 50% to 77% of cases [8]. The pleural fluid lactate dehydrogenase (LDH) level is elevated in approximately 75% of cases, with levels commonly exceeding 500 IU/L [7]. In our reported case, protein content of pleural fluid was 3.6 g/dL in second sample and LDH was also raised (240 IU/L). But the breakthrough test was pleural fluid ADA level which was 72 IU/ L and guided us to the final diagnosis of TPE. According to several large studies and a recent meta-analysis, among the available biochemical markers, ADA, IFN-γ, and IL-27 are the most promising and considering that ADA has advantages of low cost and suitability for standardization, ADA is recommended for TPE diagnosis [13].

Testing pleural fluid ADA level is very useful in establishing the diagnosis of TPE especially when there is a moderate to high suspicion of TB in patients with negative pleural fluid or biopsy cultures, and non-diagnostic histology [20]. A wide range of cut-off values are in use but in the majority of studies the most accurate threshold was found to range between 40 and 60 U/L [13,19]. In a comprehensive meta-analysis, Aggarwal et al. showed that pleural fluid ADA has a sensitivity of 92% and specificity of 90% for the diagnosis of TPE [21]. Liang et al. [22] also reported almost similar sensitivity and specificity. Vorster et al. [8] suggested that in populations with a high prevalence of TB and clinical suspicion of TB effusion, elevated ADA level might be considered as a confirmatory test justifying prompt treatment initiation. In high-burden areas it is often used routinely in the investigation of undiagnosed pleural effusions, or to supplement standard pleural fluid analysis where a tuberculous effusion is suspected [7,8]. In a prospective analysis of 100 patients in a teaching hospital of Nepal, diagnosis of TB pleural effusion was made based on pleural fluid ADA cut-off value of 42.19 U/L and almost all patients diagnosed to have TB pleural effusion responded completely to anti-tubercular treatment [23]. In countries like ours, where the BCG vaccination is used nationally the Mantoux test has limited utility. So, this relatively non-invasive, inexpensive and quick diagnostic test has now become an important tool against challenging situations as seen in our case. Alsaeed et al. has reported a similar case of TPE with only evidence of high ADA and negative AFB smear, TB polymerase chain reaction (PCR), and M. tuberculosis culture [6]. Several authors have reported that in the early phase of the disease, levels of pleural fluid ADA may be low giving rise to a false negative result. However, ADA level invariably is found elevated when thoracocentesis is repeated a few days later [24]. We did also find rising ADA level in the second sample taken at 5 days interval.

The predominant nucleated cell type also varies depending on timing of collection of the pleural fluid. Pleural fluid findings are known to evolve from early neutrophil to lymphocytic predominance in TB effusions, and the value of repeat thoracocentesis has been shown to significantly increase the diagnostic yield. Fluid collected in the first few days may exhibit a neutrophil predominant effusion, while lymphocytes tend to dominate (>75%) later on [7,8]. In our case, there was mild neutrophilia (55%) in the first sample and in the second sample lymphocyte was 98%. Lo Cascio et al. [7] have shown that pleural fluid from most of the TPE cases have more than 50% lymphocytes, with some having more than 90%. When lymphocyte count is >75% or a lymphocyte neutrophil ratio is 0.75 or greater in combination with raised ADA, the sensitivity, specificity, positive predictive value, negative predictive value, and efficiency for the identification of TB were reported at 88%, 95%, 95%, 88%, and 92%, respectively [8]. In a high-burden and resource constrained setting it may

therefore be appropriate to repeat thoracocentesis before proceeding to pleural biopsy.

Vorster et al. commented that the medical treatment for TB pleural effusion is the same as for pulmonary TB with a variable rate of resolution [8]. Fever usually resolves within 2 weeks with reabsorption of the pleural fluid within 6 weeks. By using the raised ADA level as a diagnostic tool, we could start prompt anti-TB treatment and our patient also became afebrile within 2 weeks.

Conclusion

Tuberculous pleuritis in pregnancy is a rare, complex clinical condition characterized by deceptive, non-specific symptoms and limited diagnostic options. In high-prevalence settings, clinicians must maintain a high index of suspicion for TB whenever evaluating unexplained pleural effusions in pregnant or postpartum women. High ADA levels in pleural fluid with a lymphocytic exudate provide a strong basis for prompt anti-TB therapy, preventing catastrophic maternal-fetal morbidity even when traditional smears and cultures remain inconclusive.

Consent to participate and publish

The patient has given consent for publication.

Conflict of interest

The authors declare no competing financial or non-financial interests in relation to this work.

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Author contributions

SS Designed the report, prepared, reviewed, did literature search and drafted the manuscript. SJ reviewed the manuscript, RSG helped in literature search and reviewed manuscript, and AN helped in patient selection and management.

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References

1. Bangladesh: Tuberculosis. Updated 2026. Available from: https://www.theglobaleconomy.com. [Accessed in April 2026]
2. Global Tuberculosis Report 2025. Available from: https://www.who.int/teams/global-programme-on-tuberculosis-and-lung-health/tb-reports/global-tuberculosis-report-2025.[Accessed in April 2026]
3. Loto OM, Awowole I. Tuberculosis in pregnancy: A review. *J Pregnancy*. 2012; **2012**: 379271. doi: 10.1155/2012/379271.
4. Mathad JS, Gupta A. Tuberculosis in pregnant and postpartum women: epidemiology, management, and research gaps. *Clin Infect Dis***.** 2012; **55**(11): 1532-49. doi: 10.1093/cid/cis732.
5. Miele K, Bamrah Morris S, Tepper NK. Tuberculosis in pregnancy. *Obstet Gynecol*. 2020; **135**(6): 1444-1453. doi: 10.1097/AOG.0000000000003890.
6. Alsaeed MA, Sattar MFA. Diagnostic challenge of tuberculous pleural effusion: Elevated adenosine deaminase (ADA) as the key indicator in the absence of microbiological confirmation. *Cureus*. 2025; **17**(10): e93944. doi: 10.7759/cureus.93944.
7. Lo Cascio CM, Kaul V, Dhooria S, Agrawal A, Chaddha U. Diagnosis of tuberculous pleural effusions: A review. *Respir Med*. 2021; **188**: 106607. doi: 10.1016/j.rmed.2021.106607.
8. Vorster MJ, Allwood BW, Diacon AH, Koegelenberg CF. Tuberculous pleural effusions: advances and controversies. *J Thorac Dis*. 2015; **7**(6): 981-91. doi: 10.3978/j.issn.2072-1439.2015.02.18.
9. National Guidelines and Operational Manual for Tuberculosis Control: Sixth edition. October 2021. DGHS, MOHFW, Dhaka, Bangladesh.
10. Inchingolo R, Carriera L, Ielo S, Barone R, Smargiassi A, Lipsi R, et al. Tuberculous pleural effusion: integrating thoracic ultrasound and medical thoracoscopy in the diagnosis and

management: a pictorial narrative review and an algorithm proposal. *J Ultrasound*. 2026; **29**(1): 1-11. doi: 10.1007/s40477-025-01094-9.

11. Zhao T, Chen B, Xu Y, Qu Y. Clinical and pathological differences between polymorphonuclear-rich and lymphocyte-rich tuberculous pleural effusion. *Ann Thorac Med*. 2020; **15**(2): 76-83. doi: 10.4103/atm.ATM_15_20.
12. Onyenekwu CP, Zemlin AE, Erasmus RT. High pleural fluid adenosine deaminase levels: a valuable tool for rapid diagnosis of pleural TB in a middle-income country with a high TB/HIV burden. *S Afr Med J*. 2014; **104**(3): 200-3. doi: 10.7196/samj.7428.
13. Norris SC, Pipaliya BP, Pandya HB, Patel RD, Javadekar TB. Advancing pleural TB diagnosis: A comprehensive review of adenosine deaminase and GeneXpert approaches. *IJIRM*. 2026; **11**(1): 27-32. doi: 10.18231/j.ijirm.12287.1770102640.
14. Zhang M, Li D, Hu ZD, Huang YL. The diagnostic utility of pleural markers for tuberculosis pleural effusion. *Ann Transl Med*. 2020; **8**(9): 607. doi: 10.21037/atm.2019.09.110.
15. Macías A, Sánchez-Montalvá A, Salvador F, Villar A, Tórtola T, Saborit N, et al. Epidemiology and diagnosis of pleural tuberculosis in a low incidence country with high rate of immigrant population: A retrospective study. *Int J Infect Dis*. 2019; **78**: 34-38. doi: 10.1016/j.ijid.2018.10.005.
16. Ahuja V, Gombar S, Kumar N, Goyal N, Gupta K. Pregnancy with bilateral tubercular pleural effusion: challenges. *Trop Doct*. 2014; **44**(2): 116-8. doi: 10.1177/0049475513517117.
17. Bielsa S, Acosta C, Pardina M, Civit C, Porcel JM. Tuberculous pleural effusion: Clinical characteristics of 320 patients. *Arch Bronconeumol* (Engl Ed). 2019; **55**(1): 17-22. English, Spanish. doi: 10.1016/j.arbres.2018.04.014.
18. McNally E, Ross C, Gleeson LE. The tuberculous pleural effusion. *Breathe (Sheff)*. 2023; **19**(4): 230143. doi: 10.1183/20734735.0143-2023.
19. Huo Z, Yang M, Chen J, Peng L. Improved early diagnosis of difficult cases of tuberculous pleural effusion by combination of thoracoscopy with immunological tests. *Int J Infect Dis*. 2019; **81**: 38-42. doi: 10.1016/j.ijid.2019.01.045.
20. Krenke R, Korczyński P. Use of pleural fluid levels of adenosine deaminase and interferon gamma in the diagnosis of tuberculous pleuritis. *Curr OpinPulm Med*. 2010; **16**(4): 367-75. doi: 10.1097/MCP.0b013e32833a7154.
21. Aggarwal AN, Agarwal R, Sehgal IS, Dhooria S. Adenosine deaminase for diagnosis of tuberculous pleural effusion: A systematic review and meta-analysis. *PLoS One*. 2019; **14**(3): e0213728. doi: 10.1371/journal.pone.0213728.
22. Garcia-Zamalloa A, Taboada-Gomez J. Diagnostic accuracy of adenosine deaminase and lymphocyte proportion in pleural fluid for tuberculous pleurisy in different prevalence scenarios. *PLoS One.* 2012; **7**(6): e38729. doi: 10.1371/journal.pone.0038729.
23. Devkota KC, Shyam BK, Sherpa K, Ghimire P, Sherpa MT, Shrestha R, et al. Significance of adenosine deaminase in diagnosing tuberculous pleural effusion. *Nepal Med Coll J*. 2012; **14**(2): 149-52.
24. Kotsiou OS, Tzortzi P, Beta RAA, Kyritsis A, Gourgoulianis KI. Repeatability of pleural adenosine deaminase measurements in diagnostic evaluation of pleural effusions. *J Clin Lab Anal*. 2018; **32**(5): e22371. doi: 10.1002/jcla.22371.

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