RESEARCH PAPER

Pulmonary Tuberculosis Beyond the Lungs: Disseminated Disease with Adrenal Tuberculosis, Primary Adrenal Insufficiency, and ATT Drug Malabsorption - A Case Report

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ABSTRACT

Introduction: Tuberculosis (TB), while primarily a pulmonary disease, is capable of disseminating haematogenously to virtually any organ system. Adrenal gland involvement is a clinically significant but frequently overlooked manifestation, with the capacity to precipitate life-threatening primary adrenal insufficiency (PAI). Persistent or worsening clinical deterioration despite apparently compliant anti-tuberculous therapy (ATT) should prompt investigation of both extrapulmonary dissemination and ATT drug malabsorption.

Case Summary: We report a 65-year-old male farmer with microbiologically confirmed pulmonary TB (rifampicin-sensitive) on ATT for one month, who presented in a lethargic state with haemoptysis, dyselectrolytaemia refractory to repeated correction, and relative hypotension. CT abdomen revealed bulky bilateral adrenal glands; serum cortisol was critically suppressed at 2.7 µg/dL. A diagnosis of disseminated TB involving the lungs, mediastinal nodes, and bilateral adrenal glands with consequent PAI (tuberculous Addison's disease) was established. Therapeutic drug monitoring (TDM) identified sub-therapeutic isoniazid levels, mandating ATT regimen modification. He demonstrated rapid clinical and biochemical improvement following steroid replacement.

Conclusion: This case underscores the imperative to look beyond the lungs in any patient with TB showing paradoxical deterioration. The diagnostic triad of refractory dyselectrolytaemia, haemodynamic instability, and bilateral adrenal enlargement on CT should prompt urgent cortisol assay and steroid rescue. Concurrent ATT drug level monitoring is essential in patients with suboptimal treatment response.

Keywords: Disseminated tuberculosis; adrenal tuberculosis; primary adrenal insufficiency; Addison's disease; therapeutic drug monitoring; isoniazid malabsorption; dyselectrolytaemia; tuberculous Addison's disease

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1. INTRODUCTION

Tuberculosis (TB) remains one of the foremost infectious threats to global health. The World Health Organization (WHO) 2024 Global Tuberculosis Report estimates 10.8 million new TB cases worldwide in 2023, with 1.25 million deaths-once again overtaking HIV/AIDS as the leading cause of death from a single infectious agent. [1] While pulmonary TB accounts for the majority of cases, extrapulmonary TB constitutes approximately 15% of the global burden, with the potential to involve virtually any organ system through haematogenous or lymphatic dissemination. [1]

Among extrapulmonary manifestations, adrenal gland involvement merits special clinical attention. The adrenal glands are highly vascularised organs that receive one of the highest rates of blood flow per gram of tissue in the body, rendering them susceptible to haematogenous seeding by Mycobacterium tuberculosis. Adrenal TB is found in approximately 6% of patients with active pulmonary or disseminated TB on autopsy series. [2][3] Critically, adrenal insufficiency-clinically manifest as Addison's disease-does not become apparent until more than 90% of the adrenal cortex is destroyed; by this time, the patient may present in frank adrenal crisis. [4] Persistent hyponatraemia and hypokalaemia refractory to standard electrolyte correction in a TB patient are cardinal clinical clues that should trigger adrenal evaluation.

A further dimension of complexity in managing TB patients is the phenomenon of sub-therapeutic ATT drug concentrations due to malabsorption or pharmacokinetic

variability. Low serum isoniazid levels, the most commonly identified abnormality on TDM, are associated with poorer bacteriological outcomes and risk of acquired drug resistance. [5] We present a case that illustrates both of these challenges-adrenal TB with PAI and isoniazid malabsorption-in a single patient with apparently compliant standard-of-care ATT, highlighting the paradigm of 'pulmonary TB beyond the lungs.'

2. CASE REPORT

2.1 Presentation and History

A 65-year-old male farmer, Mr. A (name withheld for confidentiality), was brought to the Emergency Room (ER) in a lethargic state. He was a known case of microbiologically confirmed pulmonary TB with sputum AFB 3+, sputum CBNAAT demonstrating MTB detected with rifampicin-sensitive pattern-and had been initiated on weight-based first-line ATT (three tablets daily) approximately one month prior.

He presented with four days of insidious, progressive generalised weakness and lethargy, haemoptysis (blood-tinged sputum, approximately four episodes over two days), persistent hiccups since the preceding day, and reduced oral intake for one week. He additionally reported three episodes of loose, non-bloody stools over the preceding two days. There was a significant history of 12 kg weight loss over the preceding three months. He denied fever, chest pain, breathlessness, orthopnoea, or paroxysmal nocturnal dyspnoea.

Past medical history: The current TB episode represented a new, never-treated case; no prior ATT exposure was documented. He was not known to have bronchial asthma, type 2 diabetes mellitus, systemic hypertension, coronary artery disease, or cerebrovascular accident. COVID-19 vaccination (2 doses) had been received.

Social history: Mixed diet. Former smoker: 10 cigarettes per day for 20 years (10 pack-years); cessation 10 years prior. Moderate alcohol use: 180 mL per week. No biomass fuel exposure.

2.2 Examination Findings

On general examination, the patient was conscious but appeared lethargic and ill. He was afebrile. Pallor was present. No icterus, cyanosis, clubbing, generalised lymphadenopathy, or pedal oedema was identified. Vitals: Blood pressure 100/70 mmHg (reflecting relative hypotension in the context of his clinical state), pulse rate 96/min (regular), respiratory rate 21/min, SpO_2 99% at rest, 97% on exertion.

Systemic examination: Respiratory auscultation revealed fine inspiratory crackles in the right infraclavicular, mammary, interscapular, and axillary areas. Heart sounds S1 and S2 were audible with no murmurs. Abdomen was soft with no organomegaly. No skin hyperpigmentation was documented at initial presentation.

2.3 Initial Investigations and Provisional Diagnosis

Chest X-ray demonstrated fibrocavitary changes with surrounding non-homogeneous consolidatory opacities in the right upper and mid zones. HRCT chest confirmed bilateral upper lobe consolidation with air bronchogram, a thick-walled bronchocentric cavity with surrounding consolidation in the right upper lobe, and collapse-consolidation of the right middle lobe.

Initial blood investigations revealed a leucocyte count of 29,300/cmm (markedly elevated), haemoglobin 9.1 g/dL, ESR 160 mm/hour, and CRP 24 mg/dL (positive). Serum electrolytes showed profound hyponatraemia (Na^+ 126 mEq/L) and hypokalaemia (K^+ 2.9 mEq/L). Serum chloride 82 mEq/L. Liver function tests were within normal limits. Sputum culture: normal upper respiratory flora. Blood culture: no growth. Urine culture: no growth. Renal function was essentially preserved (creatinine 1.2 mg/dL, urea 34 mg/dL).

The provisional diagnoses at admission were: (i) ATT-induced toxicity, (ii) acute gastroenteritis, and (iii) dyselectrolytaemia. Broad-spectrum intravenous antibiotics were commenced, blood transfusion was initiated for anaemia correction, and General Medicine consultation was obtained for electrolyte management.

2.4 Serial Biochemical Profile

Table 1. Serial haematological and biochemical parameters during admission (17 March – 1 April 2024)

Parameter	17-Mar	19-Mar	22-Mar	24-Mar	26-Mar	29-Mar	01-Apr
Hb (g/dL)	9.1	8.5	8.2	7.3	9.1	9.3	10.3
TLC (/cmm)	29300	15840	12240	8730	8100	7210	6100
Urea (mg/dL)	34	19	33	29	24	26	—
Creatinine	1.2	0.8	1.1	0.9	0.9	0.9	—
Na^+ (mEq/L)	126	116	115	131	125	130	133
K^+ (mEq/L)	2.9	2.7	2.9	3.6	3.4	3.3	3.4
Cl^- (mEq/L)	82	84	83	94	89	90	98
Platelets (L)	4.5L	3.35L	2.75L	2.50L	4.02L	3.55L	3.9L

Hb = Haemoglobin; TLC = Total Leucocyte Count; Na^+ = Sodium; K^+ = Potassium; Cl^- = Chloride; L = Lakhs/cmm.

2.5 Diagnostic Workup: Pivot Towards Adrenal TB

Despite repeated, targeted intravenous correction of sodium (with hypertonic saline) and potassium, the electrolytes failed to normalise persistently - a critical clinical pivot point. Sodium values oscillated between 115–131 mEq/L across multiple correction cycles (Table

1). Concurrent haemodynamic fragility - with blood pressure consistently below 100/70 mmHg - further heightened suspicion for a mineralocorticoid and glucocorticoid deficiency state.

In the working differential of ATT-induced hepato-renal toxicity, an ultrasound and CT abdomen were obtained. CT abdomen revealed bilaterally bulky adrenal glands. In the context of known TB with refractory dyselectrolytaemia and haemodynamic compromise, this finding immediately raised the diagnostic possibility of adrenal TB with PAI. Serum cortisol levels were urgently requested.

Serum cortisol: 2.7 µg/dL - critically low (reference: 6–23 µg/dL morning value). [6] 24-hour urine cortisol was also confirmatory of cortisol deficiency. The clinical-biochemical-radiological constellation - pulmonary TB on ATT + hypotension + refractory hyponatraemia + hypokalaemia + bulky bilateral adrenal glands on CT + morning serum cortisol 2.7 µg/dL - established the diagnosis of Primary Adrenal Insufficiency secondary to Adrenal Tuberculosis (Tuberculous Addison's Disease).

2.6 Therapeutic Drug Monitoring

An important diagnostic question arose: given that the patient had been adherent to ATT (compliance confirmed by fixed-dose combination tablet dispensing records and pill count), why had his disease disseminated and clinically worsened? The possibility of ATT drug malabsorption was explored. Therapeutic drug monitoring (TDM) of first-line ATT drugs was performed using 2-hour post-dose serum sampling. [5] Results identified sub-therapeutic serum isoniazid (INH) levels, while rifampicin, pyrazinamide, and ethambutol levels were within acceptable therapeutic ranges. ATT was modified with an increased INH dosage, targeting weight-appropriate therapeutic concentrations.

2.7 Treatment and Outcome

On establishing the diagnosis of PAI secondary to adrenal TB, intravenous hydrocortisone was commenced immediately (100 mg stat IV followed by 200 mg per 24 hours). Within 48–72 hours, a striking clinical improvement was observed: generalised weakness and lethargy resolved, blood pressure normalised, and - critically - the electrolyte profile began to stabilise towards the normal range without requirement for further aggressive electrolyte infusion.

ATT was continued (with modified INH dosage per TDM) and planned for a 9-month course to cover disseminated disease. Hydrocortisone was titrated and fludrocortisone was added for mineralocorticoid replacement, with patient counselling regarding the potential for lifelong steroid dependence. Given the rifampicin-steroid interaction (rifampicin is a potent CYP3A4 inducer that accelerates glucocorticoid metabolism, effectively doubling or tripling steroid clearance), hydrocortisone doses were adjusted upward accordingly. [7] The patient was discharged with plans for outpatient follow-up of ATT response, hormone levels, and electrolytes. Clinical and radiological improvement was documented at follow-up.

Table 2. Summary of Clinical, Biochemical, Radiological, and Treatment Parameters

Parameter	Finding
Age / Sex	65 years / Male
Underlying condition	PTB (Sputum AFB 3+, CBNAAT MTB+, Rifampicin-sensitive)
ATT duration at presentation	1 month (IP phase)
Key complaints	Generalised weakness, haemoptysis, hiccups, 12 kg weight loss, loose stools
BP at admission	100/70 mmHg (relative hypotension)
Electrolytes at nadir	Na^+ 115, K^+ 2.7, Cl^- 83 mEq/L — refractory despite correction
Haemoglobin (nadir)	7.3 g/dL
CT Abdomen	Bilaterally bulky adrenal glands
Serum Cortisol	2.7 µg/dL (critically low; normal: 6–23 µg/dL)
TDM finding	Sub-therapeutic serum isoniazid levels
Final diagnosis	Disseminated TB (Pulmonary + Mediastinal nodes + Bilateral adrenal glands) + Primary Adrenal Insufficiency (Tuberculous Addison's Disease)
ATT regimen	Modified ATT × 9 months (increased INH dose per TDM)
Steroid therapy	IV Hydrocortisone → Oral Hydrocortisone + Fludrocortisone (dose-adjusted for rifampicin interaction)
Outcome	Clinical improvement; electrolytes normalised; radiological improvement on follow-up

3. DISCUSSION

3.1 The Pathophysiology of Adrenal Tuberculosis and Primary Adrenal Insufficiency

The adrenal glands receive the highest blood flow per unit mass of any organ in the body, making them uniquely susceptible to haematogenous seeding during mycobacteraemia. Adrenal TB almost always arises as a secondary manifestation of TB elsewhere - most commonly pulmonary or genitourinary TB - through haematogenous or lymphatic dissemination. [3][8] Bilateral adrenal involvement is observed in the majority of cases, and bilateral destruction is the prerequisite for overt clinical adrenal insufficiency. A critical threshold exists: PAI does not manifest until more than 90% of the

total adrenocortical mass has been destroyed by caseating granulomatous inflammation. [4][9] This physiological reserve explains why many patients with adrenal TB remain biochemically compensated until very late in the disease course, and why the clinical diagnosis is frequently delayed - sometimes presenting for the first time as a life-threatening adrenal crisis.

Contemporary evidence from a multicentre cohort study (Shanghai Public Health Clinical Centre, 2025) of nine cases of adrenal TB identified bilateral adrenal involvement in eight of nine patients, with a mean diagnostic delay of several weeks. [8] Similarly, a 2025 cross-sectional study from a high-TB-burden region demonstrated clinical features of adrenal insufficiency in 27% of 100 treatment-naïve TB patients, with confirmed adrenal insufficiency in 1%, underscoring that a significant proportion of TB patients harbour subclinical adrenocortical impairment. [10]

3.2 The Clinical Phenotype

Refractory Dyselectrolytaemia as a Diagnostic Clue

The classical triad of PAI - hypotension, hyponatraemia, and hyperkalaemia - results from combined glucocorticoid and mineralocorticoid deficiency. In adrenal TB, the mineralocorticoid deficit (aldosterone deficiency) drives urinary sodium wasting and potassium retention (hyperkalemia), while cortisol deficiency compounds the haemodynamic compromise. [11] In our patient, however, the electrolyte pattern was characterised by hyponatraemia and hypokalaemia - the latter atypical for classic PAI. This combination likely reflected concurrent gastrointestinal losses (loose stools, reduced intake) superimposed on the mineralocorticoid deficit, a pattern consistently described in TB patients with concomitant gastrointestinal involvement.

The key diagnostic clue in our case was the refractoriness of the dyselectrolytaemia to repeated aggressive correction - not merely the presence of low electrolyte values. Endocrine and metabolic reviews of TB-associated hyponatraemia confirm that adrenal insufficiency should be systematically excluded before attributing persistent hyponatraemia to SIADH or gastrointestinal losses. [11] A morning serum cortisol below 3 µg/dL carries very high specificity for adrenal insufficiency; our patient's cortisol of 2.7 µg/dL was diagnostic without the need for ACTH stimulation testing in the acute setting. [6]

3.3 CT Abdomen - Imaging in the Diagnostic Pathway

CT imaging of the abdomen is the most clinically accessible and informative initial investigation for adrenal TB. Active disease characteristically produces bilateral adrenal enlargement - which may be nodular, with central low density (caseation) and peripheral rim enhancement on contrast imaging. Chronic or healed disease produces bilateral atrophic glands with calcification, which is highly specific for TB aetiology. [3] In our patient, bilaterally bulky adrenal glands on CT in the context of active pulmonary TB, haemodynamic compromise, and refractory hyponatraemia provided sufficient evidence to initiate steroid replacement before histopathological confirmation. Where available, adrenal biopsy - increasingly performed via ultrasound-guided core needle technique - demonstrating caseating granulomas with or without acid-fast bacilli confirms the diagnosis definitively. [12]

3.4 Therapeutic Drug Monitoring: The Overlooked Dimension

A singular and instructive dimension of this case was the identification of sub-therapeutic isoniazid levels through TDM in a patient who was apparently compliant with weight-based ATT. Standard first-line ATT pharmacokinetic parameters assume normal gastrointestinal absorption; in practice, multiple factors can significantly reduce bioavailability: food-drug interactions (fatty foods and amino acids impairing intestinal pH and drug solubility), first-pass metabolism variability, gastrointestinal pH alteration from ongoing diarrhoea, hypoalbuminaemia, and pharmacogenomic variation in NAT2 acetylator status. [5]

Isoniazid is the most pharmacokinetically vulnerable first-line drug, with expected 2-hour peak serum concentrations of 3–6 µg/mL; sub-therapeutic levels (below 2 µg/mL) have been identified in up to 23 of 39 patients (59%) in one programmatic study and in 87% of patients in another series, particularly in those with comorbidities and hypoalbuminaemia. [13] Low isoniazid concentrations are independently associated with poor clinical and bacteriological outcomes and increased risk of acquired drug resistance - precisely the clinical trajectory observed in our patient, who continued to deteriorate despite one month of standard ATT. [5] TDM using 2-hour post-dose sampling (with or without a 6-hour sample to distinguish delayed absorption from true malabsorption) represents the most practical clinical approach and is increasingly recommended in patients with a slow treatment response. [13]

3.5 The Rifampicin-Steroid Drug Interaction

A critical but frequently underappreciated therapeutic challenge in managing concurrent adrenal TB and ATT is the pharmacokinetic interaction between rifampicin and glucocorticoids. Rifampicin is a potent inducer of hepatic cytochrome P450 3A4 enzymes, markedly accelerating the metabolism of hydrocortisone and other glucocorticoids. This interaction effectively reduces the bioavailability of oral hydrocortisone by 45–75%, meaning that standard replacement doses of hydrocortisone (15–25 mg/day) are grossly inadequate during rifampicin co-administration. [7][9] Clinical guidelines recommend doubling or tripling the hydrocortisone dose while the patient remains on rifampicin-containing ATT regimens, with progressive dose tapering as the ATT course concludes and rifampicin is discontinued. Fludrocortisone for mineralocorticoid replacement is less affected by this interaction but should still be monitored clinically.

3.6 Prognosis and the Question of Adrenal Recovery

The prognosis for adrenal function recovery in tuberculous

PAI is guarded. Because adrenal insufficiency only manifests after >90% cortical destruction by caseating necrosis, substantial and often irreversible architectural damage has already occurred by the time the diagnosis is made. Published literature - including a 2019 review - confirms that adrenal function does not recover in the majority of patients with Addison's disease caused by TB, necessitating lifelong glucocorticoid and mineralocorticoid replacement. [9] An exception exists for patients diagnosed very early in the course (acute disease with minimal necrosis), where a minority achieves adrenal recovery after successful ATT, as documented in one European case series. [6] Our patient was counselled accordingly, and serial cortisol monitoring was planned at 6-monthly intervals during and after ATT completion.

3.7 Causes of Disease Dissemination Despite ATT - A Framework

This case prompts a systematic consideration of why a patient on standard; correctly dosed ATT may develop progressive or disseminated TB:

Table 3. Causes of Clinical Deterioration or Dissemination Despite ATT

Cause	Mechanism / Comment
Sub-therapeutic drug levels (malabsorption)	Gastrointestinal pH changes, food interactions, hypoalbuminaemia, NAT2 polymorphisms; most common with isoniazid
Drug resistance (primary or acquired)	Rifampicin or MDR-TB not excluded at diagnosis; inadequate dosing accelerates resistance selection
Malnutrition	Impairs cellular immunity; reduces drug albumin binding; 12 kg weight loss in 3 months in this patient
Alcoholism	Alters CYP450 metabolism; hepatotoxicity risk; compliance challenge; immunosuppressive
Immunosuppressive comorbidity	HIV, diabetes mellitus, CKD, connective tissue disorders, biologic DMARDs (infliximab, etanercept)
Non-compliance / irregular dosing	Verified by pill count and dispensing records in this case; ruled out as primary cause
Pre-existing extrapulmonary foci at diagnosis	Haematogenous seeding of adrenals, mediastinal nodes may have preceded ATT initiation

4. LEARNING POINTS AND CONCLUSION

This case encapsulates four critical and generalisable clinical lessons for practitioners managing patients with TB:

1. Think beyond the lungs: Any TB patient presenting with systemic symptoms disproportionate to the pulmonary disease burden, particularly refractory dyselectrolytaemia and haemodynamic compromise, must be evaluated for extrapulmonary dissemination. Adrenal TB should be in every physician's differential for unexplained hyponatraemia in a TB patient.

2. Refractory dyselectrolytaemia is a red flag: Electrolyte abnormalities that do not correct with standard replacement therapy - the hallmark of aldosterone and cortisol deficiency - should prompt urgent serum cortisol measurement and CT abdomen rather than escalating electrolyte infusion volumes.

3. Sub-therapeutic drug levels are common and clinically significant: TDM should be considered in any patient who fails to respond appropriately to standard ATT, particularly when compliance has been verified. Isoniazid is the most frequently sub-therapeutic drug and is the most clinically consequential.

4. The rifampicin-steroid interaction demands dose adjustment: Concurrent management of adrenal TB (ATT + steroid replacement) requires awareness that rifampicin markedly accelerates steroid metabolism, necessitating dose escalation of hydrocortisone during ATT and re-tapering after rifampicin discontinuation.

In conclusion, this case of disseminated TB presenting with adrenal involvement, refractory dyselectrolytaemia, and isoniazid malabsorption illustrates the critical imperative to maintain a broad diagnostic lens in any patient with TB who deteriorates on standard therapy. The diagnosis of adrenal TB and PAI remains challenging because early clinical features are non-specific, and the condition manifests clinically only after extensive adrenocortical destruction. Prompt recognition, steroid rescue, and ATT regimen optimisation through TDM are the pillars of successful management.

AUTHOR CONTRIBUTIONS, ETHICS & DECLARATIONS

Author Contributions: Dr. Elambarithi M: conception, Dr. Puneeth KS : clinical management , : Dr. Vijay A , Dr. Elambarithi M: data collection , Dr. Elambarithi M ,Dr. Palwai Santhoshi Dr Jai Kishan M : manuscript drafting **Dr. Nirmala Devi Chandrasekaran :**conception, Literature review, final approval and supervision

Ethics Statement: This case report was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the patient for

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Data Availability: De-identified data supporting the findings are available from the corresponding author upon reasonable request.

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