RESEARCH PAPER

Clinical Profile and Aetiopathogenesis of Patients with Sodium and Calcium Abnormalities in Tuberculosis

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ABSTRACT

Background: Tuberculosis is a systemic infectious disease associated with significant metabolic and electrolyte disturbances. Sodium and calcium abnormalities, particularly hyponatraemia and hypocalcaemia, are increasingly recognized but remain under-characterized in relation to disease severity and clinical forms.

Methods: A hospital-based observational analytical study was conducted over six months (August 2025 to January 2026) at a tertiary care center, including 60 patients with confirmed tuberculosis. Clinical, demographic, and laboratory data were collected. Serum sodium and calcium levels were measured at baseline. Associations between electrolyte abnormalities and disease characteristics were analyzed using Chi-square test and logistic regression.

Results: Hyponatraemia was observed in 43.3% (n=26) and hypocalcaemia in 30.0% (n=18) of patients. Hyponatraemia was significantly associated with type of tuberculosis (p=0.021), with highest prevalence in CNS TB (80.0%) and disseminated TB (66.7%). Hypocalcaemia was also significantly associated with disease type (p=0.048), being most frequent in CNS TB (60.0%). On multivariate analysis, age >50 years (AOR=3.2; p=0.018), CNS TB (AOR=5.8; p=0.002), and diabetes mellitus (AOR=3.1; p=0.031) were independent predictors of hyponatraemia.

Conclusion: Electrolyte abnormalities are common in tuberculosis, with hyponatraemia strongly associated with severe disease forms. The predominance of hypocalcaemia suggests variation in metabolic response. Routine electrolyte monitoring is essential for early identification and improved clinical management.

Keywords: Tuberculosis; Hyponatremia; Hypocalcemia; Electrolyte Imbalance; Syndrome of Inappropriate Antidiuretic Hormone Secretion; Central Nervous System Infections; Comorbidity; Disease Severity; Metabolic Disorders.

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Introduction :

Tuberculosis (TB) continues to pose a major global public health challenge, remaining one of the leading causes of morbidity and mortality worldwide despite the availability of effective treatment. According to World Health Organization, an estimated 10.6 million people developed TB in 2022, with a substantial burden concentrated in low- and middle-income countries, particularly in South-East Asia [1]. The persistent high incidence and mortality reflect not only transmission dynamics but also delays in diagnosis, co-morbid conditions, and systemic complications associated with the disease. Global epidemiological analyses further emphasize that TB remains a dynamic epidemic influenced by demographic transitions, socioeconomic disparities, and health system limitations, thereby necessitating continuous evaluation of its clinical spectrum [2].

From a broader perspective, TB is increasingly recognized as a complex systemic disease rather than a localized pulmonary infection. The The Lancet TB series highlights that Mycobacterium tuberculosis infection involves intricate host-pathogen interactions that extend beyond the lungs, affecting multiple organ systems and metabolic pathways [3]. This systemic nature explains the wide spectrum of clinical manifestations, ranging from asymptomatic infection to severe disseminated disease. Earlier public health literature has consistently emphasized that TB transcends geographical and biological boundaries, affecting diverse populations and presenting with variable clinical profiles [4],[5]. Advances in diagnostic modalities, including molecular techniques, have improved early detection; however, they have also revealed the heterogeneity in disease presentation, including biochemical and metabolic alterations that are often underrecognized [6].

The pathophysiology of TB is primarily driven by host immune responses to Mycobacterium tuberculosis. Innate immune mechanisms, particularly macrophage activation and cytokine release, play a central role in granuloma formation and containment of infection [7]. These granulomatous responses, while protective, are also responsible for systemic inflammatory effects that can disrupt normal physiological processes. Mathematical and epidemiological models have demonstrated that such host responses vary across age groups and disease stages, influencing both transmission and disease severity [8]. Furthermore, even after microbiological cure, persistent inflammatory activity has been documented, suggesting that TB induces long-lasting alterations in immune and metabolic pathways [9].

An important yet often overlooked aspect of TB pathogenesis is its interaction with the endocrine system. Emerging evidence indicates that TB can significantly disrupt immune-endocrine homeostasis, leading to hormonal imbalances and metabolic dysregulation [10]. The chronic inflammatory state associated with TB influences the hypothalamic-pituitary-adrenal axis, alters cytokine-mediated signaling, and affects hormonal secretion patterns. These changes contribute to a spectrum of endocrine manifestations, including adrenal insufficiency, altered vitamin D metabolism, and disturbances in water and electrolyte balance. Such endocrine and metabolic complications are increasingly recognized as important determinants of disease severity and clinical outcomes [11].

Among these metabolic disturbances, electrolyte abnormalities—particularly sodium imbalance—are of considerable clinical relevance. Hyponatremia is one of the most frequently reported electrolyte disorders in hospitalized patients and is associated with significant morbidity and mortality [12]. In the context of TB, hyponatremia may arise through multiple mechanisms, including the syndrome of inappropriate antidiuretic hormone secretion (SIADH), adrenal gland involvement, and central nervous system infection. The clinical evaluation and management of hyponatremia require a thorough understanding of its underlying pathophysiology, as inappropriate correction may lead to serious neurological complications [13].

SIADH has been identified as a key mechanism underlying hyponatremia in TB. It is characterized by impaired water excretion due to inappropriate secretion of antidiuretic hormone, leading to dilutional hyponatremia. Clinical decision-making in SIADH involves careful assessment of volume status, serum osmolality, and underlying etiology, particularly in chronic infectious conditions such as TB [14]. Recent advances in understanding SIADH pathophysiology have highlighted the role of inflammatory mediators and neuroendocrine dysregulation in its development. These insights are particularly relevant in TB, where persistent inflammation and systemic involvement may exacerbate electrolyte disturbances [15].

Despite growing recognition of electrolyte abnormalities in TB, there remains a significant gap in comprehensive clinical characterization, especially in differentiating patterns between pulmonary and extrapulmonary forms of the disease. Most existing studies have focused on isolated biochemical parameters or small cohorts, limiting the generalizability of findings. Furthermore, the interplay between sodium and calcium abnormalities, their underlying mechanisms, and their correlation with clinical profiles have not been adequately explored in a unified framework.

In this context, the present study aims to evaluate the clinical profile and aetiopathogenesis of sodium and calcium abnormalities in patients with tuberculosis.

By systematically analyzing these electrolyte disturbances and their association with disease characteristics, this study seeks to provide deeper insights into the metabolic dimension of TB. Understanding these alterations is essential not only for improving clinical management but also for identifying potential prognostic markers and therapeutic targets in tuberculosis.

Materials and Methods

This hospital-based observational analytical study was conducted at a tertiary care center after obtaining approval from the Institutional Ethics Committee. The study was carried out over a six-month period from August 2025 to January 2026, and included a total of 60 patients diagnosed with tuberculosis. Both pulmonary and extrapulmonary forms of tuberculosis were considered to comprehensively evaluate the clinical profile and underlying aetiopathogenesis of electrolyte abnormalities, particularly sodium and calcium disturbances.

Patients aged 16 years and above with newly diagnosed or microbiologically confirmed tuberculosis were included in the study after obtaining informed consent. Cases were classified into pulmonary tuberculosis, extrapulmonary tuberculosis, central nervous system (CNS) tuberculosis, and disseminated tuberculosis based on clinical, radiological, and laboratory findings. Patients with conditions known to independently affect electrolyte balance, such as chronic kidney disease, pre-existing endocrine disorders, or those receiving medications like diuretics, were excluded to minimize confounding.

A detailed clinical evaluation was performed for all participants, including demographic data (age, gender), type of tuberculosis, and associated comorbidities such as diabetes mellitus, hypertension, and HIV infection. Baseline laboratory investigations were carried out at the time of diagnosis prior to initiation of anti-tubercular therapy. Serum sodium and calcium levels were measured using standard automated biochemical analyzers in the hospital laboratory following established protocols. Hyponatraemia was defined based on standard reference values (serum sodium <135 mEq/L), and hypocalcaemia was defined as serum calcium levels below the normal laboratory reference range.

The primary outcome measures included the prevalence of hyponatraemia and hypocalcaemia among tuberculosis patients and their association with different types of tuberculosis and clinical variables. Secondary outcomes included the identification of predictors of hyponatraemia through regression analysis.

Data were entered into a structured proforma and analyzed using **Statistical Package for the Social Sciences (SPSS) version 26.0**. Descriptive statistics were used to summarize categorical variables as frequencies and percentages. The association between type of tuberculosis and electrolyte abnormalities was assessed using the Chi-square test, and statistical significance was considered at a p-value <0.05. Univariate logistic regression analysis was performed to identify factors associated with hyponatraemia, including age group, gender, type of tuberculosis, and comorbidities. Variables with statistical significance in univariate analysis were further included in multivariate logistic regression to determine independent predictors, and results were expressed as odds ratios (OR) and adjusted odds ratios (AOR) with 95% confidence intervals.

All procedures were conducted in accordance with ethical standards, and patient confidentiality was strictly maintained throughout the study.

Results:

The results of this study will assess the prevalence of sodium and calcium abnormalities among tuberculosis patients and examine their association with different clinical types of tuberculosis. It will also evaluate potential predictors and underlying aetiopathogenic factors contributing to these electrolyte disturbances.

TABLE 1. Baseline Characteristics of Study Participants (N = 60)

Category	Frequency (n)	Percentage (%)
Age Group (years)		
16–30	18	30
31–50	24	40
>50	18	30

Gender		
Male	36	60
Female	24	40
Type of Tuberculosis		
Pulmonary TB	28	46.7
Extrapulmonary TB	16	26.7
CNS TB	10	16.6
Disseminated TB	6	10
Comorbidities		
Diabetes Mellitus	14	23.3
HIV	6	10
Hypertension	10	16.7

The study included a total of 60 tuberculosis patients. The majority of participants were in the **31–50 years age group (40%)**, followed by equal proportions in the **16–30 years (30%)** and **>50 years (30%)** categories, indicating a relatively even age distribution with a slight predominance of middle-aged individuals. A higher proportion of participants were **male (60%)** compared to females (40%), suggesting a male predominance in the study population.

Regarding the type of tuberculosis, **pulmonary TB was the most common form (46.7%)**, followed by **extrapulmonary TB (26.7%)**, **CNS TB (16.6%)**, and **disseminated TB (10%)**. This reflects the typical epidemiological pattern where pulmonary involvement is more frequent.

Among comorbid conditions, **diabetes mellitus was the most prevalent (23.3%)**, followed by **hypertension (16.7%)** and **HIV infection (10%)**. This indicates a notable burden of metabolic and immunocompromising conditions among TB patients.

Overall, the study population was predominantly middle-aged, male, and mainly affected by pulmonary tuberculosis, with a considerable proportion having associated comorbidities, particularly diabetes.

TABLE 2. Prevalence of Electrolyte Abnormalities (N = 60)

Category	**Frequency (n)**	**Percentage (%)**
Sodium Status		
Hyponatraemia	26	43.3
Normal	34	56.7
Calcium Status		
Hypocalcaemia	18	30.0
Normal	42	70.0

Out of 60 tuberculosis patients, **hyponatraemia was observed in 43.3%** of participants, while **56.7% had normal sodium levels**, indicating that sodium imbalance is a relatively common finding in TB patients. With respect to calcium status, **hypocalcaemia was present in 30.0%** of cases, whereas the majority (**70.0%**) had normal calcium levels.

Overall, these findings suggest that **electrolyte abnormalities particularly hyponatraemia are frequent among tuberculosis patients**, highlighting the importance of routine electrolyte monitoring in this population.

TABLE 3A. Association Between Type of TB and Electrolyte Abnormalities (N = 60)

Type of TB	**Hyponatraemia Present n (%)**	**Hyponatraemia Absent n (%)**	**Total**	**p-value**
Pulmonary TB	8 (28.6)	20 (71.4)	28	0.021
Extrapulmonary TB	6 (37.5)	10 (62.5)	16	
CNS TB	8 (80.0)	2 (20.0)	10	
Disseminated TB	4 (66.7)	2 (33.3)	6	

There was a **statistically significant association between type of tuberculosis and hyponatraemia (p = 0.021)**.

Hyponatraemia was most prevalent among patients with **CNS TB (80.0%)**, followed by those with **disseminated TB (66.7%)**, indicating a strong association of sodium imbalance with more severe or systemic forms of the disease. In contrast, lower proportions were observed in **extrapulmonary TB (37.5%)** and **pulmonary TB (28.6%)**.

The majority of patients with pulmonary and extrapulmonary TB had normal sodium levels, whereas hyponatraemia predominated in CNS and disseminated TB cases.

Overall, these findings suggest that **hyponatraemia is significantly more common in CNS and disseminated tuberculosis**, possibly due to mechanisms such as SIADH or extensive disease involvement affecting electrolyte regulation.

TABLE 3B. Association Between Type of TB and Hypocalcaemia (N = 60)

Type of TB	Hypocalcaemia Present n (%)	Hypocalcaemia Absent n (%)	Total	p-value
Pulmonary TB	6 (21.4)	22 (78.6)	28	0.048
Extrapulmonary TB	4 (25.0)	12 (75.0)	16	
CNS TB	6 (60.0)	4 (40.0)	10	
Disseminated TB	2 (33.3)	4 (66.7)	6	

There was a **statistically significant association between type of tuberculosis and hypocalcaemia (p = 0.048)**.

Hypocalcaemia was most frequently observed in patients with **CNS TB (60.0%)**, followed by **disseminated TB (33.3%)**, indicating a higher occurrence in more severe or systemic forms of tuberculosis. In contrast, lower proportions were seen in **extrapulmonary TB (25.0%)** and **pulmonary TB (21.4%)**.

Most patients with pulmonary and extrapulmonary TB had normal calcium levels, whereas hypocalcaemia was comparatively more common in CNS TB.

Overall, these findings suggest that **hypocalcaemia is significantly associated with the type of TB**, with higher prevalence in CNS involvement, possibly due to disease severity, nutritional factors, or metabolic alterations related to tuberculosis.

TABLE 4. Univariate Logistic Regression for Hyponatraemia (N = 60)

Category	Hyponatraemia Present n (%)	Hyponatraemia Absent n (%)	OR (95% CI)	p-value
Age group				
≤50 years	12 (28.6)	30 (71.4)	Reference	0.003
>50 years	14 (77.8)	4 (22.2)	4.8 (1.6-14.2)	
Gender				
Female	8 (33.3)	16 (66.7)	Reference	0.041
Male	18 (50.0)	18 (50.0)	2.0 (0.8-5.1)	
Type of TB				
Pulmonary TB	8 (28.6)	20 (71.4)	Reference	-
Extrapulmonary TB	6 (37.5)	10 (62.5)	1.5 (0.4-5.3)	0.512
CNS TB	8 (80.0)	2 (20.0)	6.5 (1.8-22.9)	<0.001
Disseminated TB	4 (66.7)	2 (33.3)	2.5 (0.6-10.1)	0.201
Diabetes Mellitus				
No	16 (34.8)	30 (65.2)	Reference	0.006
Yes	10 (71.4)	4 (28.6)	4.7 (1.5-14.5)	
HIV Status				
No	22 (40.7)	32 (59.3)	Reference	

Yes	4 (66.7)	2 (33.3)	2.9 (0.5-15.3)	0.089

In the univariate logistic regression analysis, several factors were found to be associated with hyponatraemia among tuberculosis patients. Patients aged **>50 years had significantly higher odds of hyponatraemia (OR = 4.8; 95% CI: 1.6–14.2; p = 0.003)** compared to those ≤50 years, indicating age as an important risk factor.

With respect to gender, **males had higher odds (OR = 2.0)** of hyponatraemia compared to females; however, the confidence interval includes unity, suggesting a weaker association despite statistical significance (p = 0.041).

Regarding the type of tuberculosis, **CNS TB showed a strong and statistically significant association with hyponatraemia (OR = 6.5; 95% CI: 1.8–22.9; p < 0.001)** when compared to pulmonary TB (reference group). Although disseminated TB also showed increased odds (OR = 2.5), this was not statistically significant (p = 0.201). Extrapulmonary TB did not show a significant association. Among comorbidities, **patients with diabetes mellitus had significantly higher odds of hyponatraemia (OR = 4.7; 95% CI: 1.5–14.5; p = 0.006)** compared to non-diabetics.

Although **HIV-positive patients had higher odds (OR = 2.9)** of hyponatraemia, this association was not statistically significant (p = 0.089). Overall, **age >50 years, CNS tuberculosis, and diabetes mellitus were significant predictors of hyponatraemia in univariate analysis**, and these variables were considered for multivariate analysis.

TABLE 5. Multivariate Logistic Regression for Hyponatraemia (N = 60)

Category	AOR (95% CI)	p-value
Age Group		
≤50 years	Reference	0.018
>50 years	3.2 (1.2-8.6)	
Gender		
Female	Reference	0.211
Male	1.6 (0.6-4.2)	
Type of TB		
Pulmonary TB	Reference	-
Extrapulmonary TB	1.4 (0.4-5.1)	0.612
CNS TB	5.8 (1.9-17.4)	0.002
Disseminated TB	2.1 (0.5-8.9)	0.298
Diabetes Mellitus		
No	Reference	0.031
Yes	3.1 (1.1-8.9)	

In the multivariate logistic regression analysis, after adjusting for potential confounders, **age >50 years, CNS tuberculosis, and diabetes mellitus remained independent predictors of hyponatraemia**. Patients aged **>50 years had significantly higher adjusted odds of hyponatraemia (AOR = 3.2; 95% CI: 1.2–8.6; p = 0.018)** compared to those ≤50 years.

Among the different types of tuberculosis, **CNS TB showed a strong independent association (AOR = 5.8; 95% CI: 1.9–17.4; p = 0.002)** when compared to pulmonary TB (reference group). Extrapulmonary and disseminated TB did not show statistically significant associations after adjustment.

Diabetes mellitus was also an independent predictor (AOR = 3.1; 95% CI: 1.1–8.9; p = 0.031), indicating that diabetic patients were more likely to develop hyponatraemia. Although male gender showed increased odds in univariate analysis, it was **not statistically significant in multivariate analysis (AOR = 1.6; p = 0.211)**, suggesting that its effect may be confounded by other variables.

Overall, these findings indicate that **advanced age, CNS involvement, and diabetes mellitus independently increase the risk of hyponatraemia in tuberculosis patients**, highlighting the need for

closer electrolyte monitoring in these high-risk groups.

DISCUSSION

In the present study, **hyponatraemia was observed in 43.3% (n=26)** and **hypocalcaemia in 30.0% (n=18)** of tuberculosis patients, indicating that electrolyte disturbances are frequent and clinically relevant. Notably, hyponatraemia was markedly higher in **CNS tuberculosis (80.0%)** and **disseminated tuberculosis (66.7%)**, highlighting its association with severe disease.

The prevalence of **hyponatraemia (43.3%)** in our study closely aligns with findings from Kalaiyarasan et al. [17], who reported electrolyte abnormalities in **approximately 46%** of pulmonary tuberculosis patients. Similarly, Soni et al. [18] observed hyponatraemia in **around 38–45%** of cases prior to treatment, which is comparable to our findings. Kaur et al. [20] also documented electrolyte imbalance in nearly **30–40%** of newly diagnosed tuberculosis patients.

Shyama et al. [16] demonstrated significant biochemical alterations in both pulmonary and extrapulmonary tuberculosis, with electrolyte disturbances more prominent in extrapulmonary disease, supporting our observation that abnormalities increase with disease severity. Ganiger et al. [19] reported electrolyte derangements in **about 35–40%** of patients, slightly lower than our findings, possibly due to differences in study population and disease spectrum.Taken together, our observed prevalence falls within the upper range of reported values, suggesting a substantial burden of electrolyte imbalance in our cohort.

In our study, hyponatraemia was present in **43.3%**, with a striking increase in **CNS TB (80.0%)**, compared to **28.6% in pulmonary TB**, indicating a strong association with neurological involvement. Yoshida et al. [21] reported hyponatraemia in **51%** of pulmonary tuberculosis patients, slightly higher than our overall prevalence but without stratification into CNS involvement.

Jafari et al. [22], in a large cohort, documented hyponatraemia in **approximately 45%** of cases, closely matching our results. Importantly, our study demonstrates a gradient of increasing prevalence from pulmonary to disseminated and CNS tuberculosis, which is less clearly defined in earlier studies.

Zetter et al. [23] described vasopressin-mediated mechanisms contributing to hyponatraemia, while Vorherr et al. [24] demonstrated antidiuretic hormone activity within tuberculous lung tissue. Chung et al. [25] reported hyponatraemia in **30–40%** of untreated tuberculosis patients, which is lower than the **80% observed in CNS TB** in our study, reinforcing the role of central nervous system involvement in exacerbating sodium imbalance.Thus, compared to previous literature, our study not only confirms the high prevalence of hyponatraemia but also clearly demonstrates its strong association with disease severity and CNS involvement.

In contrast to hyponatraemia, our study found **hypocalcaemia in 30.0%** of patients, with a higher prevalence in **CNS TB (60.0%)**. This finding differs from earlier studies that predominantly report hypercalcaemia.

Chan et al. [26] documented **hypercalcaemia in 25–27%** of pulmonary tuberculosis patients, particularly in those with extensive disease. Keleştimur et al. [27] similarly reported hypercalcaemia in **20–25%** of cases, attributing it to increased 1,25-dihydroxyvitamin D production by activated macrophages. Rajendra et al. [28] described cases of severe hypercalcaemia, further supporting this mechanism.

However, our finding of **30% hypocalcaemia** suggests a different biochemical pattern, likely influenced by factors such as malnutrition, chronic inflammation, and reduced albumin levels. The higher prevalence in CNS tuberculosis (60%) also indicates that severe disease may lead to altered calcium homeostasis in a direction opposite to that reported in granulomatous hypercalcaemia.This contrast highlights the variability of calcium metabolism in tuberculosis and suggests that regional and nutritional factors may significantly influence outcomes.

Our study also demonstrated that electrolyte abnormalities were more frequent in severe forms of tuberculosis, particularly CNS and disseminated disease. Morris et al. [29] reported significant biochemical disturbances in severe pulmonary tuberculosis, supporting the association between disease severity and metabolic imbalance.

Olalekan et al. [30] observed electrolyte abnormalities in **approximately 40–50%** of

tuberculosis patients, with hyponatraemia being the most common disturbance. This closely corresponds to our finding of **43.3%**, reinforcing the consistency of sodium imbalance across different populations.

Furthermore, our study identified **age >50 years (AOR 3.2)**, **CNS TB (AOR 5.8)**, and **diabetes mellitus (AOR 3.1)** as independent predictors of hyponatraemia, providing additional clinical context that is not consistently detailed in earlier studies.

CONCLUSION

The present study demonstrates that electrolyte abnormalities are highly prevalent among tuberculosis patients, with hyponatraemia affecting 43.3% of cases and showing a strong association with severe forms of the disease, particularly CNS tuberculosis. A clear gradient was observed, with increasing sodium imbalance corresponding to greater disease severity. In contrast, hypocalcaemia was noted in 30% of patients, differing from earlier reports that predominantly describe hypercalcaemia, thereby suggesting possible population-specific or disease-related variations. These findings emphasize the need for routine monitoring of electrolyte levels in tuberculosis patients, as early identification and correction of these abnormalities may improve clinical outcomes and provide better insight into the metabolic and systemic impact of the disease.

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