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Serum Albumin as a Predictor of Disease Severity in Pulmonary Tuberculosis: A Hospital-Based Cross-Sectional Study

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Abstract

Background: Pulmonary tuberculosis (PTB) remains a major cause of hospital admission in India. Serum albumin, a negative acute phase protein that also reflects nutritional status, is measured routinely and may help identify patients with severe disease.

Objective: To evaluate the role of serum albumin as a marker in assessing the severity of pulmonary tuberculosis.

Methods: This hospital-based cross-sectional study included 100 adults with sputum-positive or radiologically diagnosed PTB admitted to the hospitals attached to J.J.M. Medical College, Davangere, between June 2025 and June 2026. Clinical features, vital signs, haemogram, liver function tests, sputum AFB, CBNAAT and chest X-ray were recorded. Albumin was compared across severity grades, bacillary load and radiological findings, with ROC analysis.

Results: The mean age was 42.16 ± 14.09 years, and 70.0% were men. Hypoalbuminaemia (below 3.5 g/dL) was present in 61.0%. Mean albumin fell from 3.74 ± 0.38 g/dL in mild to 3.19 ± 0.32 g/dL in moderate and 2.56 ± 0.41 g/dL in severe disease ($p < 0.001$). Albumin was lower with 3+ smear grade ($p = 0.003$), higher CBNAAT load ($p = 0.030$), far advanced disease, cavitation and bilateral involvement ($p < 0.001$). It correlated with SpO_2 ($r = 0.531$), haemoglobin ($r = 0.662$), radiographic zones ($r = -0.606$) and hospital stay ($r = -0.628$). At 2.7 g/dL or below, albumin identified

severe disease with an AUC of 0.932, sensitivity of 86.8% and specificity of 96.8%.

Conclusion: Serum albumin falls with increasing severity of pulmonary tuberculosis and is a simple, inexpensive marker for identifying severe disease at admission.

Keywords: Pulmonary tuberculosis; Serum albumin; Hypoalbuminaemia; Disease severity; Chest radiography

Introduction

Tuberculosis (TB), caused by Mycobacterium tuberculosis, is still one of the leading infectious causes of illness and death in the world, and most of this burden falls on low- and middle-income countries. The disease spreads through airborne droplets from people with active pulmonary disease, so every patient with untreated smear-positive or cavitary TB keeps transmission going within the household and the wider community. Infection does not always lead to disease. In most infected people the bacilli are held in check by a cell-mediated immune response built around activated macrophages and T lymphocytes, and active TB develops only in the fraction of people in whom this containment fails or later breaks down.[1]

India carries the largest share of the global TB burden. Based on World Health Organization estimates for 2022, TB incidence in India was about 210 per 100,000 population, with roughly 75 new smear-positive cases per 100,000 population every year.[2] The National Tuberculosis Elimination Programme has widened access to free diagnosis, including cartridge-based nucleic acid amplification testing (CBNAAT), and to free treatment. Even so, late presentation, coexisting diabetes mellitus, HIV infection, alcohol use and poverty continue to decide how sick patients are when they first reach hospital. In tertiary care hospitals, a large proportion of patients are admitted with extensive radiological disease, a high bacillary load, marked weight loss, hypoxia or other complications. Deciding early which of these patients are at the greatest risk is difficult, particularly where high dependency and intensive care beds are few and advanced investigations are not always available.

The clinical severity of pulmonary TB depends on both the organism and the host. Host immunity, nutritional status and coexisting illnesses all influence whether the infection stays limited to a small area of lung or spreads through one or both lungs. Undernutrition is especially relevant in India. It weakens cell-mediated immunity, raises the risk that latent infection will progress to active disease, and is associated with drug toxicity, relapse and death once TB has developed.[3] The relationship runs in both directions. Active TB causes anorexia, fever, increased resting energy expenditure and breakdown of muscle protein, and these changes worsen the nutritional state of a patient who was often undernourished to begin with. In a large cluster-randomised trial from Jharkhand, food rations given to the household contacts of patients with pulmonary TB reduced TB incidence among those contacts by 39% for all forms of TB and by 48% for microbiologically confirmed pulmonary TB.[4]

Serum albumin sits at the meeting point of nutrition and inflammation. It is the most abundant protein in plasma and is made only by the liver. It maintains colloid oncotic pressure, carries fatty acids, bilirubin, hormones, calcium and many drugs, and acts as an antioxidant in the extracellular fluid. Albumin is also a negative acute phase reactant. During an inflammatory response the liver increases the synthesis of positive acute phase proteins such as C-reactive protein, fibrinogen and ferritin, and reduces the synthesis of albumin, transferrin

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and transthyretin.[5] In active TB, pro-inflammatory cytokines, particularly interleukin-1, interleukin-6 and tumour necrosis factor-alpha, suppress albumin production in hepatocytes, while increased capillary permeability moves albumin out of the circulation into the tissues. A poor dietary protein intake adds to the fall. A low serum albumin in a patient with TB therefore reflects two processes at the same time: the intensity of the systemic inflammatory response and the depth of protein-energy depletion.

Both processes matter for host defence against M. tuberculosis. Hypoalbuminaemia has been linked with impaired T-cell mediated immunity, which is the main mechanism that keeps the bacilli confined within granulomas. Albumin also binds and neutralises reactive oxygen species and modulates the activation of immune cells, so its depletion may leave tissues more exposed to oxidative injury during infection. Low albumin changes the way drugs are handled. Several anti-tubercular drugs are bound to plasma proteins, and reduced binding can alter free drug concentrations, drug clearance and the risk of hepatotoxicity. Inflammation and hypoalbuminaemia are further linked with disturbances of haemostasis. Inflammatory mediators shift the balance towards a prothrombotic state, and in advanced or disseminated disease, involvement of the liver may reduce the synthesis of clotting factors along with albumin.

Clinical studies have tried to relate these mechanisms to the course of TB. In a retrospective cohort from Beijing, serum albumin rose steadily during treatment in patients who had good outcomes but showed no significant change in those who had poor outcomes, suggesting that the early albumin response could identify patients at risk of treatment failure.[6] Among adults hospitalised with TB at a referral hospital in Brazil, a low serum albumin at admission was independently associated with in-hospital death after adjustment for age, comorbidities, HIV co-infection and previous treatment default.[7] A Japanese cohort with a large proportion of elderly patients reached a similar conclusion, with hypoalbuminaemia and lymphocytopenia on admission predicting in-hospital mortality.[8] Low albumin has also been related to the clinical picture at diagnosis. In a cross-sectional study of patients with pulmonary TB from Shandong province, China, hypoalbuminaemia was associated with a higher clinical severity score and with a positive sputum smear.[9]

The clinical presentation of pulmonary TB covers a wide range. Some patients have a few weeks of cough and low-grade fever with a small upper lobe infiltrate, while others arrive with months of weight loss, breathlessness, haemoptysis and bilateral cavitary disease. Patients at the severe end of this range are more likely to have respiratory failure, need oxygen or ventilatory support, develop drug-induced hepatitis, and die during the intensive phase of treatment. Many of these deaths happen in the first weeks after diagnosis, before the effect of anti-tubercular drugs is established. Early recognition of patients at high risk allows closer monitoring of vital signs and oxygen saturation, earlier admission to a high dependency unit, more careful monitoring of liver function, and timely nutritional support. In a busy government hospital, where a single unit may admit many patients with TB in a week, simple tools that help sort patients by risk at the time of admission have real practical value. Such tools need to be cheap, quick, available round the clock and easy to interpret by the admitting doctor without specialist input. Laboratory markers already measured as part of routine

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admission work-up meet these needs better than tests that require extra cost, special equipment or referral.

In routine practice, the severity of pulmonary TB is judged from several sources. Clinicians look at symptoms such as weight loss, breathlessness and haemoptysis, at vital signs including oxygen saturation, at the sputum smear grade, and at the extent of disease and the presence of cavities on the chest radiograph. Radiological severity can be graded with simple scores. One validated score adds the estimated percentage of lung affected to a fixed value when cavitation is present, and this score correlated with sputum smear grade, body mass index, haemoglobin, lung function and the sputum result at two months of treatment.[10] These measures are useful, but each has limits. Smear grading depends on the quality of the sputum sample and of the laboratory, CBNAAT is semi-quantitative, and radiograph reading varies between observers. A low-cost blood test that tracks with clinical, microbiological and radiological severity would add an objective measure that is available at the time of admission.

Serum albumin fits this role well on practical grounds. It is measured as part of the routine liver function test in almost every hospital in India, it costs very little, and results are usually available within a few hours. Despite this, albumin is not part of routine TB severity assessment. Much of the published work on albumin in TB comes from China, Japan, Brazil and other settings outside India, and most of it looks at mortality or treatment outcome rather than at disease severity at the time of presentation. Patients in Indian hospitals differ from many of these cohorts. Undernutrition is far more common, diabetes is frequent, presentation is often late, and many patients have extensive disease when first seen. Findings from other populations may therefore not apply directly. There is little information from Karnataka on how serum albumin at admission relates to the clinical, sputum and radiological severity of pulmonary TB in hospitalised adults.

The present study was therefore undertaken to evaluate serum albumin as a marker of disease severity in adults admitted with pulmonary TB to two teaching hospitals in Davangere, Karnataka. Serum albumin at admission was compared with clinical features and vital signs, sputum AFB smear grade, CBNAAT bacterial load and the radiological extent of disease on chest X-ray. The aim was to find out whether a simple, inexpensive and widely available test can help clinicians recognise patients with more severe pulmonary TB early in their hospital stay, so that closer monitoring and nutritional support can be directed to those who need them most.

Aims And Objectives

The objective of the study was to evaluate the role of serum albumin as a marker in assessing the severity of pulmonary tuberculosis.

Materials and Methods

Study Design

The present study was a hospital-based cross-sectional study. The study was initiated after approval was obtained from the Institutional Scientific Committee and the Institutional Ethics Committee of J.J.M. Medical College, Davangere (Ref. No. JJMMC/IEC-Syc-55-2025, Protocol No. Syc-55-2025, dated 05-06-2025).

Study Setting and Duration

The study was carried out in the Department of General Medicine of Chigateri General Hospital and Bapuji Hospital, attached to J.J.M. Medical College, Davangere, Karnataka. Patients were recruited from the General ward (fever ward), Tuberculosis ward, High Dependency Unit (HDU) and Medical Intensive Care Unit (MICU) of

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these hospitals. The study was conducted over a period of one year, from June 2025 to June 2026.

Sample Size and Calculation

The sample size was calculated with the formula for estimating a single proportion, $n = Z^2 \times p \times (1 - p) / d^2$, where Z is 1.96 at the 5% level of significance, p is the anticipated proportion of 15% (0.15) and d is the absolute precision of 7% (0.07). This gave $n = 3.8416 \times 0.15 \times 0.85 / 0.0049 = 99.96$, which was rounded to 100. Hence, the goal sample size was N = 100. The study included 100 patients with pulmonary tuberculosis who satisfied the inclusion and exclusion criteria.

Sampling Technique

A random sampling technique was used. All patients admitted to the General ward (fever ward), Tuberculosis ward, HDU and MICU of Chigateri General Hospital and Bapuji Hospital during the study period were evaluated, and 100 patients who satisfied the inclusion and exclusion criteria were selected at random and enrolled after written informed consent was obtained.

Inclusion Criteria

- All patients above 18 years of age.
- Sputum positive tuberculosis patients.
- Radiologically detected pulmonary tuberculosis patients.
- Patients consenting to participate in the study.

Exclusion Criteria

- Patients below 18 years of age.
- Patients with known liver disease, nephrotic syndrome, protein losing enteropathy.
- Patients on corticosteroid or immunosuppressive therapy.
- Patients with multidrug resistant tuberculosis.
- Patients not consenting to the study.

Data Collection Procedure

The purpose and procedure of the study were explained to every eligible patient in a language they understood. Written informed consent was obtained in English or Kannada before enrolment, using the consent form approved by the Institutional Ethics Committee. The identity and personal details of the participants were kept confidential throughout the study.

Data were recorded in a pre-designed case proforma. A detailed history was taken from every patient, including name, age, sex and in-patient number. The presenting complaints of fever, cough with expectoration, weight loss, haemoptysis, loss of appetite, myalgia and malaise were recorded, along with the duration of each. A history of severe weight loss, seizures, prolonged cough, fever and other symptoms was also noted. Past history, personal history (including smoking and alcohol use) and treatment history, including any previous anti-tubercular treatment, were recorded.

A detailed general physical examination was performed, noting build and nourishment and the presence of pallor, cyanosis, clubbing, icterus, pedal oedema and lymphadenopathy. Vital signs, namely pulse rate, respiratory rate, blood pressure, temperature and oxygen saturation, were recorded. Systemic examination of the cardiovascular system, respiratory system, abdomen and central nervous system was carried out and the findings were entered in the proforma.

The following investigations were done for all patients: complete haemogram, liver function test including serum albumin, sputum for acid-fast bacilli (AFB) and CBNAAT, and chest X-ray PA view. Sputum samples were sent according to the protocol followed for patients admitted to the fever wards. An adequate amount of venous blood was drawn under sterile aseptic

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precautions. The samples were analysed in the Department of Pathology, J.J.M. Medical College, and the results were provided within four hours. Serum albumin was expressed in g/dL, and the normal serum albumin level of 3.5 to 5.0 g/dL was used as the reference range in the study. Values below 3.5 g/dL were taken as low serum albumin (hypoalbuminaemia).

Disease severity was assessed from the clinical features and vital signs at admission, the sputum AFB smear grade and CBNAAT result, and the radiological extent of disease on the chest X-ray. The level of serum albumin was then compared across these measures of severity.

Imaging Protocol

A chest X-ray in the posteroanterior (PA) view was obtained for every patient at the time of admission. The radiographs were examined for the side of involvement (unilateral or bilateral), the zones involved, and the type of lesion, including infiltrates, consolidation, cavitation, nodular shadows, fibrosis, miliary mottling and pleural effusion. The radiological extent of disease was graded as minimal, moderately advanced or far advanced. Minimal disease referred to lesions of slight to moderate density without demonstrable cavitation, confined to a small part of one or both lungs. Moderately advanced disease referred to lesions in one or both lungs that did not exceed the volume of one lung in total, with any cavitation less than 4 cm in total diameter. Far advanced disease referred to lesions more extensive than moderately advanced disease. The presence or absence of cavitation was recorded separately for each patient.

Data Analysis

The data collected were entered into a Microsoft Excel spreadsheet and analysed using SPSS version 25.0. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as descriptive statistics in the form of mean and standard deviation. The distribution of continuous variables was checked for normality using the Shapiro-Wilk test.

The association between categorical variables, such as serum albumin category (normal or low) and sputum smear grade, CBNAAT result, radiological extent and presence of cavitation, was tested using the Chi-square test, or Fisher's exact test where expected cell counts were small. Mean serum albumin between two groups was compared using the independent samples t-test, or the Mann-Whitney U test for data that were not normally distributed. Mean serum albumin across more than two severity grades was compared using one-way analysis of variance (ANOVA), or the Kruskal-Wallis test where appropriate. The correlation of serum albumin with continuous clinical and laboratory variables was assessed using Pearson's or Spearman's correlation coefficient, depending on the distribution of data. Receiver operating characteristic (ROC) curve analysis was used to assess the ability of serum albumin to identify severe disease, and the area under the curve, sensitivity and specificity were calculated. Other tests were applied at the time of analysis as required by the distribution of data. A p value less than 0.05 was considered statistically significant.

Results

A total of 100 adults with pulmonary TB were included. The mean age was 42.16 ± 14.09 years (range 18 to 82 years), and the largest age group was 31 to 40 years (27.0%). Men made up 70.0% of the study population, giving a male to female ratio of 2.33:1. All patients had cough and fever, with a mean cough duration of 6.04 ± 2.60 weeks and a mean fever duration of 24.45 ± 9.32 days. Significant weight loss was reported by 64.0%,

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breathlessness by 51.0% and haemoptysis by 18.0%. At admission, 44.0% had tachycardia above 100/min, 24.0% had tachypnoea above 24/min and 13.0% had an SpO_2 below 90% on room air. Anaemia was present in 91.0% of patients (mean haemoglobin 10.39 ± 1.78 g/dL), and leucocytosis above 11,000 cells/mm³ in 37.0% (Table 1).

Table 1: Baseline characteristics of the study participants (n = 100)

Characteristic	Value
Age (years), Mean ± SD	42.16 ± 14.09
Age group: 18 to 30 / 31 to 40 / 41 to 50 / 51 to 60 / above 60 years, n	21 / 27 / 25 / 17 / 10
Male / Female, n (%)	70 (70.0) / 30 (30.0)
Cough, n (%)	100 (100.0)
Fever, n (%)	100 (100.0)
Significant weight loss, n (%)	64 (64.0)
Breathlessness, n (%)	51 (51.0)
Haemoptysis, n (%)	18 (18.0)
Duration of cough (weeks), Mean ± SD	6.04 ± 2.60
Duration of fever (days), Mean ± SD	24.45 ± 9.32
Pulse rate (/min), Mean ± SD	99.72 ± 14.00
Respiratory rate (/min), Mean ± SD	21.45 ± 5.21
SpO_2 on room air (%), Mean ± SD	93.64 ± 4.35
SpO_2 below 90%, n (%)	13 (13.0)
Haemoglobin (g/dL), Mean ± SD	10.39 ± 1.78
Anaemia, n (%)	91 (91.0)
Total leucocyte count (cells/mm³), Mean ± SD	9,948 ± 2,954
Total protein (g/dL), Mean ± SD	6.76 ± 0.62

*$p < 0.05$ considered statistically significant

The mean serum albumin was 3.13 ± 0.62 g/dL, with a median of 3.15 g/dL (IQR 2.60 to 3.60). Hypoalbuminaemia below 3.5 g/dL was found in 61.0% of patients; it was mild (3.0 to 3.4 g/dL) in 19.0%, moderate (2.5 to 2.9 g/dL) in 28.0% and severe (below 2.5 g/dL) in 14.0%. Sputum AFB smear was positive in 91.0% of patients, most often at grade 1+ (41.0%), and CBNAAT detected MTB in 97.0%. On chest X-ray, 47.0% had bilateral involvement, 37.0% had cavitation and 33.0% had far advanced disease. Disease was graded as mild in 32.0%, moderate in 30.0% and severe in 38.0% of patients. Eighteen patients (18.0%) needed HDU or MICU care, the mean hospital stay was 10.69 ± 5.42 days, and four patients (4.0%) died (Table 2).

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Table 2: Serum albumin, microbiological, radiological and severity profile (n = 100)

Variable	n (%)
Serum albumin (g/dL), Mean ± SD	3.13 ± 0.62
Albumin ≥3.5 g/dL (normal)	39 (39.0)
Albumin 3.0 to 3.4 g/dL	19 (19.0)
Albumin 2.5 to 2.9 g/dL	28 (28.0)
Albumin <2.5 g/dL	14 (14.0)
Sputum AFB: negative / scanty / 1+ / 2+ / 3+	9 / 12 / 41 / 24 / 14
CBNAAT: not detected / very low / low / medium / high	3 / 9 / 26 / 37 / 25
Bilateral involvement on CXR	47 (47.0)
Cavitation on CXR	37 (37.0)
CXR extent: minimal / moderately advanced / far advanced	40 / 27 / 33
Disease severity: mild / moderate / severe	32 / 30 / 38
HDU or MICU admission	18 (18.0)
Duration of hospital stay (days), Mean ± SD	10.69 ± 5.42
Death	4 (4.0)

*p < 0.05 considered statistically significant

Mean serum albumin fell in a graded manner with increasing disease severity: 3.74 ± 0.38 g/dL in mild disease, 3.19 ± 0.32 g/dL in moderate disease and 2.56 ± 0.41 g/dL in severe disease ($F = 86.02$, $p < 0.001$). Tukey post hoc analysis showed significant differences between every pair of grades ($p < 0.001$ for each). Low serum albumin was present in 12.5% of patients with mild disease, 73.3% with moderate disease and 92.1% with severe disease, and the association was statistically significant ($\chi^2 = 49.01$, $df = 2$, $p < 0.001$) (Table 3, Figure 1).

Table 3: Serum albumin according to disease severity

Variable	Mild (n = 32)	Moderate (n = 30)	Severe (n = 38)	p value
Serum albumin (g/dL), Mean ± SD	3.74 ± 0.38	3.19 ± 0.32	2.56 ± 0.41	<0.001* (ANOVA, F = 86.02)
Normal albumin (≥3.5 g/dL), n (%)	28 (87.5)	8 (26.7)	3 (7.9)	<0.001* (χ^2 = 49.01)
Low albumin (<3.5 g/dL), n (%)	4 (12.5)	22 (73.3)	35 (92.1)	

*p < 0.05 considered statistically significant

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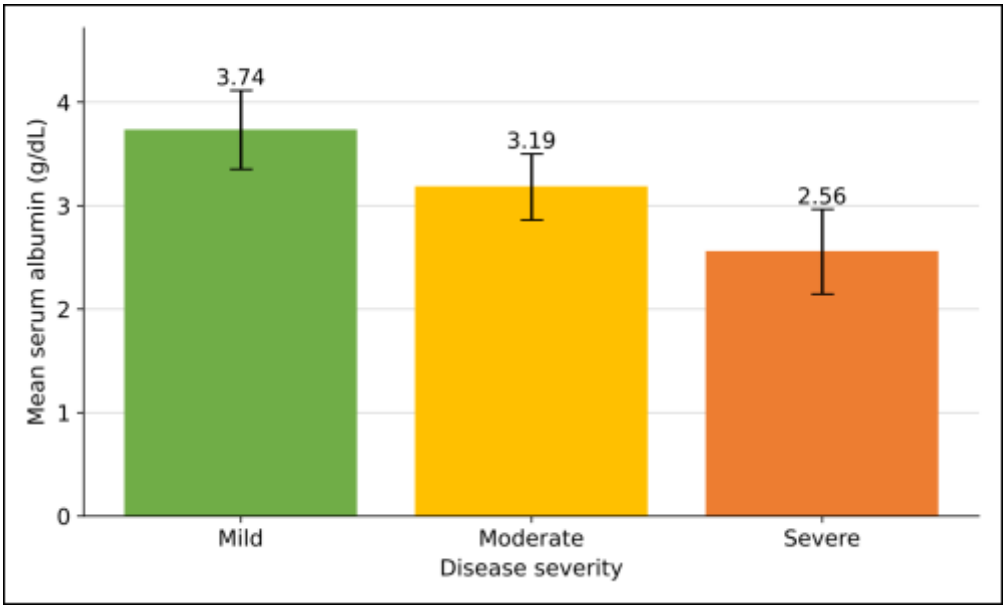

Figure 1: Mean serum albumin across grades of disease severity (error bars show SD)

Serum albumin was related to both bacillary load and radiological extent (Table 4). It differed significantly across sputum smear grades ($F = 4.34$, $p = 0.003$) and was lowest with a 3+ smear (2.66 ± 0.39 g/dL); on post hoc testing, the 3+ group differed from the 1+ ($p = 0.028$) and scanty ($p = 0.002$) groups. Albumin also fell as CBNAAT bacterial load increased, from 3.31 ± 0.70 g/dL with very low load to 2.96 ± 0.56 g/dL with high load ($F = 2.81$, $p = 0.030$). The association with chest X-ray findings was stronger. Mean albumin was 3.57 ± 0.53 g/dL in minimal, 3.16 ± 0.36 g/dL in moderately advanced and 2.56 ± 0.41 g/dL in far advanced disease ($F = 46.08$, $p < 0.001$). It was significantly lower in patients with cavitation (2.79 ± 0.50 against 3.32 ± 0.60 g/dL, $t = 4.60$, $p < 0.001$) and with bilateral involvement (2.81 ± 0.59 against 3.40 ± 0.50 g/dL, $t = 5.34$, $p < 0.001$).

Table 4: Serum albumin according to sputum AFB grade, CBNAAT bacterial load and chest X-ray findings

Variable	**n**	**Serum albumin (g/dL), Mean ± SD**	**Test value, p value**
Sputum AFB smear grade			
Negative	9	3.27 ± 0.78	F = 4.34, p = 0.003*
Scanty	12	3.54 ± 0.62	
1+	41	3.20 ± 0.63	
2+	24	3.00 ± 0.47	
3+	14	2.66 ± 0.39	
CBNAAT bacterial load			
MTB not detected	3	4.03 ± 0.58	F = 2.81, p = 0.030*
Very low	9	3.31 ± 0.70	
Low	26	3.23 ± 0.60	
Medium	37	3.04 ± 0.60	

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High	25	2.96 ± 0.56	
Radiological extent			
Minimal	40	3.57 ± 0.53	F = 46.08, p <0.001*
Moderately advanced	27	3.16 ± 0.36	
Far advanced	33	2.56 ± 0.41	
Cavitation			
Present	37	2.79 ± 0.50	t = 4.60, p <0.001*
Absent	63	3.32 ± 0.60	
Lung involvement			
Unilateral	53	3.40 ± 0.50	t = 5.34, p <0.001*
Bilateral	47	2.81 ± 0.59	

*p < 0.05 considered statistically significant

Serum albumin correlated strongly and positively with total protein (r = 0.783) and haemoglobin (r = 0.662), and moderately with SpO_2 (r = 0.531). It correlated negatively with the duration of hospital stay (r = -0.628), the number of radiographic zones involved (r = -0.606), total leucocyte count (r = -0.523), respiratory rate (r = -0.464) and pulse rate (r = -0.402). All these correlations were significant at p < 0.001. Albumin was not significantly correlated with the duration of cough (r = -0.090, p = 0.374) or fever (r = -0.188, p = 0.060) (Table 5).

Table 5: Correlation of serum albumin with clinical, laboratory and radiological parameters

Parameter	Pearson's r	p value	Interpretation
Duration of cough (weeks)	-0.090	0.374	Very weak negative
Duration of fever (days)	-0.188	0.060	Very weak negative
Pulse rate (/min)	-0.402	<0.001*	Moderate negative
Respiratory rate (/min)	-0.464	<0.001*	Moderate negative
Systolic BP (mmHg)	0.338	<0.001*	Weak positive
SpO_2 (%)	0.531	<0.001*	Moderate positive
Haemoglobin (g/dL)	0.662	<0.001*	Strong positive
Total leucocyte count	-0.523	<0.001*	Moderate negative
Total protein (g/dL)	0.783	<0.001*	Strong positive
Number of CXR zones involved	-0.606	<0.001*	Strong negative
Duration of hospital stay (days)	-0.628	<0.001*	Strong negative

*p < 0.05 considered statistically significant

On ROC analysis, serum albumin discriminated severe from non-severe disease with an AUC of 0.932 (95% CI 0.874 to 0.990, p < 0.001). The optimal cut-off by the Youden index was 2.7 g/dL or below, at which sensitivity was 86.8%,

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specificity 96.8%, positive predictive value 94.3%, negative predictive value 92.3% and overall accuracy 93.0% (Table 6, Figure 2).

Table 6: Diagnostic performance of serum albumin for severe pulmonary tuberculosis (ROC analysis)

Parameter	**Value**
Area under the curve (AUC)	0.932
Standard error	0.030
95% confidence interval	0.874 to 0.990
p value	<0.001*
Optimal cut-off (Youden index)	≤2.7 g/dL
Sensitivity	86.8%
Specificity	96.8%
Positive predictive value	94.3%
Negative predictive value	92.3%
Diagnostic accuracy	93.0%

*p < 0.05 considered statistically significant

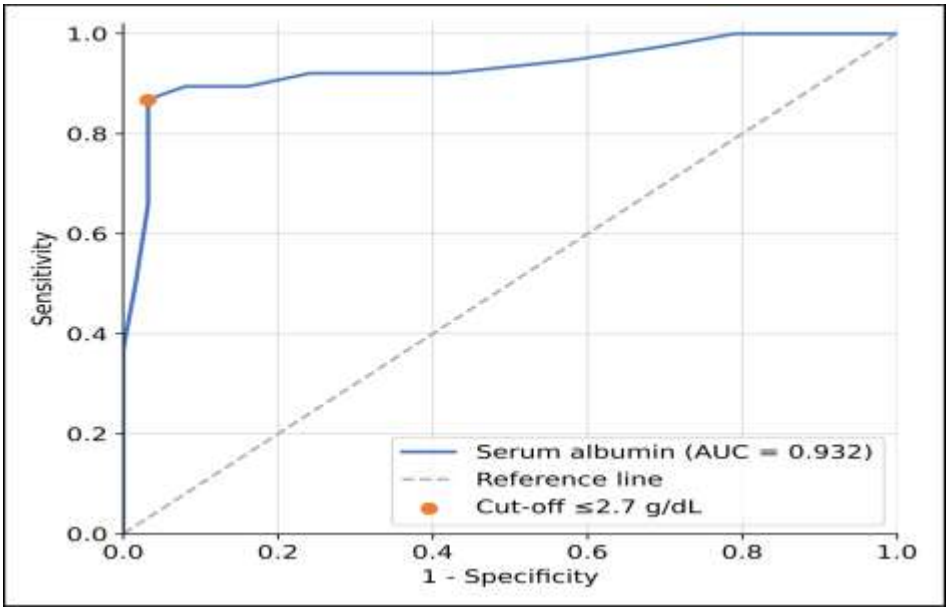

Figure 2: ROC curve of serum albumin for predicting severe pulmonary tuberculosis

Discussion

Pulmonary tuberculosis remains one of the commonest reasons for admission to medical wards in India, and the clinician admitting such a patient has to decide quickly who is likely to deteriorate. The present study examined whether a single, inexpensive test, serum albumin, could help with that decision. In 100 adults admitted with pulmonary TB, serum albumin fell in a graded manner from mild to moderate to severe disease, was lower in patients with heavier bacillary load and more extensive radiographs, and identified severe disease with an area under the ROC curve of 0.932. The sections below compare these findings with published work.

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Demographic profile

The mean age of the patients was 42.16 ± 14.09 years, and 73.0% were 50 years or younger. TB in India continues to fall mainly on adults in their working years, and this pattern was seen in our cohort, where the 31 to 40 year group was the largest (27.0%). Cohorts from East Asia are much older. In the Henan study of older adults by Wang et al, all patients were elderly, and advanced age, retreatment, cavities on chest X-ray and hypoalbuminaemia were independent risk factors for TB-specific death, with a nomogram c-index of 0.844.[11] Honjo et al, who studied 275 hospitalised Japanese patients aged 65 years or more, found that 40 died of TB-related causes.[12] The younger age of our patients is one reason why mortality was low in our series (4.0%).

Men made up 70.0% of the study population, a male to female ratio of 2.33:1. This matches the wider evidence closely. In a meta-analysis of 56 national and sub-national prevalence surveys from low- and middle-income countries, Horton et al reported a pooled male to female prevalence ratio of 2.21 (95% CI 1.92 to 2.54) for bacteriologically positive TB and 2.51 for smear-positive TB, with the highest ratios in South-East Asia.[13] The authors also showed that men were less likely than women to be diagnosed and notified, which may partly explain why men often present later and sicker. Kim et al reported a lower male to female ratio of 1.6:1 among 156 inpatients with pulmonary TB in Seoul.[14] Smoking, alcohol use, occupational exposure and delayed health seeking among men in our region probably account for the higher proportion of men in the present study.

Clinical presentation and vital parameters

Cough and fever were present in every patient, with a mean cough duration of 6.04 ± 2.60 weeks and a mean fever duration of 24.45 ± 9.32 days. Significant weight loss was reported by 64.0% and breathlessness by 51.0%, while haemoptysis was seen in 18.0%. Mukherjee et al, in a study of new pulmonary TB cases at AIIMS Rishikesh, also found cough, fever, breathlessness and haemoptysis to be the commonest symptoms, in that order, both in patients with and without anaemia.[15] The long duration of symptoms before admission in our patients suggests delayed presentation, and the high proportion with breathlessness reflects the extensive disease seen in a tertiary hospital. At admission, 44.0% had tachycardia, 24.0% had tachypnoea and 13.0% had an SpO_2 below 90%. These figures underline that a large share of hospitalised patients had physiological compromise at the time of admission.

In our study, serum albumin was lower in patients with breathlessness (2.85 ± 0.59 against 3.41 ± 0.51 g/dL, $p < 0.001$) and in those with haemoptysis (2.83 ± 0.48 against 3.19 ± 0.63 g/dL, $p = 0.024$). It also correlated with the vital signs, positively with SpO_2 ($r = 0.531$) and systolic blood pressure ($r = 0.338$), and negatively with respiratory rate ($r = -0.464$) and pulse rate ($r = -0.402$). Hu et al found that patients with severe TB in a Chinese multicentre cohort of 298 adults were older and more often had lymphopenia, hypoalbuminaemia, neutrophilia and raised lactate dehydrogenase than those with mild or moderate disease.[16] Our findings fit this picture, in which the systemic inflammatory response that drives tachycardia, tachypnoea and hypoxia also depresses hepatic albumin synthesis.

Haematological and biochemical profile

Anaemia was present in 91.0% of our patients, with a mean haemoglobin of 10.39 ± 1.78 g/dL. Agrawal et al, using the same cut-offs (haemoglobin below 13 g/dL in men and 12 g/dL in women), found anaemia in 81% of

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200 patients with pulmonary TB in Haryana, about 60% of which was due to iron deficiency in smear-positive cases. They also observed that all 12 patients with delayed sputum conversion had moderate or severe anaemia.[17] Mukherjee et al reported mostly mild, normocytic normochromic anaemia, and found that the severity of anaemia was related to body mass index.[15] The rather higher prevalence in our study may reflect the more severe, hospitalised population and the high background prevalence of nutritional anaemia in the region.

Haemoglobin showed a strong positive correlation with serum albumin ($r = 0.662$, $p < 0.001$). Both reflect chronic inflammation and poor nutrition. Inflammatory cytokines reduce iron availability for red cell production and also suppress albumin synthesis, while poor dietary intake lowers both protein and iron stores. Leucocytosis above 11,000 cells/mm³ was found in 37.0% of patients, and the total leucocyte count correlated negatively with albumin ($r = -0.523$). Zalke et al, who followed 50 newly diagnosed patients through treatment in Nagpur, found that ESR, CRP, NLR, TLC and red cell distribution width were raised at diagnosis, fell progressively with treatment, and correlated with radiological severity.[18] Taken together, these results suggest that albumin moves in the opposite direction to the inflammatory markers and carries similar information about disease activity.

Serum albumin and disease severity

Hypoalbuminaemia below 3.5 g/dL was present in 61.0% of our patients, and in 14.0% the level was below 2.5 g/dL. The mean serum albumin was 3.13 ± 0.62 g/dL. Mean albumin fell from 3.74 ± 0.38 g/dL in mild disease to 3.19 ± 0.32 g/dL in moderate and 2.56 ± 0.41 g/dL in severe disease ($F = 86.02$, $p < 0.001$), and every pairwise difference was significant on post hoc testing. Low albumin was found in only 12.5% of patients with mild disease but in 92.1% of those with severe disease ($\chi^2 = 49.01$, $p < 0.001$). The gradient across all three grades is the central finding of this study and supports the use of albumin as a marker of severity rather than only as a marker of nutritional status.

Other investigators have reached similar conclusions using different definitions of severity. Hu et al defined severe TB on clinical and radiological grounds and found hypoalbuminaemia to be one of the features that separated severe from mild or moderate disease.[16] Li et al studied 463 patients with active pulmonary TB in China and classified chest CT lesions as severe when they exceeded the volume of one lung or contained a cavity of 4 cm or more. Severe lesions were present in 61.6%, and on multivariate analysis a lower albumin-globulin ratio was independently associated with severe lesions (OR 0.179, 95% CI 0.084 to 0.383).[19] Wu et al developed a nomogram to separate active from inactive pulmonary TB in 356 patients, and serum albumin was one of five independent predictors, the model reaching an AUC of 0.913.[20]

Serum albumin, bacillary load and radiological extent

Serum albumin was lowest in patients with a 3+ sputum smear (2.66 ± 0.39 g/dL) and differed significantly across smear grades ($F = 4.34$, $p = 0.003$). It also fell as the CBNAAT bacterial load increased, from 3.31 ± 0.70 g/dL with very low load to 2.96 ± 0.56 g/dL with high load ($p = 0.030$). The relationship with bacillary load was weaker than that with clinical severity or radiological extent. This is expected, since smear grade depends on sample quality and on whether a cavity communicates with an airway, and CBNAAT load is only semi-quantitative. Zhang et al found that patients

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with TB and diabetes, who in their series had lower serum albumin, were more likely to have a positive AFB smear (OR 15.81) and cavities, and they observed the heaviest mycobacterial load at higher HbA1c levels.[21]

The relationship between albumin and the chest radiograph was strong. Mean albumin fell from 3.57 ± 0.53 g/dL in minimal disease to 2.56 ± 0.41 g/dL in far advanced disease ($F = 46.08$, $p < 0.001$). It was lower with cavitation (2.79 ± 0.50 against 3.32 ± 0.60 g/dL) and with bilateral involvement (2.81 ± 0.59 against 3.40 ± 0.50 g/dL), both at $p < 0.001$, and it correlated negatively with the number of zones involved ($r = -0.606$). These results agree with those of Li et al, in whom the albumin-globulin ratio was independently linked with severe CT lesions.[19] In the Henan cohort of Wang et al, cavities on chest X-ray and hypoalbuminaemia were both independent predictors of TB-specific mortality, which suggests that the two findings tend to occur together in patients with the most destructive disease.[11]

Albumin, hospital stay and outcome

Serum albumin showed a strong negative correlation with the duration of hospital stay ($r = -0.628$, $p < 0.001$), and patients who died had a mean albumin of only 2.12 ± 0.22 g/dL. Only four patients died, so mortality could not be analysed in depth. Larger cohorts, however, have shown a clear link between low albumin and death. Kim et al used a nutritional risk score that included albumin below 30 g/L and found that a high score was an independent risk factor for in-hospital death (overall mortality 13.5%) and for 30-day survival (OR 16.8, $p < 0.001$), even after adjusting for disease severity.[14] Honjo et al found that lower serum albumin was an independent predictor of TB-related death in elderly patients on competing risk regression (HR 0.228, 95% CI 0.099 to 0.524).[12] In a Brazilian cohort of 373 hospitalised patients, Matos et al found that an albumin of 2.7 g/dL or less was independently associated with in-hospital death (adjusted OR 3.38, 95% CI 1.51 to 7.59), a threshold identical to the cut-off derived in our study.[7]

Diagnostic performance of serum albumin

On ROC analysis, serum albumin identified severe pulmonary TB with an AUC of 0.932 (95% CI 0.874 to 0.990). At a cut-off of 2.7 g/dL or below, sensitivity was 86.8%, specificity 96.8%, positive predictive value 94.3% and negative predictive value 92.3%. This performance compares well with more complex tools. The four-protein plasma panel described by Hu et al separated severe from mild or moderate TB with an AUC of 0.908 (sensitivity 87.5%, specificity 83.8%), and needed proteomic discovery and ELISA validation.[16] The combined clinical score of Li et al for severe CT lesions had an AUC of 0.753.[19] Albumin, in contrast, is already part of the routine liver function test in every hospital laboratory, gives a result within hours and costs very little. For a district or medical college hospital that admits many patients with TB every week, this practical advantage matters as much as the statistical performance, because the test can be applied to every patient at admission without extra blood samples, equipment or referral.

The cut-off of 2.7 g/dL in our study is lower than the conventional limit of 3.5 g/dL. At 3.5 g/dL the sensitivity for severe disease was high, but specificity fell, because many patients with moderate disease also had mildly low albumin. A threshold close to 2.7 g/dL may therefore be more useful at the bedside for flagging patients who need closer monitoring, earlier HDU care and nutritional support. Oza et al found that low BMI and high NLR marked the most severe cases among 75

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patients in Udaipur, and that sputum conversion fell as severity increased.[22] Combining albumin with such simple measures could further improve risk stratification and should be tested in future work.

Strengths and limitations

The study used routine, widely available tests and assessed albumin against clinical, microbiological and radiological measures in the same patients. The main limitations should be noted. It was a single-centre, cross-sectional study, so albumin was measured only once and changes during treatment could not be assessed. The composite disease severity grade included clinical and radiological features, and some of these were also analysed separately against albumin. Liver disease and nephrotic syndrome were excluded, but other causes of low albumin, such as chronic undernutrition without TB, could not be fully separated from the effect of TB itself. C-reactive protein, prealbumin and body mass index were not measured, so composite indices could not be calculated. The small number of deaths did not allow analysis of mortality. Larger prospective studies with follow-up through treatment are needed to confirm the cut-off and to test whether correcting hypoalbuminaemia improves outcome.

Conclusion

Hypoalbuminaemia was present in nearly two-thirds of adults admitted with pulmonary tuberculosis, and serum albumin fell steadily from mild to moderate to severe disease. Lower albumin was associated with a heavier bacillary load on smear and CBNAAT, with cavitation, bilateral involvement and far advanced disease on chest X-ray, and with tachycardia, tachypnoea, hypoxia, anaemia, leucocytosis and a longer hospital stay.

A serum albumin of 2.7 g/dL or below identified severe disease with an AUC of 0.932, a sensitivity of 86.8% and a specificity of 96.8%. Serum albumin is cheap, quick and already part of the routine liver function test, so it can be used at the time of admission to flag patients with severe pulmonary tuberculosis who need closer monitoring, earlier high-dependency care and nutritional support. Larger prospective multicentre studies with serial albumin measurements and follow-up through treatment are needed to validate this cut-off and to test whether correcting hypoalbuminaemia improves outcomes.

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