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Prevalence of Tuberculosis Septic Shock Syndrome among Tuberculosis Patients in Central India

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ABSTRACT

Background: Septic shock is an important but under-recognised complication of tuberculosis (TB), reported in less than 1% of TB cases. This study estimated the prevalence of tuberculosis-associated septic shock among TB patients at a tertiary care centre in central India and assessed the utility of biochemical parameters in identifying impending septic shock.

Methods: This retrospective observational study was conducted in the Department of Pulmonary Medicine, School of Excellence in Pulmonary Medicine (SEPM), Netaji Subhash Chandra Bose Medical College & Hospital, Jabalpur, Madhya Pradesh, India, from August 2024 to January 2025. Records of 400 patients above 13 years of age with a clinical or microbiological diagnosis of tuberculosis were reviewed. Patients with septic shock attributable to other identified causes were excluded. Clinical, biochemical, and radiological parameters, including serum lactate, procalcitonin, and cortisol, were analysed.

Results: Of 400 patients, 98 (24.5%) developed septic shock, of whom 14% were microbiologically confirmed. Septic shock was more common among males (84%) and patients older than 70 years. Serum lactate was significantly associated with septic shock ($p<0.001$), while procalcitonin ($p=0.917$) and cortisol ($p=0.063$) were not. Overall mortality was 5%.

Conclusion: Septic shock occurs in a significant proportion of hospitalised tuberculosis patients and carries a high risk of mortality. A high index of clinical suspicion, supported by serum lactate estimation, along with early institution of anti-tubercular therapy, may help reduce mortality in these patients.

Key-words: Anti-tubercular therapy, Infections, Sepsis, Septic shock, Tubercular septic shock, Tuberculosis

INTRODUCTION

Sepsis is a life-threatening organ dysfunction characterised by a dysregulated host response to infection. Both sepsis and septic shock represent a major problem worldwide and a challenge for physicians because of their increasing incidence and pathophysiological, molecular, genetic, and clinical complexity.[1-3]

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The sepsis cases in India alone were estimated at 11.3 million, with 2.9 million deaths (297.7 per 100,000 population) in 2017.[4]

In patients with sepsis, bacterial infections were most common (77.9%), with the majority being Gram-negative bacteria, followed by fungal infections (9.6%). Multidrug-resistant infections were present in 44.8% of patients with sepsis.[5] The Gram-negative organisms most commonly implicated include Klebsiella and Pseudomonas species.[5] *Mycobacterium tuberculosis*, as an aetiology of sepsis and septic shock, accounting for less than 1% of cases, has been relatively less studied.

Mortality in tuberculosis may be associated with multiple factors such as dissemination of bacilli, adrenal insufficiency, respiratory distress syndrome, or immune

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reconstitution syndrome. A study conducted in South Africa found factors such as increasing age, retroviral disease state, a negative pre-treatment sputum smear status, and retreatment cases to be significant factors associated with increased mortality.[6]

Tubercular septic shock carries higher mortality compared to other bacterial septic shock (79.2% vs. 49.7%).[7] Kethireddy *et al.* highlighted the role of early initiation of anti-tubercular therapy (ATT) in tubercular septic shock to reduce mortality; the persistence of septic shock and delayed initiation of ATT (after 24 hours) were significant predictors of mortality.[7]

The main sites of infection related to sepsis are the respiratory tract/pulmonary parenchyma (43%), the urinary system (16%), the abdomen (14%), the head (associated with fever of unknown origin, FUO) (14%), and other sites/causes (13%).[8] In tuberculosis, the lungs are most commonly affected, among other sites such as lymph nodes, abdomen, CNS, and bone and joint TB. Our study aims to identify the proportion of individuals developing tuberculosis-associated septic shock among patients with tuberculosis.

MATERIALS AND METHODS

Research design- This study was an observational, retrospective study, conducted in the Department of Pulmonary Medicine, School of Excellence in Pulmonary Medicine (SEPM), Netaji Subhash Chandra Bose (NSCB) Medical College & Hospital, Jabalpur, Madhya Pradesh, India, over the period from August 2024 to January 2025.

Methodology- A total of 400 patients with clinically and microbiologically diagnosed tuberculosis were enrolled. Diagnosis was based on clinical, biochemical, and microbiological assessment, and all patients received standard anti-tuberculosis treatment. Baseline clinical, laboratory, and radiological investigations were recorded, including hematological and biochemical parameters, arterial blood gas, lactate, procalcitonin, CRP, chest radiography, and advanced imaging when indicated. TB confirmation was performed using AFB smear, CBNAAT, MGIT culture, and cytopathology where appropriate. In ICU patients, ultrasonography, including IVC assessment, cardiac evaluation, pericardial effusion assessment, and lower-limb venous Doppler, was performed, and APACHE-II scores were calculated. Clinical characteristics, comorbidities, radiological findings, hospital course, and short-term outcomes were documented. Other causes of sepsis were excluded, and the proportion of patients developing septic shock was assessed. Sociodemographic factors were also evaluated for possible associations.

Inclusion criteria- Patients with a clinical or microbiological diagnosis of tuberculosis, more than 13 years of age, admitted at SEPM, Netaji Subhash Chandra Bose Medical College & Hospital, Jabalpur (M.P).

Exclusion criteria- Patients with a diagnosis of and clinical symptoms attributable to Gram-negative sepsis, any organism growth on blood culture, cardiac disease (e.g., congestive heart failure, cardiogenic shock), hypovolemic shock, fungal sepsis, and pregnant women.

Sampling method- Patients admitted to the in-patient department of the health facility during the study period were screened, and those fulfilling the inclusion and exclusion criteria and providing written informed consent were enrolled.

Statistical analysis- Data were presented using tables and figures. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 21.0. Values in tables were presented as means and standard deviations; frequencies were expressed as numbers and percentages. Comparison of categorical data was performed using the Chi-square test. A p-value of less than 0.05 was considered statistically significant.

Ethical approval- The study was conducted using data from the medical records of the in-patient department after approval from the Institutional Ethics Committee, NSCB Medical College, Jabalpur.

RESULTS

Among 400 patients studied, 98 patients (24.5%) were found to develop septic shock. Of these, 14% were microbiologically confirmed cases. Table 1 shows the mean age of the study participants according to gender.It shows male preponderance (84%) was noted, with the older age group (>70 years) being more vulnerable to septic shock. No significant association was found between the incidence of septic shock and co morbidities such as diabetes mellitus or hypertension.

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Table 1: Mean age of the cases according to gender

	sex			
	Female		Male	
	Mean	Standard Deviation	Mean	Standard Deviation
Age	51.7	17.5	50.9	17.8

Table 2 shows the association between age, gender, and the occurrence of septic shock among the study population. As we can see in this table, there is male predominance seen in the study with 226 males and 75 females, mean age was almost same in both the sex's 51.7in females and 50.9 in males. Tuberculosis-associated septic shock was seen more in age group between 21-60 years in our study. Sixteen patients developed ARDS of which 9 patients had a negative microbiological positivity. A mortality of 5% (n= 20) was found overall among the study population. No significant association was found between age group and the occurrence of septic shock (p=0.553). However, a significant association was observed between sex and septic shock (p=0.048), with males more frequently affected.

Table 2: Association between age, gender and septic shock

		septic shock				p-value
		Absent		Present		
		Count	N %	Count	N %	
Age groups	<=20 years	8	2.7	6	6.1	0.553
	21 - 30 years	49	16.3	18	18.4	
	31 - 40 years	29	9.6	11	11.2	
	41 - 50 years	52	17.3	17	17.3	
	51 - 60 years	46	15.3	17	17.3	
	61 - 70 years	44	14.6	10	10.2	
	>70 years	73	24.3	19	19.4	
sex	Female	75	24.9	15	15.3	0.048
	Male	226	75.1	83	84.7	

Table 3 shows the association between comorbidities and the occurrence of septic shock among the study population.Among the 400 patients, the majority (n=353, 88.3%) had no comorbidities, while 25 (6.3%) had hypertension and 21 (5.3%) had diabetes mellitus. No significant association was found between the presence of comorbidities and the occurrence of septic shock (p=0.793); the proportion of patients without comorbidities was similar between those with (88.8%) and without (88.4%) septic shock.

Table 3: Association between comorbidities and septic shock

		septic shock				p-value
		Absent		Present		
		Count	N (%)	Count	N (%)	
Comorbidities	NIL	266	88.4	87	88.8	0.793
	HTN	20	6.6	5	5.1	
	DM	15	5.0	6	6.1	

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Table 4 shows the association between study variables and septic shock. Serum lactate showed a highly significant association with septic shock (p<0.001): 64.3% of patients with septic shock had a lactate level of 1–2, and 32.7% had a level >2, whereas the majority of patients without septic shock (51.8%) had a lactate level below 1, and none had a level above 2. No significant association was found between septic shock and serum procalcitonin (p=0.917) or serum cortisol (p=0.063), although the cortisol association approached the conventional threshold for significance. Outcome (death vs. discharge) also showed no significant association with septic shock status (p=0.266); although mortality was numerically higher among patients with septic shock (7.1%) than without (4.3%), this difference did not reach statistical significance.

Table 4: Association between laboratory parameters, outcome, and septic shock

		septic shock				p-value
		Absent		Present		
		Count	N (%)	Count	N (%)	
Serum lactate	0	67	22.3	0	0.0	<0.001
	<1	156	51.8	3	3.1	
	1-2	78	25.9	63	64.3	
	>2	0	0.0	32	32.7	
Serum procalcitonin	Negative	201	66.8	66	67.3	0.917
	Positive	100	33.2	32	32.7	
Serum cortisol (mcg/dl)	<=15	115	38.2	38	38.8	0.063
	16 – 25	130	43.2	32	32.7	
	>25	56	18.6	28	28.6	
Outcome	Death	13	4.3	7	7.1	0.266
	Discharge	288	95.7	91	92.9	

DISCUSSION

With a global burden of 10 million new cases per year [9]. TB remains a major health problem in developing countries such as India (2.69 million cases).[10] Most patients present with constitutional symptoms in the initial stage of the disease, with clinical features such as fever of prolonged duration and chest radiograph findings suggestive of tuberculosis; latent TB, as diagnosed by a positive skin tuberculin test [11] is highly suspicious for active TB infection.

Akin to these clinical parameters, a predictive model has been proposed for the diagnosis of TB in hospitalised patients.[12] The major indications for Intensive Care Unit (ICU) admission include respiratory failure due to tuberculous pneumonia, miliary TB, or acute respiratory distress syndrome (ARDS), sepsis, and neurological deterioration due to central nervous system tuberculosis.[13-15] Factors increasing the odds of death in TB patients include advancing age, female gender, HIV co-infection or unknown HIV status, a negative or missing pre-treatment sputum smear result, and retreatment status; appropriate targeting of care to these high-risk groups may be considered.[6]

Based on available literature, the mean total leucocyte count (TLC) in tubercular septic shock usually remains within the normal range and is significantly lower than in bacterial sepsis.[6] Our study showed a mean TLC of 14,000, associated with a greater incidence of mortality. Shah *et al.* [16] found anaemia with a microcytic, hypochromic pattern common in pulmonary TB, along with decreased PCV, MCV, MCH and MCHC, and raised WBC count, neutrophil count, platelet count and ESR.[16] Other reports found serum procalcitonin (normal <0.04 ng/mL) useful in distinguishing pneumonia aetiology, with higher levels in community-acquired pneumonia than tuberculosis,[17] as also seen in this study. Uddin *et al.* [18] found PCT levels fell significantly after six months of ATT, irrespective of age, sex, BMI and other laboratory values, suggesting a link between PCT reduction and successful treatment. A similar correlation was seen by Malik *et al.* [19] where TLC remained normal or low, as in our study, but serum PCT was significantly raised in

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community-acquired pneumonia. These findings [20,21] together with negative sputum examination, made early diagnosis of tubercular sepsis difficult and delayed ATT initiation. As tubercular septic shock carries higher morbidity and mortality than other bacterial septic shock, it should be suspected early, even without a sputum-positive status.

As *M. tuberculosis* culture reports take time, early diagnosis must rely on biomarkers and negative blood and urine culture reports. Biomarkers may allow early, largely supportive, intervention that can reduce the mortality. Although lactate remains the most common biomarker for sepsis, others such as pro-inflammatory cytokines, CRP, procalcitonin, and markers of neutrophil and monocyte activation may add value. [22] Gastric aspirate analysis may also help where sputum examination is inconclusive. Kethireddy *et al.* highlighted the role of early ATT in tubercular septic shock in reducing mortality [7] the persistence of septic shock and delayed ATT (after 24 hours) were significant predictors of mortality.[7]

Elevated CRP correlated positively with mortality in our study,[23] though CRP is non-specific. Similarly, Kwas *et al.* found CRP useful in assessing disease activity, severity and course.[24] Kethireddy *et al.*[7] also found serum lactate significantly raised in TB shock syndrome, and early antimicrobial and anti-tubercular therapy reducing mortality, similar to our study. Lam and Lo found adrenal involvement in 6% of active tuberculosis cases, with only one-fourth of these cases having the adrenal gland as the sole site of infection.[25]

Costanzo *et al.* found adrenal insufficiency to be an important consideration in disseminated TB; serum cortisol, ACTH stimulation testing, and abdominal CT to evaluate for haemorrhage, inflammatory infiltration, or adrenal enlargement may aid diagnosis.[26] Neogi *et al.* found the prevalence of overt and subclinical adrenal dysfunction in pulmonary tuberculosis to be high, correlating with disease severity and duration.[27] As found by Chaudhry and Tyagi [23]. ARDS is a common reason for ICU admission in tuberculosis, as also seen in our study.

ARDS is particularly seen in patients with miliary or disseminated tuberculosis, in immunocompromised patients, those with a shorter duration of illness, lymphopenia, and elevated serum alanine aminotransferase. An efficient testing protocol paired with high clinical suspicion is needed for a specific diagnosis, as Mtb culture takes time. Typical chest radiograph findings raise clinical suspicion but lack specificity.[28] and can be falsely interpreted as community-acquired pneumonia (CAP).[29]

Patients are frequently managed with CAP-targeted antimicrobial agents prior to a diagnosis of tuberculosis.[30] Sputum microscopy with acid-fast staining, the initial diagnostic step, is prompt and simple, but lacks sensitivity. As per WHO recommendations, diagnosis of pulmonary tuberculosis requires demonstration of bacteria in sputum; culture remains the gold standard but is time consuming, taking two to six weeks. Nucleic acid amplification testing (NAAT), either TruNAAT or CBNAAT, on sputum provides a swift and reliable microbiological diagnosis; sensitivities differ between sputum-positive and sputum-negative patients, and CBNAAT can be offered as the first-line investigation, particularly with a longer duration of illness or prior treatment default.

CONCLUSIONS

Septic shock is an important, though relatively uncommon, complication of tuberculosis, occurring in about a quarter of the patients in this study and carrying a high risk of mortality. A high index of clinical suspicion for septic shock should be maintained in tuberculosis patients, particularly in older male patients, and biochemical markers such as serum lactate can aid early recognition. Early institution of anti-tubercular therapy, along with steroid therapy where indicated and appropriate use of laboratory markers, may help ensure timely management and reduce mortality in these patients. Future multicentric studies with larger cohorts are needed to validate the role of biomarkers such as procalcitonin and cortisol in predicting septic shock in tuberculosis and to develop a simple bedside prediction score. Strengthening awareness among clinicians managing tuberculosis at peripheral health centres about this complication could facilitate earlier referral and supportive care, thereby improving patient outcomes.

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