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The Role of Total Body Fluid in Pulmonary Tuberculosis Patients with Risk of Obstructive Sleep Apnea

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Abstract:

Background: Pulmonary Tuberculosis (TB) patients are vulnerable to sleep-disordered breathing, particularly Obstructive Sleep Apnea (OSA), due to chronic pulmonary inflammation, structural lung damage, and systemic metabolic disturbances. Alteration of total body fluid, especially extracellular fluid accumulation, may contribute to upper airway collapsibility through rostral fluid shift during sleep. However, evidence regarding the relationship between total body fluid and OSA risk in TB patients remains limited.

Aim: An analytic observational study with a cross-sectional design was conducted on 85 TB patients selected using total sampling. OSA risk was assessed using the STOP-BANG questionnaire and categorized as mild, moderate, and severe. Total body water was measured using Bioelectrical Impedance Analysis (BIA) and classified as low, normal, or high. Data were analyzed using the Chi-Square test with a significance level of $p < 0.05$.

Results: The prevalence of OSA risk was high, with 34.1% mild, 32.9% moderate, and 32.9% severe. Most respondents had normal body fluid (68.2%), while 23.5% had high fluid levels. A significant association was found between OSA risk and total body fluid ($p = 0.000$). Patients with severe OSA showed a markedly higher proportion of fluid overload (46.6%) compared to those with mild OSA (10.3%).

Conclusion: There is a significant relationship between OSA risk and total body fluid in TB patients. Higher OSA severity is associated with increased likelihood of fluid overload. Assessment of hydration status should be included in TB management to reduce the risk of respiratory complications and sleep disturbances.

Keywords: Pulmonary Tuberculosis, obstructive sleep apnea, total body fluid, bioelectrical impedance analysis, STOP-BANG.

INTRODUCTION

Obstructive Sleep Apnea (OSA) is a sleep-related breathing disorder characterized by recurrent episodes of partial (hypopnea) or complete (apnea) upper airway obstruction during sleep, resulting in airflow reduction or cessation for at least 10 seconds despite ongoing respiratory effort. These events are commonly accompanied by oxygen desaturation and arousals, leading to fragmented and non-restorative sleep. Clinically, OSA presents with loud snoring, witnessed apneas, nocturnal choking, and excessive daytime sleepiness. The severity of OSA is determined by the Apnea–Hypopnea Index (AHI), with ≥5 events per hour considered diagnostic. OSA is a major public health concern due to its association with cardiovascular disease, metabolic dysfunction, neurocognitive impairment, and reduced quality of life.

The pathophysiology of OSA is multifactorial, involving anatomical, neuromuscular, and systemic factors. Structural abnormalities such as increased neck circumference, macroglossia, retrognathia, and pharyngeal narrowing predispose to airway collapse. Obesity is the most significant modifiable risk factor, while male sex, aging, alcohol consumption, smoking, and sedative use further increase airway collapsibility. In addition to structural factors, emerging evidence highlights the role of body fluid redistribution in OSA pathogenesis. During the supine position, fluid accumulated in the lower extremities during the day may shift rostrally toward the neck, increasing peripharyngeal tissue volume and narrowing the upper airway. This mechanism is particularly relevant in individuals with fluid imbalance or chronic systemic disease.

Total body fluid constitutes approximately 50–70% of adult body weight. Approximately 95% of the human body's fluid is water, and two-thirds of it is intracellular fluid. Fluid balance plays a crucial role in oxygen transport, metabolic regulation, and maintenance of tissue homeostasis. Alterations in hydration status can influence respiratory function and upper airway stability. Bioelectrical Impedance Analysis (BIA) is a practical, non-invasive method widely used to estimate total body water and body composition in clinical settings.

Pulmonary tuberculosis (TB), caused by Mycobacterium tuberculosis, remains a major global health problem, particularly in Southeast Asia, including Indonesia. Globally, there are 8.2 million reported cases of TB, 84% of which are pulmonary TB and 16% are extrapulmonary TB. The highest number of TB cases occurred in Southeast Asia (45%), followed by Africa (24%), and the Western Pacific region (17%). The prevalence of TB cases

was highest in India (27%), Indonesia (10%), China (7.1%), the Philippines (7.0%), Pakistan (5.7%), Nigeria (4.5%), Bangladesh (3.6%), and the Democratic Republic of Congo (3.0%). In Indonesia, 821,200 cases of pulmonary TB were reported, with West Java ranking first, followed by South Papua, Central Papua, Central Java, Banten, and others. South Sulawesi ranked 28th nationally. Pulmonary TB leads to chronic inflammation, parenchymal destruction, fibrosis, and impaired respiratory mechanics. Patients frequently experience weight loss, malnutrition, reduced physical activity, and systemic inflammatory responses, all of which may disrupt fluid balance and sleep quality.

Several mechanisms suggest a potential interaction between pulmonary TB and OSA. Chronic airway inflammation and structural lung changes may alter airway stability during sleep. Additionally, disturbances in fluid regulation due to malnutrition, systemic inflammation, and long-term pharmacotherapy may promote fluid retention and nocturnal rostral fluid shift, thereby exacerbating upper airway obstruction. Conversely, untreated OSA may worsen hypoxemia and systemic inflammation, potentially affecting TB outcomes.

Although previous studies have demonstrated an association between fluid redistribution and OSA severity, as well as the impact of body fluid status on TB outcomes, no study has specifically examined the relationship between OSA risk and total body fluid in patients with pulmonary tuberculosis. Considering the high burden of TB and the clinical consequences of OSA, investigating this relationship is essential to improve comprehensive and multidisciplinary management.

Therefore, this study aims to analyze the characteristics of total body fluid in relation to OSA risk among patients with pulmonary tuberculosis treated at YW-UMI Ibnu Sina Hospital in Makassar, to address an existing research gap and contribute to integrated TB care strategies.

Recent scientific developments show increasing attention to the relationship between sleep-disordered breathing and chronic respiratory diseases, particularly Obstructive Sleep Apnea (OSA). OSA is widely recognized as a disorder characterized by repeated upper airway obstruction during sleep, leading to hypoxia, fragmented sleep, and multiple systemic complications such as cardiovascular disease and metabolic disorders. At the same time, pulmonary tuberculosis (TB) remains a major global health burden, especially in Southeast Asia and Indonesia. TB causes chronic inflammation, lung tissue damage, fibrosis, and impaired respiratory mechanics, all of which can affect breathing patterns and

sleep quality. Patients with TB frequently experience systemic metabolic disturbances, malnutrition, and reduced physical activity, factors that may alter body fluid balance and contribute to respiratory complications. Several studies have separately investigated:

1. The association between fluid redistribution and OSA severity, and
2. The impact of TB on body composition and metabolic status.

However, most previous research has focused on general populations or patients with cardiovascular and renal diseases, rather than individuals with infectious respiratory diseases such as pulmonary TB. Therefore, current scientific evidence suggests that both fluid imbalance and structural lung disease may contribute to OSA, but the interaction between these factors in TB patients has not been widely explored.

METHOD

Study Design

This study used a quantitative analytical approach with a cross-sectional design to examine the relationship between the risk of Obstructive Sleep Apnea (OSA) and Total Body Fluid in patients with pulmonary tuberculosis at YW-UMI Ibnu Sina Hospital, Makassar. The design enabled simultaneous measurement of OSA risk and total body fluid at a single point in time without intervention.

Population and Sample

The population of this study comprised all patients with pulmonary tuberculosis receiving treatment at YW-UMI Ibnu Sina Hospital, with an estimated total of 560 patients. A purposive sampling technique was used, with predetermined inclusion and exclusion criteria. The minimum sample size was calculated using the Slovin formula with a margin of error of 10%, resulting in 85 respondents. Data collection was conducted until the required sample size was achieved.

Inclusion and Exclusion Criteria

Inclusion criteria included patients diagnosed with pulmonary tuberculosis who were undergoing inpatient or outpatient treatment at YW-UMI Ibnu Sina Hospital, were able to communicate properly, and were willing to participate by providing informed consent after receiving an explanation of the study's objectives, benefits, and risks. Exclusion criteria were patients with medical conditions that prevented them from standing or maintaining body stability, patients with metal implants or pins in the body that could

interfere with Bioelectrical Impedance Analysis (BIA) measurements, and patients who refused to follow the entire research procedure.

Operational Definition of Variables

The independent variable in this study was the risk of Obstructive Sleep Apnea (OSA) measured using the STOP-BANG questionnaire and categorized into low risk (score 0–2), moderate risk (score 3–4), and high risk (score 5–8) on an ordinal scale. The dependent variable was Total Body Fluid, measured using Bioelectrical Impedance Analysis (BIA) on the Onemed 825 Body Fat Scan with dual-frequency technology. The results were classified as: in men, low/dehydration (<50%), normal (50–65%), and overhydration (>65%); in women, low/dehydration (<45%), normal (45–60%), and overhydration (>60%), using an ordinal scale. Intervening variables included age, sex, Body Mass Index (BMI), smoking history, and diabetes mellitus status obtained from medical records and structured interviews.

Research Instruments and Data Collection Procedures

OSA risk data were obtained using the STOP-BANG questionnaire through direct interviews with respondents. Total body fluid was measured using the BIA device according to standard operating procedures. Additional data, such as age, sex, medical history, smoking status, and diabetes mellitus, were collected from patient medical records and structured data collection forms. After obtaining informed consent, respondents completed the STOP-BANG questionnaire followed by a BIA examination conducted by trained researchers. All collected data were reviewed to ensure completeness and accuracy before analysis.

Data Analysis

Data were analyzed using SPSS software. Univariate analysis was performed to describe the characteristics of respondents, OSA risk level, and total body fluid, using frequencies and percentages. Bivariate analysis was conducted using the Chi-square test to assess the relationship between OSA risk and total body fluid, with a significance level of $p < 0.05$, while accounting for potential confounding variables.

RESULT AND DISCUSSION

This study was conducted to determine the relationship between the risk of Obstructive Sleep Apnea (OSA) and total body fluid in patients with tuberculosis. Among inpatients and outpatients during the 2025 period, with a total study population of 85 TB patients, the results are shown below

Frequency Distribution of Respondent Characteristics and Research Variables among Pulmonary TB Patients at YW-UMI Ibnu Sina Hospital, Makassar, 2025

Based on table 1. presents the combined characteristics of 85 pulmonary tuberculosis patients. The majority of respondents were male (56.5%), while females accounted for 43.5%. The largest age group was the senior category (>65 years) at 24.7%, followed by early elderly (46–55 years) at 22.4%.

Regarding smoking status, 43.5% were active smokers, 27.1% passive smokers, and 29.4% had no history of smoking. Most respondents did not have diabetes mellitus (83.5%), whereas 16.5% had a confirmed diagnosis of DM.

Based on OSA risk assessment, 34.1% were categorized as mild risk, 32.9% moderate, and 32.9% severe. In terms of nutritional status, 40.0% were underweight, 30.6% normal, and 29.4% overweight. Measurement of total body fluid showed that most respondents were within the normal range (68.2%), while 23.5% had high body fluid and 8.2% low body fluid.

Table 1. Characteristics of Respondents

Category	Frequency (n)	Percentage (%)
Gender		
Male	48	56.5
Female	37	43.5
Age		
Late Adolescence (17–25)	11	12.9
Early Adulthood (26–35)	5	5.9
Late Adulthood (36–45)	15	17.6
Early Elderly (46–55)	19	22.4
Late Elderly (56–65)	14	16.5
Senior (>65)	21	24.7
Smoking Status		
Active Smoker	37	43.5
Passive Smoker	23	27.1
Non-Smoker	25	29.4
Diabetes Mellitus Status		
Yes	14	16.5
No	71	83.5
OSA Risk		
Mild	29	34.1
Moderate	28	32.9

Severe	28	32.9
BMI		
Underweight	34	40.0
Normal	26	30.6
Overweight	25	29.4
Total Body Fluid		
Low	7	8.2
Normal	58	68.2
High	20	23.5
Total	**85**	**100.0**

Source*: *Primary Data

Descriptive Statistics of OSA, BMI, and Total Body Fluid

Based on Table 2, the mean OSA score was 3.35 indicating moderate risk. Mean BMI was 19.31 reflecting predominantly normal–low nutritional status. Mean total body fluid was 57.10 with moderate variability.

Table 2. Descriptive Statistics of OSA, BMI, and Total Body Fluid

Variable	n	Min	Max	Mean	Median	SD
OSA Score	85	0.00	7.00	3.35	4.00	1.84
BMI	85	13.10	28.90	19.31	19.20	3.62
Total Body Fluid	85	39.10	76.90	57.10	56.80	7.50

Source: Primary Data

Relationship between OSA and Total Body Fluid

In the mild OSA group, 86.2% had normal body fluid, whereas in the severe OSA group, 46.6% had high body fluid and only 35.7% were normal. Overall, most cases of high body fluid were found in moderate–severe OSA groups. The Chi-Square test showed $p = 0.000$ ($p < 0.05$), indicating a significant relationship between OSA risk and total body fluid in pulmonary TB patients. Thus, higher OSA risk is associated with greater likelihood of increased body fluid.

Table 3. Relationship between OSA Risk and Total Body Fluid

OSA	Low	Normal	High	Total	Sig.
Mild	1 (3.4%)	25 (86.2%)	3 (10.3%)	29 (100%)	**0.000**
Moderate	1 (3.6%)	23 (82.1%)	4 (14.3%)	28 (100%)	
Severe	5 (17.9%)	10 (35.7%)	13 (46.6%)	28 (100%)	
Total	7 (8.2%)	58 (68.2%)	20 (23.5%)	85 (100%)	

Chi-Square test, p < 0.05
Source: Primary Data, 2025

DISCUSSION

Prevalence of Obstructive Sleep Apnea (OSA) Risk among Pulmonary Tuberculosis Patients

The results of this study showed that among 85 patients with pulmonary tuberculosis, the distribution of Obstructive Sleep Apnea (OSA) risk was nearly evenly divided. A total of 29 respondents (34.1%) were categorized as having mild OSA risk, 28 respondents (32.9%) as moderate risk, and 28 respondents (32.9%) as severe risk. This proportion indicates that two-thirds of respondents (65.8%) experienced moderate to severe OSA risk, reflecting a high burden of sleep-related breathing disorders in the pulmonary TB population. The prevalence of moderate–severe OSA among respondents, at over half, is considerably higher than in the general population, where the prevalence of severe OSA is usually only around 3–7%. Therefore, this finding indicates that pulmonary TB patients constitute a high-risk group for sleep-related breathing disorders, particularly OSA.

Physiologically, OSA occurs due to repetitive collapse of the upper airway during sleep, characterized by partial obstruction (hypopnea) or complete obstruction (apnea) lasting ≥10 seconds. This collapse is triggered by decreased tone of the pharyngeal dilator muscles, such as the genioglossus, tensor veli palatini, and geniohyoid muscles, which allows posterior displacement of the tongue and palate and narrowing of the pharyngeal lumen. This condition leads to intermittent hypoxia, sleep fragmentation, increased respiratory effort, and excessive daytime sleepiness. The severity of OSA is classified based on the Apnea–Hypopnea Index (AHI): <5 is considered normal, 5–20 mild, 20–40 moderate, and >40 severe OSA.

The high prevalence of OSA risk in pulmonary TB patients in this study has a strong pathophysiological basis. TB patients commonly experience structural and functional lung abnormalities, including pleural thickening, fibrosis, atelectasis, bronchiectasis, cavitation, and parenchymal deformity, all of which can increase airway resistance and alter breathing patterns during sleep. These abnormalities may lead to chronic hypoventilation, increased work of breathing, and instability of respiratory control, thereby facilitating upper airway obstruction at night.

These findings are consistent with those of Kim et al. (2024), who reported that the prevalence of OSA risk was higher among individuals with airflow limitation (AFL) than in the general population. The presence of AFL reflects structural or functional impairment of the respiratory system, directly increasing the risk of OSA. The study also noted that factors

such as obesity, hypertension, and a history of lung disease increase STOP-BANG scores, thereby elevating OSA risk in populations with respiratory disorders.

Furthermore, a narrative review by Layek (2025) emphasized that pulmonary TB patients experience poorer sleep quality than the general population, with sleep disturbances including snoring, sleep-disordered breathing, and OSA. In some cases, TB involvement of the nasopharynx can cause upper airway narrowing, triggering snoring and obstructive sleep apnea during sleep. Devassy (2022) also demonstrated that TB patients have a higher prevalence of sleep disturbances due to respiratory symptoms, chronic inflammation, and side effects of anti-TB therapy that affect sleep patterns.

Non-structural risk factors such as age, sex, smoking, alcohol consumption, and comorbidities (hypertension, diabetes, metabolic syndrome) further aggravate OSA risk in the TB population, considering that TB patients often present with one or more of these risk factors. Weight loss due to TB does not necessarily protect against OSA, as structural factors, such as reduced pharyngeal muscle tone caused by malnutrition, may increase pharyngeal collapsibility.

Thus, the high prevalence of OSA risk in this study reflects a complex interaction between structural lung changes due to TB, anatomical risk factors, chronic respiratory impairment, and instability of respiratory control. These findings are in line with theoretical and empirical evidence that pulmonary TB patients are highly vulnerable to OSA.

Total Body Fluid Profile among Pulmonary Tuberculosis Patients

The results showed that of 85 respondents, 68.2% had normal total body fluid, 23.5% had high body fluid, and 8.2% had low body fluid. The relatively high proportion of patients with high body fluid indicates the presence of overhydration (fluid overload) in a subset of patients with pulmonary TB.

Physiologically, body fluid consists of two major compartments: intracellular fluid (ICW, approximately 40% of body weight) and extracellular fluid (ECW, approximately 20%), which includes plasma and interstitial fluid. The balance between these compartments is essential to maintain cellular function, electrolyte transport, and hemodynamic stability. Fluid imbalance, particularly increased ECW, can cause tissue edema, impaired oxygen diffusion, and increased airway resistance, which are highly relevant in pulmonary disease.

Body fluid measurements in this study used Bioelectrical Impedance Analysis (BIA), a technology that objectively measures TBW, ECW, ICW, fat-free mass, and hydration status. BIA relies on the electrical impedance of body tissues and can detect subtle changes in total body water, including fluid overload or dehydration. This method is particularly suitable for pulmonary TB populations known to experience metabolic and inflammatory alterations.

These findings are consistent with those of Sutrisno et al. (2024), who reported significant changes in body composition during the intensive phase of TB treatment, including increased fat mass and visceral fat, as well as an imbalance in fat-free mass that may affect ICW and ECW. This indicates that body composition in TB patients is highly dynamic and influenced by chronic inflammation, anti-TB therapy, malnutrition, and metabolic changes.

In patients with pulmonary TB, chronic inflammation induces capillary leakage, increased endothelial permeability, and fluid accumulation in the interstitium, leading to increased ECW. Fibrosis, cavitation, bronchiectasis, and permanent lung damage may also disrupt fluid regulation and increase body fluid burden. Moreover, malnutrition commonly found in TB reduces muscle mass, decreases ICW storage capacity, and increases the relative proportion of ECW to TBW.

The increase in body fluid in some TB patients can be explained by several interconnected mechanisms: chronic inflammation promoting extracellular accumulation; reduced ICW due to muscle wasting; pulmonary dysfunction with hypoxia and pulmonary vasoconstriction favoring fluid retention; effects of anti-TB drugs such as isoniazid and rifampicin on electrolyte metabolism; and decreased physical activity that worsens peripheral fluid retention. The combination of these factors results in increased body fluid in a proportion of TB patients.

Therefore, the increased proportion of TB patients with high body fluid in this study is consistent with the physiological and pathological characteristics of TB. Previous studies have also shown that fluid imbalance is common in chronic diseases involving inflammation and lung tissue damage.

These findings highlight the importance of considering body fluid status in TB management, as fluid imbalance may affect lung function, exercise tolerance, treatment effectiveness, and the risk of complications, including sleep disorders such as OSA.

Relationship between OSA Risk and Total Body Fluid in Pulmonary Tuberculosis Patients

Cross-tabulation and Chi-Square analysis demonstrated a significant relationship between OSA risk and total body fluid in patients with pulmonary TB ($p = 0.000$). Data distribution showed clear differences across OSA categories. In the mild OSA group, most patients (86.2%) had normal body fluid, and only 10.3% had high body fluid. In the moderate group, 82.1% were normal, and 14.3% had high body fluid. However, in the severe OSA group, the proportion with high body fluid increased sharply to 46.6%, far higher than in the mild or moderate groups.

This indicates that the higher the OSA risk, the greater the likelihood of body fluid imbalance, particularly an increase in body fluid. Conversely, patients with mild OSA risk tended to have normal fluid composition. These findings confirm a clinical link between sleep-disordered breathing and body fluid regulation in patients with pulmonary TB.

Physiologically, the association between OSA and total body fluid is explained by the rostral fluid shift mechanism, namely the movement of fluid from the lower extremities to the neck during supine sleep. During daytime activity, gravity promotes fluid accumulation in the legs; when lying down, fluid shifts rostrally to peripharyngeal tissues, causing tissue edema, increased external pressure on the upper airway, pharyngeal narrowing, and greater airway collapsibility. Thus, patients with higher body fluid volume—especially increased ECW—are more likely to develop moderate-to-severe OSA.

These results are consistent with those of Chuang et al. (2025), who showed that hydration status and total body water are directly related to OSA severity; increased TBW, particularly ECW, was associated with higher AHI values. Zhang et al. (2024) also reported that high-risk OSA patients more frequently experienced fluid overload, and the combination of OSA risk and overhydration was associated with worse clinical outcomes. These studies support the notion that overhydration is not merely a comorbidity but a pathophysiological factor that aggravates OSA.

In pulmonary TB, fluid imbalance becomes more relevant. Chronic inflammation increases capillary permeability and interstitial retention; structural lung changes disrupt lymphatic drainage, thereby worsening fluid accumulation. Malnutrition decreases ICW and increases the ECW/ICW ratio—an indicator of fluid imbalance. Even without marked elevation of TBW, altered fluid distribution creates conditions that favor rostral fluid shift during sleep and increase OSA risk.

Therefore, these findings have a strong physiological basis: TB causes fluid retention and changes in body composition; excess extracellular fluid increases peripharyngeal tissue pressure; and nocturnal fluid shifts narrow the airway and aggravate OSA. These mechanisms act synergistically, explaining why severe OSA was predominantly found in patients with high body fluid.

This relationship is consistent with the theory that increased ECW enhances upper airway collapsibility. Patients with high body fluid experience nocturnal pharyngeal tissue thickening that disrupts airway patency. The findings align with Chuang et al. (2025) and Zhang et al. (2024), reinforcing that hydration status and total body fluid are key determinants of OSA severity. Thus, this study strengthens the evidence that overhydration is an important determinant of OSA pathogenesis, particularly in populations with chronic respiratory comorbidities, such as pulmonary tuberculosis.

The novelty of this research lies in several aspects:

1. Integration of Three Clinical Variables. This study simultaneously examines the relationships among pulmonary tuberculosis, obstructive sleep apnea risk, and total body fluid status.
2. Application in a Specific Clinical Population (TB Patients). Previous studies mostly evaluated OSA and body fluid status in cardiovascular, renal, or obese populations, whereas this study focuses specifically on patients with pulmonary tuberculosis.
3. Use of Bioelectrical Impedance Analysis (BIA) for Fluid Assessment
   The study uses BIA to objectively measure total body fluid, allowing a non-invasive evaluation of hydration status in TB patients.
4. Clinical Evidence from the Indonesian TB Population. The study provides empirical data from TB patients in Indonesia, a country with one of the highest TB burdens globally.
5. Identification of Fluid Overload as a Potential Risk Factor for OSA in TB
   The findings demonstrate that higher OSA severity is associated with increased total body fluid, suggesting that hydration status may play an important role in the pathophysiology of sleep-disordered breathing among TB patients.

Despite the growing literature on OSA and fluid redistribution, several research gaps remain:

1. Limited Evidence Linking TB and OSA Pathophysiology
   Most studies address OSA in the context of obesity, cardiovascular disease, or chronic

kidney disease, while the relationship between pulmonary tuberculosis and OSA risk remains poorly understood.

2. Lack of Research on Body Fluid Status in TB-Related Sleep Disorders Previous studies on TB primarily focus on infection control, treatment outcomes, and nutritional status, with little attention to body fluid composition and its respiratory implications.
3. Insufficient Investigation of Fluid Redistribution Mechanisms in TB Patients Although rostral fluid shift is recognized in OSA pathogenesis, its role in TB patients with chronic lung inflammation and structural damage has not been widely examined.
4. Limited Clinical Screening Strategies for OSA in TB Management Routine TB management rarely includes OSA screening or monitoring of hydration status, even though both may influence respiratory outcomes.

Although previous studies have demonstrated associations between body fluid redistribution and obstructive sleep apnea, as well as metabolic and physiological changes in patients with pulmonary tuberculosis, the relationship between total body fluid status and OSA risk in these individuals remains insufficiently explored. Evidence on integrating these variables within a TB population remains limited, particularly in high-burden settings such as Indonesia.

CONCLUSION

This study confirms a significant relationship between obstructive sleep apnea (OSA) risk and total body fluid status among patients with pulmonary tuberculosis. The majority of respondents exhibited moderate to severe OSA risk, indicating a substantial burden of sleep-disordered breathing in this population. Most patients had normal total body fluid; however, a considerable proportion demonstrated overhydration, particularly among those with severe OSA. Increased total body fluid, especially in the extracellular compartment, was associated with higher OSA risk, supporting the role of fluid imbalance in the pathophysiology of airway obstruction during sleep. These findings emphasize the importance of assessing hydration status and OSA risk in comprehensive tuberculosis management. Integrating routine OSA screening and body fluid monitoring into TB care is recommended to improve respiratory outcomes and overall patient quality of life.

Implication

The findings of this study carry important clinical and public health implications for the management of pulmonary tuberculosis. The demonstrated association between OSA risk and total body fluid suggests that sleep-disordered breathing may represent an underrecognized comorbidity in TB patients, potentially contributing to poorer respiratory function, reduced treatment tolerance, and diminished quality of life. Routine screening for OSA using simple validated tools, combined with periodic assessment of body fluid status through Bioelectrical Impedance Analysis, could enable earlier identification of high-risk patients. These results also provide a foundation for future longitudinal and interventional studies aimed at evaluating whether correction of fluid imbalance and targeted OSA management can enhance clinical outcomes and accelerate recovery in patients with pulmonary tuberculosis

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