

Change in lean body mass in pulmonary tuberculosis patients during treatment in a tertiary care hospital, Puducherry

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Abstract

Background

As weight gain during tuberculosis treatment does not differentiate between fat and lean mass changes, assessment of lean body mass (LBM) becomes necessary.

Methodology:

We followed-up 48 microbiologically confirmed newly diagnosed pulmonary tuberculosis patients ($BMI < 21 \text{ kg/m}^2$). LBM was measured at the end of intensive phase (IP) by Bio-electrical Impedance Analysis (BIA) only and at the end of treatment by Dual-energy X-ray Absorptiometry (DXA) and BIA. Longitudinal changes in LBM were analysed using repeated measures ANOVA (RMANOVA).

Results

Mean $\pm$ SD ages of 39 males and nine females were 47.2 ± 13.4 years and 45.2 ± 12.1 years respectively. Mean LBM measured by BIA increased from 35.6 ± 6.9 kg at baseline to 36.8 ± 8.0 kg at end of IP and to 37.6 ± 8.6 kg at end of treatment, with a statistically significant overall change ($p = 0.0004$). DXA measurements similarly showed a significant rise in LBM from 33.6 ± 5.7 kg to 35.9 ± 6.4 kg over the course of treatment. Change in LBM contributed 58.5% in change in weight.

Conclusion

Only about half of the increase in weight was contributed by change in lean body mass, which was higher among males.

INTRODUCTION

In 2024, globally, 10.8 million people were newly diagnosed and reported to have tuberculosis (TB), of which 81% were pulmonary TB and 19% extrapulmonary TB. India alone accounts for about 25% of global TB incidence, with higher rates in adults and males. Malnutrition and tuberculosis share bidirectional relationship. Undernutrition is a significant risk factor for TB susceptibility and progression from latent to active TB, accounting for an estimated one million cases globally and 5.4 lakh TB episodes in India in 2025, while active TB itself often causes wasting and weight loss.¹ Prevalence of undernutrition among Indian TB patients is among the highest globally (above 50% in many cohorts), but average weight gains during treatment are comparable to those reported from other countries.² Studies showed malnutrition not only in terms of body weight or BMI, but also in terms of fat mass index (FMI)

and lean mass index (LMI) as measured by bio-electrical impedance analysis (BIA) is present at the time of TB diagnosis.³

Most TB patients experience substantial weight gain during TB treatment.⁴ But, measurement of BMI for assessment of nutritional status is not adequate as it primarily reflects overall weight change and cannot distinguish whether gains are due to fat or lean mass. As a result, patients may show improvement in BMI despite persistent deficits in LBM, leading to an incomplete picture of nutritional recovery. BIA has been recommended as a practical and reproducible method for clinical assessment of fat and LBM and has been commonly used in most studies assessing LBM of TB patients.⁵ However we know that the gold-standard for body composition analysis is dual-energy x-ray absorptiometry (DXA) which has been used in very few studies. More importantly, studies on change in body composition of TB patients on treatment are very few with most of the available literature among HIV positive African population. Most of Indian studies have described weight and BMI changes during TB treatment and body composition of TB patients only at presentation, through cross-sectional or retrospective study designs. This makes it important to longitudinally assess LBM changes during treatment in Indian TB patients, especially those having low BMI, by BIA and DXA to understand the quality of nutritional rehabilitation and its implications for long-term outcomes. The objective of this study is to describe the change in lean body mass from the initiation of tuberculosis treatment to the second and sixth months of anti-tuberculosis therapy among microbiologically confirmed newly diagnosed pulmonary tuberculosis patients having BMI<21kg/m².

METHODOLOGY

Study design and Study population

A longitudinal prospective study was done from January 2024 to June 2025 in a tertiary care hospital, Puducherry among more than 18 years newly diagnosed microbiologically confirmed pulmonary TB patients having BMI < 21kg/m². Focussing on newly diagnosed TB patients provides a clearer picture of treatment-related changes, avoiding confounding from prior TB episodes or earlier exposure to anti-TB therapy that may already have altered body composition. Newly diagnosed patients are also at highest risk of rapid weight and LBM loss, particularly those with BMI < 21 kg/m², who are more vulnerable to muscle wasting and poor treatment outcomes. The study focused on pulmonary TB patients because they represent the largest proportion of TB cases, contribute most to transmission, and typically present with greater systemic manifestations such as weight and LBM loss compared to many forms of extrapulmonary TB. Among Asian population, AFB smear positive pulmonary TB patients have greater extent of malnutrition as measured by BMI compared to AFB smear negative pulmonary TB patients at the time of diagnosis, correlating to African population having malnutrition as measured by BMI, FMI and LMI.⁶⁻⁸ The exclusion criteria were pregnancy, children, drug resistant TB, concurrent HIV positivity and known case of malignancy. HIV status of adult TB patients does not affect body composition as measured by BIA and DXA at baseline.^{6,9}

Sampling technique and Sample size

Sample size was calculated taking alpha and beta error of 5%, Cohen effect size (f) = 0.25 (medium effect size), considering single group and three repeated measures. Using ANOVA-Repeated measures, within factors method in G*Power version 3.1.9.2 the initial estimate was 43. The final estimate was rounded to 50 after considering 10% lost to follow-up. We consecutively enrolled all TB patients satisfying inclusion and exclusion criteria notified during our study duration.

Study procedure and data collection

Body weight and LBM were assessed at three time points: within five days of initiation of anti-tubercular therapy (ATT), at completion of the intensive phase (IP), and after treatment completion. LBM was measured using both BIA and DXA at baseline and treatment completion, while only BIA was used at the end of the intensive phase.

Dietary intake was assessed using a 24-hour dietary recall. Daily calorie and protein intake were estimated using the *Nutritive Value of Indian Foods* published by the National Institute of Nutrition. Calorie and protein adequacy were evaluated against recommended requirements for patients with tuberculosis.¹⁰

Information on comorbidities was based on self-reported data. History of alcohol consumption with its frequency and amount within last one year was taken. Tobacco consumption history was taken along with frequency per week and pack per day within last one year. Both the history was cross verified with the accompanying family member whenever possible. Baseline chest radiographs were graded for disease severity by help of a pulmonologist and categorized as mild, mild-to-moderate, moderate, moderate-to-severe, or severe.

Height was measured to the nearest centimetre using a stadiometer. Body composition was assessed using an Omron Karada Scan Body Composition Monitor (HBF-375) which uses BIA method by passing very weak (500 μ A) current flow not stimulant and very safe to human body. Participants were instructed to empty their bladder, remove metallic objects, and stand barefoot on the analyzer. The device was calibrated according to the manufacturer's recommendations. Lean body mass was calculated as total body weight minus fat mass.

Whole-body DXA scans were performed using a Hologic Discovery Wi scanner by trained personnel following standard protocols. Participants removed all metallic objects and were scanned in the supine position.

Follow-up assessments at the end of the intensive phase and treatment completion included measurement of weight, body mass index (BMI), and BIA-derived body composition. DXA assessment was repeated at treatment completion.

Height-adjusted body composition indices were calculated, including lean mass index (LMI) and fat mass index (FMI). Body wasting was defined as an LMI < 16.7 kg/m² in males and < 14.6 kg/m² in females.^{6,11–13} Limb lean mass was calculated as the sum of lean mass in the arms and legs, while trunk lean mass represented the remaining lean tissue excluding the head region.

Statistical analysis

To estimate the change in lean body mass from baseline to the end of IP and to the end of ATT for study participants, Repeated measures ANOVA (RMANOVA) was used as statistical test. Longitudinal changes in lean body mass were analysed using mixed-effects linear regression models with maximum likelihood (ML) estimation. Time of follow-up (baseline, end of IP and treatment completion) was included as a fixed effect, and a random intercept was specified for each participant to account for within-subject correlation and variability in baseline LBM. Covariates such as tobacco use, alcohol use, comorbidities, sputum smear grading, and chest X-ray severity were included in separate stratified models as fixed effects. Results were reported as mean difference [regression coefficients (β)] with 95% confidence intervals (CI) and p-values. The p-value of less than 0.05 was considered statistically significant. All the data analysis was carried out in STATA version 17 (Stata Corp, Texas, USA).

Ethics approval

The scientific approval for the conduct of the study was obtained from Postgraduate Research Monitoring Committee (PGRMC) and ethical approval for the study was obtained from Institutional Ethics Committee (IEC) for observational human studies, JIPMER, Puducherry (JIP/IEC-0S/18/2023). The study was conducted in accordance with the National Ethical Guidelines for Biomedical and Health Research Involving Human Participants issued by the Indian Council of Medical Research (ICMR). Administrative clearance was obtained from NTEP state operational research (OR) committee, Puducherry. Informed written consent was obtained from all participants prior to the study after explaining the purpose of the study in local language.

RESULTS

Among 55 participants recruited, five (9.1%) died during intensive phase and two (3.6%) during continuation phase of anti-tubercular therapy (ATT). Forty-eight participants (87.3%; 39 males and nine females) completed follow-up and were included in analysis. On average, ATT was initiated within seven days of tuberculosis diagnosis. Baseline characteristics of participants are presented in Table 1.

Table 1
Baseline characteristics of all the recruited (N = 55) and followed-up participants (N = 48)

Characteristics	Participants at baseline n (%)	Participants who completed follow-up n (%)
Age in years, Mean (SD)	48.5 ± 13.6	46.8 ± 13.1
Gender		
Male	44 (80.0)	39 (81.2)
Female	11 (20.0)	09 (18.8)
Comorbidity		
DM	13 (23.6)	12 (25.0)
DM + HTN	03 (05.5)	03 (06.3)
Liver disease	01 (01.8)	01 (02.1)
Kidney disease	06 (10.9)	04 (08.3)
None	32 (58.2)	28 (58.3)
Tobacco use		
Yes	11 (20.0)	08 (16.7)
No	44 (80.0)	40 (83.3)
Alcohol use		
Yes	30 (54.6)	25 (52.1)
No	25 (45.4)	23 (47.9)
Hepatitis co-infection		
Positive for HbsAg	02 (03.6)	01 (02.1)
Negative for HbsAg	53 (96.4)	47 (97.9)
Sputum smear grading (n = 18 and n = 15)		
Scanty+	03 (16.7)	03 (20.0)
1+	04 (22.2)	03 (20.0)
2+	06 (33.3)	06 (40.0)
3+	05 (27.8)	03 (20.0)
Bacterial load in Gene Xpert for MTB (n = 37 and n = 33)		

Characteristics	Participants at baseline n (%)	Participants who completed follow-up n (%)
Very low	04 (10.8)	04 (12.1)
Low	16 (43.2)	14 (42.4)
Medium	13 (35.2)	11 (33.4)
High	04 (10.8)	04 (12.1)
Disease severity in Chest X-ray		
Mild	12 (21.8)	12 (25.0)
Mild to moderate	15 (27.3)	14 (29.2)
Moderate	11 (20.0)	10 (20.8)
Moderate to severe	09 (16.4)	08 (16.7)
Severe	08 (14.5)	04 (08.3)
Diet		
Normal	01 (01.8)	01 (02.1)
Calorie deficit	04 (07.3)	04 (08.4)
Protein deficit	-	-
Both calorie and protein deficit	50 (90.9)	43 (89.5)

At baseline, 40 (83.3%) participants were wasted according to BMI criteria, while all males and eight of nine females were classified as wasted based on LMI.

Participants showed progressive improvements in weight, BMI, and LBM during TB treatment. Mean $\pm$ SD LBM measured by BIA increased from 35.6 ± 6.9 Kgs at baseline to 36.8 ± 8.0 Kgs at end of intensive phase and to 37.6 ± 8.6 Kgs at treatment completion ($p = 0.0004$, repeated-measures ANOVA). Similarly, DXA showed a significant increase in mean $\pm$ SD LBM from 33.6 ± 5.7 Kgs to 35.9 ± 6.4 Kgs. (Table 2, 3 and Fig. 1) The observed effect size was large (partial $\eta^2 = 0.173$; Cohen's $f = 0.457$) compared to medium which was taken for sample size calculation. Significant LBM gain was observed over the course of treatment by both BIA and DXA; however, BIA detected significant gains primarily during intensive phase.

Table 2

Mean anthropometric and biochemical parameters of participants during the course of tuberculosis treatment (N = 48)

Parameters	Baseline Mean ± SD	At the end of intensive phase of ATT n (%)	At the end of ATT n (%)
Weight (kg)	44.9 ± 6.6	47.0 ± 7.4	49.1 ± 8.3
Height (cm)	163.5 ± 09.2	163.5 ± 09.2	163.5 ± 9.2
Body mass index (Kg/m ²)	16.6 ± 2.1	17.5 ± 2.3	18.3 ± 2.9
Lean Mass Index (Kg/m ²)	12.5 ± 1.8	-	13.4 ± 2.0
Lean body mass by BIA (kg)	35.6 ± 6.9	36.8 ± 8.0	37.6 ± 8.6
Lean body mass by DXA (kg)	33.5 ± 5.7	-	35.9 ± 6.4
Lean body mass by DXA: Trunk (Kg)	16.8 ± 3.4	-	18.1 ± 2.9
Lean body mass by DXA: Limb (Kg)	14.4 ± 2.8	-	15.7 ± 4.1

Table 3

Change in mean lean body mass during the course of tuberculosis treatment (N = 48)

Time points	BIA	p value*	DXA	p value#
Baseline	35.6 ± 6.9	0.0004	33.6 ± 5.7	< 0.01
First follow-up	36.8 ± 8.0		-	
Second follow-up	37.6 ± 8.6		35.9 ± 6.4	
*Repeated measures ANOVA; #Paired t-test				

Mean ± SD weight gain during ATT was 4.3 ± 3.1 Kgs. Corresponding mean ± SD increases in LBM were 2.4 ± 2.6 Kgs by BIA and 2.0 ± 3.7 Kgs by DXA. Male participants gained 4.4 ± 3.1 Kgs and females gained 3.6 ± 3.0 Kgs in body weight. During intensive and continuation phases, mean ± SD weight gain was 2.1 ± 1.9 Kgs and 2.2 ± 3.0 Kgs, respectively, while BIA-measured LBM increased by 1.2 ± 2.9 Kgs and 0.8 ± 2.6 Kgs. (Table 2, 3, 4)

Table 4
Mean difference of lean body mass during the course of tuberculosis treatment (N = 48)

Method	BIA*			DXA#		
Comparison	Mean difference	95% CI	p value	Mean difference	95% CI	p value
Baseline vs End of IP	1.2	0.07–2.3	0.033	-	-	-
End of IP vs End of ATT	0.8	-0.3-1.9	0.22	-	-	-
Baseline vs End of ATT	2.0	0.9–3.1	< 0.01	2.4	1.6–3.2	< 0.01
* Repeated measures ANOVA and post-hoc Bonferroni test; #Paired t-test						

Overall, 44 (91.7%) participants gained LBM, three (6.3%) lost LBM, and one (2.0%) showed no change. During intensive phase, 35 (72.9%) of participants gained LBM, compared with 29 (60.4%) during continuation phase.

DXA-based regional analysis showed mean increases of 1.3 ± 2.9 Kgs in trunk lean mass and 1.3 ± 2.3 Kgs in limb lean mass. Corresponding gains in trunk and limb fat mass were 1.1 ± 1.2 Kgs and 0.8 ± 1.2 Kgs, respectively. (Table 2)

Approximately 50% (BIA) and 60% (DXA) of total weight gain during ATT was attributable to increases in LBM. Contribution of LBM to weight gain was greater during intensive phase (63.2%) than during continuation phase (36.4%).

DISCUSSION

LBM, assessed using both BIA and DXA, demonstrated distinct pattern of recovery during TB treatment. Nearly three-fourths of total gain in LBM occurred during the intensive phase of ATT whereas, higher proportion of participants experienced decline in LBM during continuation phase despite ongoing microbiological improvement. These findings suggest that intensive phase is the most critical period for recovery of lean tissue, whereas sustaining or further improving lean mass becomes more challenging later in treatment.

The observed longitudinal change in LBM was substantial, with a large effect size (partial $\eta^2 = 0.173$; Cohen's $f = 0.457$), exceeding the medium effect size assumed during sample size estimation. This suggests that Despite loss of participants during follow-up, the study retained sufficient ability to detect significant changes in LBM over the course of ATT. This pattern is consistent with previous studies

describing rapid nutritional recovery early in ATT, driven by suppression of hyper-catabolism, reduction of systemic inflammation, and improvement in appetite as bacillary load decreases. Recovery may subsequently slow or plateau, with deterioration in some patients due to persistent undernutrition, comorbidities, or inadequate nutritional supplementation.^{6,7} Studies from Africa and India have similarly reported that early gains in lean tissue are not always sustained, highlighting need for continued nutritional support and targeted interventions during continuation phase to prevent relapse into wasting and optimize functional recovery.^{7,14,15}

By BIA, mean LBM increased by about 3.0 kg in men and 2.9 kg in women over six months, while DXA showed gains of 3.7 kg and 1.5 kg, respectively. Although women gained more body weight than men (5.6 kg vs. 5.0 kg), their proportionate increase in LBM was lower (5.9% vs. 10.8%), suggesting that a larger share of weight gain was attributable to fat mass. Overall, 22.2% of women and 2.6% of men experienced LBM decline during treatment. During the intensive phase, only one woman experienced LBM decline, whereas during the continuation phase approximately 42% of men lost lean mass compared with 22% of women. Thus, men demonstrated greater early gains in lean tissue, but these appeared less sustainable, whereas women showed smaller but relatively steadier improvements. This pattern suggests gender-specific nutrient partitioning and underscores the need for tailored nutritional interventions, consistent with findings from Uganda where wasted men preferentially regained lean tissue while women tended to regain fat mass.⁶

The limited restoration of lean tissue observed in our study is consistent with findings from rural India, where severe chronic undernutrition often persisted despite successful treatment completion.¹⁴ A study from Singapore demonstrated that TB-associated wasting involves depletion of both fat and lean tissue, particularly skeletal muscle, with significant functional consequences including reduced grip strength and impaired physical performance.^{16–18} Studies from the United Kingdom showed that much of the weight regained during treatment was attributable to fat rather than protein mass, while studies from Malawi linked lean mass depletion with micronutrient deficiencies and more severe disease.^{15,19,20} Collectively, these findings indicate that nutritional recovery in TB remains incomplete unless interventions specifically target restoration of lean tissue. Increases in body weight does not necessarily reflect restoration of muscle mass.

Taken together, our findings emphasize that LBM is more robust and functionally relevant marker of nutritional recovery than weight or BMI alone. Although weight and BMI are practical indicators in routine programmes, they do not adequately reflect the quality of nutritional recovery. Assessment of LBM using DXA or BIA can identify patients whose apparent anthropometric recovery is driven largely by fat gain rather than restoration of muscle mass, with potential implications for physical function, relapse risk, and long-term outcomes.

The observation that a substantial proportion of patients lost LBM during the continuation phase suggests that while early gains are driven by reduction in infection-related catabolism, sustained recovery requires adequate nutritional intake and metabolic support. Identifying patients who fail to

regain or who lose LBM during therapy may facilitate timely nutritional interventions. Women, individuals with metabolic comorbidities, and those with tobacco or alcohol use may particularly benefit from enhanced monitoring and targeted support.

From a policy perspective, reliance on weight or BMI alone is insufficient to assess nutritional recovery during TB treatment. Incorporating lean mass assessment at defined time points could provide a more accurate picture of nutritional recovery.

The study has several limitations. Social desirability bias may have led to underreporting of tobacco and alcohol use, and objective tools such as AUDIT-C were not used to assess alcohol consumption which might have led to information bias. Although BIA is portable and inexpensive, its accuracy is influenced by hydration status and illness-related factors, contributing to the wide limits of agreement observed with DXA. Finally, as the study was conducted at a single tertiary care centre, the findings may not be fully generalizable to other settings with different socioeconomic, dietary practices and epidemiological profiles.

Declarations

Funding: No funding

Conflict of interest: None

Artificial intelligence (AI): AI was used only to improve the language and quality of the manuscript and was not used to generate or modify any data or results. All scientific content was reviewed and verified by the authors.

Clinical trial number: Not applicable

Ethics approval and consent to participate:

Ethical approval (JIP/IEC-OS/18/2023) for the study was obtained from the Institute Ethics Committee (IEC), JIPMER, Puducherry. Written informed consent was obtained from all participants prior to enrolment in the study.

Consent for publication:

Not applicable. No individual participant-identifiable information is included in this manuscript.

Competing interests:

The authors declare that they have no competing interests.

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Authors' contributions:

Sonali Sarkar and Palanivel C conceptualised the study and developed the main research idea. Saptarshi Biswas contributed to data collection, data analysis, and drafting of the manuscript. Ruben Raj contributed to the data analysis. All authors reviewed and critically revised the manuscript and approved the final version.

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Data availability:

The data supporting the findings of this study are available from the corresponding author on reasonable request, subject to appropriate ethical and institutional restrictions.

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Figures

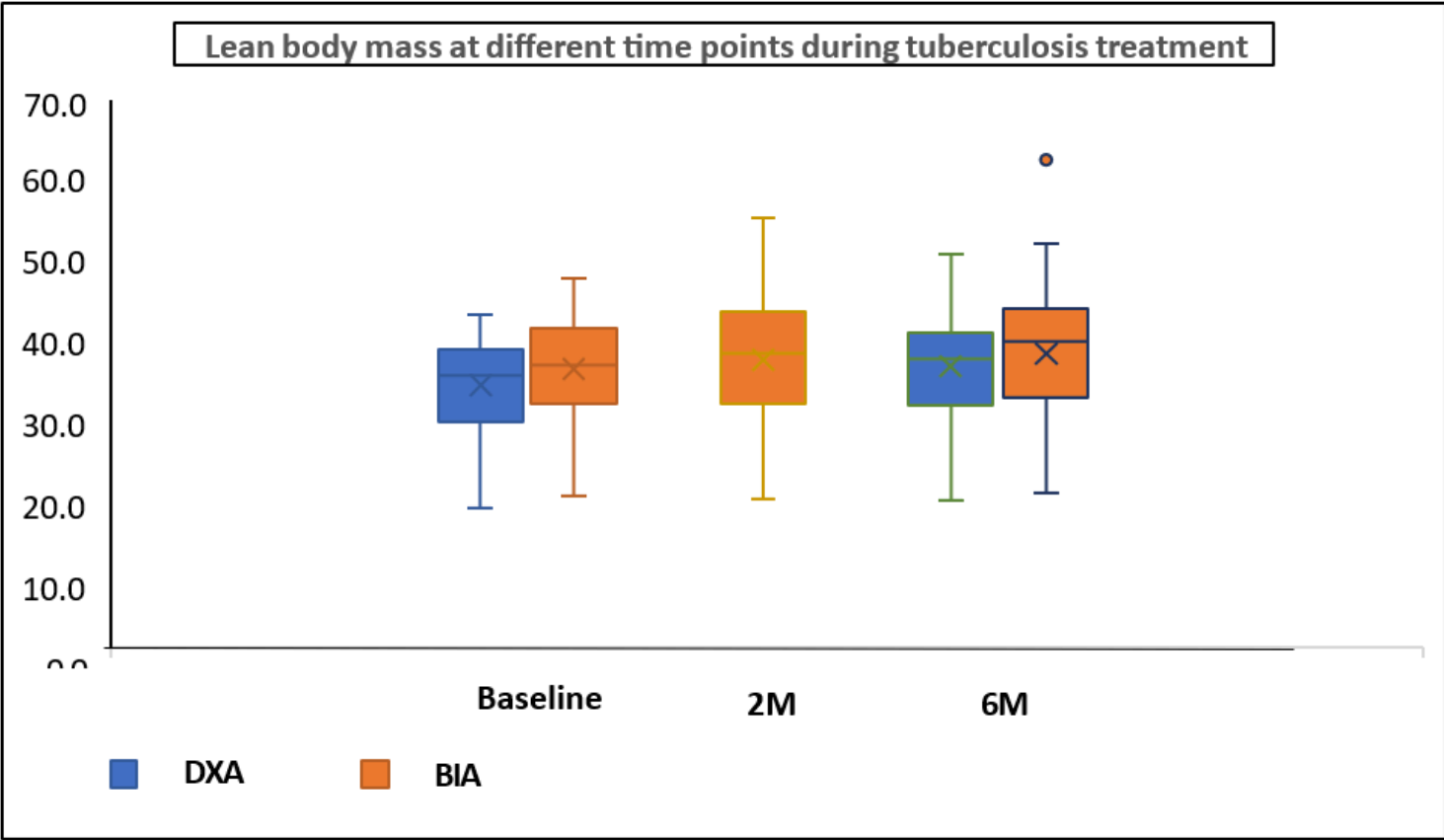


Figure 1

Lean body mass at different time points during tuberculosis treatment