

Original Article

Body Composition Alterations in Patients with Chronic Stroke: A Cross-Sectional Study

Jittima Saengsuwan¹, Charuwan Nimphan¹, Charoonsak Somboonporn², Preeda Arrayawichanon¹¹Department of Rehabilitation Medicine, Faculty of Medicine, Srinagarind Hospital, Khon Kaen University, Thailand;²Nuclear Medicine Division, Department of Radiology, Faculty of Medicine, Srinagarind Hospital, Khon Kaen University, Thailand

Abstract

Objectives: To investigate differences in body composition (fat mass, lean body mass [LBM] and bone mineral content [BMC]) between the paretic and non-paretic sides in patients with chronic stroke, and to identify predictive factors for paretic LBM and BMC. **Methods:** This cross-sectional study included 84 patients (58 men, 26 women; age 58.3 ± 9.5 years). Body composition was assessed using dual-energy X-ray absorptiometry to measure fat mass, LBM and BMC of the paretic and non-paretic limbs. **Results:** LBM and BMC of both the upper and lower extremities were significantly lower on the paretic side. Fat mass in the paretic lower limb was significantly higher. The greatest percentage differences were found in upper limb BMC (-16.8%), lower limb LBM (-7.9%) and lower limb BMC (-7.3%). Predictors of paretic LBM included age, body mass, motor strength (Motricity Index), and body fat percentage. For paretic BMC, sex and height were significant predictors in both limbs: paretic arm LBM predicted arm BMC, while body mass and age predicted paretic leg BMC. **Conclusions:** Significant differences in body composition exist between the paretic and non-paretic sides in chronic stroke. This study identified key predictors of paretic LBM and BMC.

Keywords: Body Composition, Bone Mineral Content, Fat Mass, Lean Body Mass, Stroke

Introduction

Changes in body composition, including a decrease in muscle and bone mass, are common with ageing^{1,2}. In individuals with stroke, significant skeletal muscle loss has been well documented, particularly on the hemiparetic side. However, declines in muscle mass and strength have also been observed in the non-paretic limbs³. In addition to muscle atrophy resulting from neuronal deafferentation, other contributing factors include catabolic-anabolic imbalance, systemic inflammatory activation and local metabolic alterations within the muscles⁴⁻⁷. Moreover, physical inactivity, fatigue, deconditioning and poor nutrition further exacerbate these changes^{4,6}.

Studies have shown that stroke survivors have a higher prevalence of sarcopenia compared to age-, sex- race- and BMI-matched controls⁸. Muscle loss is also associated with osteoporosis⁹. Alongside the decline in muscle mass, bone mineral content (BMC) also declines significantly. In some patients with stroke, bone loss in the paretic arm during the first year post-stroke can be equivalent to more than 20 years of bone loss in healthy individuals of the same age¹⁰. Ramnemark et al. found that BMC on the paretic side significantly decreased within the first year post-stroke, beginning as early as 4-month follow-up, whereas changes in lean mass were relatively minor¹¹. This bone loss may result from several factors including muscle weakness, reduced bone loading, limited mobility, poor nutrition, iatrogenic influences and decreased sunlight exposure¹². A reduction in bone mass, particularly to osteoporotic levels, significantly increases fracture risk¹³. Stroke survivors are estimated to have a 1.2 to 1.7 fold increased risk of hip fracture compared to the general population¹⁴⁻¹⁶.

In contrast to the loss of muscle and bone mass, fat mass tends to increase following a stroke^{17,18}. Contributing mechanisms may include impaired glucose metabolism, immobilization, and fatty infiltration of disused muscles¹⁹.

The authors have no conflict of interest.

Corresponding author: Jittima Saengsuwan, Department of Rehabilitation Medicine, Faculty of Medicine, Srinagarind Hospital, Khon Kaen University, Khon Kaen, 40002, Thailand
E-mail: sjittima@kku.ac.th

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A meta-analysis revealed a slight increase in fat mass in the paretic arm compared to the non-paretic arm, but no significant differences were observed in the legs²⁰.

Previous studies examining post-stroke changes in body composition have often involved small sample sizes, and focused solely on group comparisons, limiting the ability to explore a broad range of predictive factors²⁰⁻²². The objectives of this study were: (1) to investigate differences in body composition (fat mass, lean body mass [LBM] and bone mineral content [BMC]) between the paretic and non-paretic sides in patients with chronic stroke; and (2) to identify predictive factors of LBM and BMC in the paretic arm and leg.

Materials and Methods

This study is a secondary analysis of previously collected data from 84 patients with chronic stroke, recruited between October 2020 and January 2022. Detailed methodology has been described in a prior publication²³. Inclusion criteria were: age ≥ 18 years, a history of ischaemic or haemorrhagic stroke with onset more than 6 months, the ability to understand and follow commands, and the capacity to provide informed consent. Exclusion criteria included unstable vital signs, use of medications known to affect body mass or composition (e.g. steroids, thyroid hormones, diuretics), and the presence of comorbidities that could impair walking performance (e.g. chronic joint pain). This study was approved by the Khon Kaen University Ethics Committee for Human Research (Ref. HE631455), and written informed consent was obtained from all participants prior to their enrolment.

Clinical assessments

Clinical assessments included measurements of body mass, height, body mass index (BMI) and handgrip strength, as well as evaluations using National Institutes of Health Stroke Scale (NIHSS), the Motricity Index for the upper and lower extremities²⁴, the Modified Rankin Scale (MRS), the Functional Ambulation Category (FAC), and assessments of spasticity.

Body composition measurements

Participants' lean body mass (LBM), fat mass and the bone mineral content (BMC) were measured using dual-energy X-ray absorptiometry (DXA). Measurements were conducted according to standard procedures with a Lunar Prodigy DXA scanner (GE Healthcare, Wisconsin, USA). All participants were instructed to remove their clothing and wear examination gowns during the scan.

Statistical Analysis

Continuous data are presented as mean and standard deviation (SD) for normally distributed variables and as

Table 1. Baseline demographic and clinical characteristics of patients with stroke (n = 84).

| Variables | n (%) |
|---|-------------------|
| Male | 58 (69.0) |
| Age (years), mean (SD) | 58.3 (9.5) |
| BMI (kg/m ²), mean (SD) | 24.2 (3.7) |
| Smoking | 2 (2.4) |
| Alcoholic drinking | 11 (13.1) |
| Comorbidities | |
| Hypertension | 56 (66.7) |
| Diabetes mellitus | 31 (36.9) |
| Dyslipidemia | 22 (26.2) |
| Charlson comorbid score, median (p25, p75) | 3 (2, 4) |
| Ischaemic stroke | 55 (65.5) |
| Left hemiparesis | 50 (59.5) |
| Duration after first stroke, median (p25, p75) | 2.2 (1.3, 3.5) |
| Recurrent stroke | 12 (14.3) |
| NIHSS, median (p25, p75) | 4 (2, 7) |
| FAC 4-5 | 40 (47.6) |
| Motricity index | |
| Upper extremity, mean (SD) | 52.1 (29.2) |
| Lower extremity, mean (SD) | 55.9 (26.6) |
| Maximal hand grip (kg), mean (SD) | 25.2 (9.3) |
| Comfortable gait speed (m/s), median (p25, p75) | 0.47 (0.23, 0.86) |
| % Fat mass, mean (SD) | 31.1 (7.3) |
| ASMI (kg/m ²), mean (SD) | 6.9 (1.2) |
| <i>Abbreviations: ASMI = appendicular skeletal muscle mass index; BMI = body mass index; FAC = Functional Ambulation Category; NIHSS = National Institutes of Health Stroke Scale; p25 = 25th percentile; p75 = 75th percentile; SD = standard deviation.</i> | |

median with 25th and 75th percentiles for non-normally distributed variables. The Shapiro-Wilk test was used to assess the distribution of continuous variables. Categorical data are shown as frequency and percentage. Paired t-tests were used to compare differences between the paretic and non-paretic sides. Percentage differences were calculated per participant as $\% \Delta_i = 100 \times (X_{i, \text{paretic}} - X_{i, \text{non-paretic}}) / X_{i, \text{non-paretic}}$, where X is LBM, BMC, or fat mass. We summarized the set of $\% \Delta_i$ values across participants as mean (95% CI); 95% CIs came from a one-sample t-interval. Negative values indicate paretic < non-paretic.

To explore the predictive factors for LBM and BMC in the paretic arm and leg, a stepwise multiple linear regression analysis was conducted. Based on Pang et al., key predictors of BMC included lean body mass, height, muscle strength and

Table 2. Comparison of body composition between the paretic and non-paretic sides in patients with chronic stroke.

| | Paretic side, mean (SD) | Non-paretic side, mean (SD) | Mean difference (95 % CI) | p-value | Mean Percentage difference (95 % CI) |
|--------------------------|-------------------------|-----------------------------|---------------------------|---------|--------------------------------------|
| Upper extremity | | | | | |
| Lean body mass (g) | 2335.2 (665.4) | 2527.3 (693.7) | -192.1 (-276.7 to -107.5) | < 0.001 | -6.10 (-10.05 to -2.15) |
| Fat mass (g) | 1127.5 (382.4) | 1109.5 (381.4) | 18.1 (-12.5 to 48.6) | 0.24 | 3.46 (-0.10 to 7.02) |
| Bone mineral content (g) | 138.5 (42.9) | 161.2 (39.8) | -22.6 (-28.0 to -17.2) | < 0.001 | -16.79 (-20.90 to -12.68) |
| Lower extremity | | | | | |
| Lean body mass (g) | 6641.2 (1539.3) | 7245.0 (1725.5) | -603.9 (-724.7 to -482.9) | < 0.001 | -7.90 (-9.48 to -6.33) |
| Fat mass (g) | 3216.2 (1228.2) | 3129.2 (1186.3) | 87.0 (41.3 to 132.7) | < 0.001 | 2.95 (1.40 to 4.50) |
| Bone mineral content (g) | 391.3 (100.3) | 420.1 (95.0) | -28.8 (-35.2 to -22.4) | < 0.001 | -7.32 (-9.01 to -5.63) |

Abbreviations: CI = confidence interval, g = gram, SD = standard deviation.

sex²⁵. For LBM prediction, the factors examined in this study included age, sex, body mass, height, body fat percentage, motor impairment (upper or lower extremity Motricity Index), duration after stroke, spasticity and cognitive impairment. For BMC prediction, the factors explored were age, sex, body mass, height, lean body mass, motor impairment, duration after stroke, spasticity and cognitive impairment. Multicollinearity was assessed using variance inflation factors (VIFs), with all predictors having VIF values below 5 (ranging from 1.02 to 2.04). The normality of residuals was evaluated through visual inspection (histogram and Q-Q plot), and heteroscedasticity was tested using the Breusch-Pagan test. Across all models, residuals were approximately normally distributed, and no evidence of heteroscedasticity was detected. The best-fit models were selected based on the highest adjusted coefficient of determination (adjusted R²), the lowest standard error of estimate (SEE) and the lowest Akaike Information Criterion (AIC). For all analyses, two-tailed p-values ≤ 0.05 were considered statistically significant²⁶. Statistical analyses were performed using STATA (Stata Statistical Software: Release 18, College Station, TX: StataCorp LLC.).

Results

The study included 84 patients with chronic stroke with a mean age of 58.3 years. The average duration since the first stroke was 2.2 years, and 14.3 % of participants had experienced recurrent stroke episodes. The majority had left-sided hemiparesis (59.5 %) and a history of ischaemic stroke (64.6 %) (Table 1).

A significant reduction in lean body mass (LBM) and bone mineral content (BMC) was observed in both the upper and lower extremities on the paretic side. Additionally, fat mass in the paretic lower extremity was significantly higher compared to the non-paretic side. The three largest

percentage differences were found in BMC of the upper extremities (-16.8 %), LBM of the lower extremities (-7.9 %) and BMC of the lower extremities (-7.3 %) (Table 2).

Multiple linear regression analysis revealed that age, body mass, motor power (assessed by the Motricity Index) and body fat percentage were predictive factors for the paretic LBM in both the arm and leg. For paretic BMC, sex and height were significant predictors in both limbs: paretic arm LBM predicted arm BMC, while weight and age predicted paretic leg BMC (Table 3).

Discussion

The aim of this study was to investigate differences in body composition (fat mass, lean body mass [LBM] and bone mineral content [BMC]) between the paretic and non-paretic sides in patients with chronic stroke, and to identify predictive factors for paretic LBM and BMC. Our results showed that the hemiparetic limbs had significantly lower LBM and BMC. Additionally, the paretic lower extremity had a higher fat mass compared to the non-paretic side. Both the paretic arm and leg demonstrated reduced lean mass. The mean difference in LBM between the paretic and non-paretic arms was 6.1 %, which is consistent with previous studies reporting differences in the range of 7 to 9 %^{19,25}. Similarly, the LBM difference between the paretic and non-paretic legs was 7.9 %, aligning with earlier findings that reported differences ranging from 3 % to 8 %^{21,27,28}.

We found that age, body mass, composite motor power (assessed by Motricity Index) and body fat percentage were significant predictors of lean mass in both the paretic arm and leg. It is well established that ageing is associated with a decline in muscle mass^{29,30}. Regarding composite motor power, our findings align with those of Pang et al. who identified muscle strength as one of the significant predictors of lean mass in the paretic arm²⁵. This highlights

Table 3. Proposed equations for estimation of LBM and BMC of hemiparetic arm and leg.

| Predictor | Univariate β (95 % CI) | p-value | Final multivariable model β (95 % CI) | p-value |
|----------------------------|------------------------------|---------|---|---------|
| Paretic LBM arm (g) | | | | |
| Age (years) | -18.3 (-33.1, -3.5) | 0.016 | -15.1 (-24.2, -6.0) | < 0.001 |
| Body mass (kg) | 41.7 (33.1, 50.2) | < 0.001 | 44.6 (37.1, 52.1) | < 0.001 |
| UEMI score | 2.3 (-2.7, 7.3) | 0.37 | 3.6 (0.7, 6.6) | 0.017 |
| Body fat percentage (%) | -10.6 (-30.5, 9.2) | 0.29 | -28.4 (-40.3, -16.6) | < 0.001 |
| Constant | NA | NA | 1011.0 (215.3, 1806.7) | 0.013 |
| Paretic LBM leg (g) | | | | |
| Age (years) | -42.0 (-76.2, -7.8) | 0.017 | -28.6 (-46.3, -10.8) | 0.002 |
| Body mass (kg) | 41.7 (33.1, 50.2) | < 0.001 | 108.3 (93.4, 123.3) | < 0.001 |
| LEMI score | 12.9 (0.5, 25.3) | 0.041 | 9.7 (3.3, 16.1) | 0.004 |
| Body fat percentage (%) | -33.5 (-79.2, 12.2) | 0.148 | -70.8 (-94.9, -46.7) | < 0.001 |
| Constant | NA | NA | 2935.8 (1285.7, 4586.0) | 0.001 |
| Paretic BMC arm (g) | | | | |
| Female | -60.8 (-76.1, -45.6) | < 0.001 | -27.1 (-42.0, -12.1) | 0.001 |
| Height (m) | 364.1 (279.8, 448.4) | < 0.001 | 141.0 (48.6, 233.5) | 0.003 |
| Paretic LBM arm (g) | 0.05 (0.04, 0.06) | < 0.001 | 0.03 (0.02, 0.04) | < 0.001 |
| Constant | NA | NA | 71.9 (45.8, 98.0) | < 0.001 |
| Paretic BMC leg (g) | | | | |
| Age (years) | -3.0 (-5.3, -0.8) | 0.007 | -1.5 (-2.8, -0.3) | 0.012 |
| Female | -151.3 (-185.1, -117.5) | < 0.001 | -79.9 (-110.6, -49.1) | < 0.001 |
| Body mass (kg) | 5.1 (3.5, 6.6) | < 0.001 | 1.8 (0.6, 3.0) | 0.003 |
| Height (m) | 963.0 (792.2, 1133.7) | < 0.001 | 505.0 (304.5, 705.4) | < 0.001 |
| Constant | NA | NA | -432.9 (-762.5, -103.3) | 0.011 |

Paretic LBM arm: Adjusted R² = 0.67, SEE = 385.0, AIC = 1243.4; Paretic LBM leg: Adjusted R² = 0.75, SEE = 768.9, AIC = 1359.6; Paretic BMC arm: Adjusted R² = 0.66, SEE = 24.9, AIC = 782.3; Paretic BMC leg: Adjusted R² = 0.73, SEE = 52.1, AIC = 907.4
Abbreviations: AIC = Akaike Information Criterion; BMC = bone mineral content; CI = confidence interval; LBM = lean body mass; LEMI = lower extremity motricity index; NA = not applicable; SEE = standard error of estimate; UEMI = upper extremity motricity index.

the essential role of muscle function in the maintenance of muscle mass.

Regarding the impact of fat mass on lean body mass, previous studies support our findings. Cameron et al. conducted a five-year prospective study in adults over the age of 70 and found that while baseline thigh lean mass did not predict future loss of muscle, a higher baseline body fat percentage was associated with greater muscle loss in men². Similarly, Kim et al. reported that visceral obesity was associated with future skeletal muscle mass loss in Korean adults³¹. In addition, Koster et al. found that higher fat mass was associated with a greater decline in leg lean mass in both men and women³². These findings, along with our results, underscore the negative influence of fat mass on muscle mass. A likely explanation involves the pro-inflammatory role of excess fat tissue, which releases cytokines such as tumor necrosis factor- α and interleukin-6^{33,34}, both of which contribute to muscle atrophy and sarcopenia³⁵. Unlike findings reported by Celik et al, we did not observe

a direct association between spasticity and muscle mass preservation²¹. It is possible that the long-term effects of spasticity on muscle preservation may be better explored through prospective studies.

Bone loss after stroke has been observed on the paretic side as early as 3 to 4 months post-stroke, with continued progression over the first year^{11,36}. Studies have shown that the rate of bone loss slows thereafter¹². Ramnemark et al. reported that BMC in the upper extremities showed a significant difference from the non-paretic side as early as four month post-stroke, whereas the difference in the lower extremities became significant at the 7-month follow-up¹¹. In our study, the greatest percentage difference in BMC was observed in the upper extremities, with a nearly 20 % reduction compared to the non-paretic side. This finding in participants with chronic stroke aligns with the study by Hamdy et al. which found that bone demineralization, as measured by DXA, was more pronounced in the upper limbs than in the lower limbs³⁶. Previous studies have

shown that BMC on the paretic side was 13.8 % to 17.4 % lower in the upper extremities and approximately 12.2 % lower in the lower extremities compared to the non-paretic side^{11,25}.

The clinical significance of BMC loss after stroke lies in its contribution to skeletal fragility and increased fracture risk, particularly hip fractures. In our study, sex and height were significant predictors of BMC in both the paretic arm and leg, while age and body mass were additional predictors for the leg. Moreover, arm LBM was a significant predictor of arm BMC. It is well known that bone mass declines with age³⁷. Regarding sex, although no studies have specifically examined sex differences in the rate of BMC decline following stroke, data from healthy populations suggest that women experience greater bone mass loss with ageing, likely due to menopause^{38,39}. LBM plays a critical role in BMC. Celik et al. demonstrated a significant correlation between LBM and BMC on both the paretic and non-paretic sides²¹. Another study identified lean mass in the paretic arm, height, and muscle strength as significant predictors of BMC in the paretic arm²⁵. In our study, although we also found paretic arm and height as significant predictors, we did not find muscle strength to be a direct predictor of BMC. This difference may be due to the indirect influence of muscle strength on BMC via its effect on LBM, as well as differences in muscle strength measurement methods. Our study used the Motricity Index, which may be less precise than the hand-held dynamometry used in previous research²⁵.

Regarding fat mass, we found a significantly higher fat mass in the paretic leg but no significant difference in the paretic arm. The literature presents mixed findings on fat mass distribution after stroke²⁰. Lazoura et al. reported that fat gain over time was similar between the paretic and non-paretic sides in their prospective study¹⁷. In contrast, Pang et al. demonstrated that the hemiparetic arm had 6.3 % higher fat mass in patients at least one-year post-stroke²⁵. Ryan et al. found that fat mass was higher on the paretic side than on the non-paretic side in ambulatory stroke patients⁴⁰. However, a subsequent study by Ryan et al. reported no differences in fat mass between the paretic and non-paretic sides of the arms, legs, and thighs¹⁹. In addition, CT scans in the same study showed no differences in mid-thigh subcutaneous fat between the paretic and non-paretic legs¹⁹. The differences in fat mass findings across studies may be attributed to variations in stroke severity among participants. Previous studies primarily recruited ambulatory stroke patients, whereas our study included patients with varying degrees of severity, including both ambulatory and non-ambulatory individuals. This broader inclusion may have contributed to the more pronounced differences observed in our study.

Post-stroke muscle changes affect both limbs, likely due to disuse atrophy and reduced descending drive via both ipsilateral and contralateral pathways. Previous studies have demonstrated that both the paretic and non-paretic limbs exhibit deficits in muscle size and strength²⁷.

Therefore, side-to-side comparisons may underestimate the true decline in both limbs. A longitudinal study is needed to track these changes over time and to provide a clearer understanding of their progression.

Changes in BMC and fat mass are also dynamic. For example, an increase in BMD in the non-affected arm has been observed, which may be due to increased activity or a redistribution of BMC from the paretic side¹¹. Additionally, the human body is not perfectly symmetric; for instance, the right lower extremity has been shown to have significantly higher BMC and fat mass in able-bodied individuals⁴¹. Therefore, the results should be interpreted with caution.

Findings from this study highlight substantial musculoskeletal alterations on the paretic side after stroke, with notable losses in bone and muscle mass and an increase in lower limb fat. The pronounced deficits in BMC, particularly in the upper extremities, and in LBM in the lower extremities, may increase the risk of osteoporosis, sarcopenia, and functional decline. Rehabilitation and nutritional strategies should aim for both muscle preservation and bone health. Early interventions tailored to modifiable predictors, such as improving muscle strength and managing body composition, may help mitigate progressive musculoskeletal deterioration and improve functional outcomes.

In the future, a longitudinal study should be conducted to assess changes in body composition after stroke, including the rate of change and the factors influencing these changes. One important limitation of our study is its cross-sectional design, which restricts our ability to determine temporal or causal relationships between the variables studied. While associations can be identified, they may be either causal or non-causal. Our primary focus was on identifying predictive factors, which do not necessarily imply causation – a distinction from studies aimed at identifying risk factors⁴². Furthermore, differences in LBM may be underestimated, as DXA cannot quantify fatty infiltration within muscle⁴³. Conversely, body fat mass may also be underestimated by DXA for the same reason. This limitation was highlighted in the study by Ryan et al. where both DXA and CT scans were used. Their findings showed a significantly higher percentage of mid-thigh low-density lean tissue relative to muscle area in the paretic legs, indicating greater intramuscular fat accumulation¹⁹. These observations raise the possibility that the prevalence of sarcopenia in individuals with stroke may be underestimated when relying solely on DXA. In addition, variable selection in this study relied on a stepwise regression approach; therefore, some risk of overfitting cannot be excluded. However, as our primary aim was exploratory, the analysis was intended to identify potential predictors. In this context, the use of stepwise regression was considered appropriate for generating initial insights. Nonetheless, the predictors identified in our study should be validated in future research⁴⁴.

Conclusion

Significant differences in body composition were identified between the paretic and non-paretic sides in patients with chronic stroke, particularly for BMC and LBM. Predictors of paretic LBM included age, body mass, motor strength (Motricity Index), and body fat percentage. For paretic BMC, sex and height were significant predictors in both limbs: paretic arm LBM predicted arm BMC, while body mass and age predicted paretic leg BMC.

Ethics approval

This study was reviewed and approved by the Khon Kaen University Ethics Committee for Human Research (Ref. HE631455).

Consent to Participate

Each participant signed a written informed consent form prior to participation.

Authors' Contributions

Jittima Saengsuwan, Charuwan Nimphan, Charoonsak Somboonporn, and Preeda Arrayawichanon contributed to the conceptualization, research design, analysis, and interpretation of the study, as well as drafting, revising, and reviewing the manuscript.

Jittima Saengsuwan and Charuwan Nimphan participated in the study implementation and data collection. All authors read and approved the final version of the manuscript.

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