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Original Article

Longitudinal Changes in Physical Function and Body Composition in Individuals with Chronic Stroke: An Ambispective Cohort Study

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Abstract

Objectives: To explore longitudinal changes in physical function and body composition in individuals with chronic stroke. **Methods**: This ambispective cohort study included participants from a prior cross-sectional study (2020–2022). Demographic and functional data were collected. Body composition—lean body mass (LBM), bone mineral content (BMC), and fat mass—was assessed using dual-energy X-ray absorptiometry. **Results**: Thirty-five of the 84 participants from the original cohort (41.7 %) completed the follow-up assessment. The mean age of the participants was 63.5 years. Over a median follow-up period of 3.9 years, body mass index increased by 0.8 kg/m^2 (95 % CI, 0.2 kg/m^2 to 1.5 kg/m^2), and comfortable gait speed changed by -0.18 m/s (95 % CI, -0.27 m/s to -0.09 m/s). Bone mineral content decreased in the paretic upper limb (-12.7 g, 95 % CI -20.3 g to -5.0 g), paretic lower limb (-27.4 g, 95 % CI -41.5 g to -13.4 g), and non-paretic lower limb (-14.8 g, 95 % CI -24.0 g to -5.7 g). No substantial changes in lean body mass were observed during follow-up. Fat mass percentage increased by 1.8 % (95 % CI, 0.3 % to 3.2 %). **Conclusions**: Individuals with chronic stroke demonstrated declining gait speed, reduced bone mineral content and increased total body adiposity over time, whereas lean body mass exhibited limited change.

Keywords: Body Composition, Body Fat Distribution, Bone Density, Muscle Strength, Stroke

Introduction

Stroke represents a major public health concern in Thailand. Many stroke survivors experience permanent disability, which substantially affects their quality of life[1]. Stroke leads to multisystem impairments, among which musculoskeletal decline plays a critical role in limiting daily functioning among individuals with stroke. Overall, functional outcomes across multiple domains—including motor performance, ambulation, cognition, language, swallowing, and activities of daily living (ADL)—show significant improvement during the first 12–18 months after stroke onset. However, this recovery phase is typically followed by a plateau and subsequent functional decline beginning approximately 30–60 months post-stroke[2]. Notably, among stroke survivors who were independent in ADL at 3 months after stroke, approximately 3 % per year deteriorated to ADL dependency during long-term follow-up[3].

In addition to changes in physical function observed in individuals with chronic stroke, body composition also undergoes significant alterations. These changes may be attributable to the complex systemic metabolic disturbances and reduced physical activity commonly seen after stroke. Following stroke, fat mass tends to increase[4,5], likely as a result of impaired glucose metabolism, immobilization, and intramuscular fat deposition[6]. In contrast, lean body mass (LBM) and bone mineral content (BMC) tend to decline. Reductions in LBM occur in both paretic and non-paretic limbs[6-8] and are associated with multiple mechanisms, including neuronal denervation, systemic catabolic activation,

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ongoing inflammation, intramuscular lipid deposition, and sympathetic activation[9-12]. Stroke-related weakness leads to restricted mobility and reduced mechanical loading on bone, thereby contributing to bone loss[13]. Ramnemark et al. reported that, within the first year after stroke, BMC in the paretic limbs decreased significantly[14].

The rate and magnitude of changes in body composition vary across studies, and differences in body composition changes have also been observed between individual limbs. These findings highlight the heterogeneous nature of body composition alterations in chronic stroke. However, evidence regarding long-term intra-individual changes in body composition among individuals with chronic stroke remains limited. Therefore, the present study aimed to explore longitudinal changes in physical function and body composition in individuals with stroke. Given the limited longitudinal evidence in this population, the study was intended to generate descriptive data and identify potential patterns of change rather than to test prespecified hypotheses.

Materials and Methods

Study Population and Design

The present study was conducted as an ambispective cohort study in which participants from a previous cross-sectional study[15] were re-contacted and invited to undergo follow-up assessments in 2025. The original study was conducted at the Rehabilitation Unit, Srinagarind Hospital, Khon Kaen University, Thailand between October 2020 and January 2022. It enrolled individuals aged ≥18 years with ischaemic or haemorrhagic stroke of more than 6 months duration who were able to understand and follow commands and provide informed consent. Exclusion criteria included unstable vital signs, use of medications known to affect body mass or body composition (e.g., steroids, thyroid hormones or diuretics), and body mass exceeding 159 kg because of machine limitations. Follow-up assessments were conducted between January 2025 and September 2025. The study was approved by the Khon Kaen University Ethics Committee for Human Research (Ref. HE671416) and written, informed consent was obtained from all participants.

Demographic Data and Clinical Assessments

Data were collected in person at baseline and follow-up through participant interviews and medical record review and included demographic information, comorbidities, stroke-related history, physical activity level (assessed using the Stroke Physical Activity Questionnaire [SPAQ])[16,17], and information regarding recurrent stroke occurring between the baseline and follow-up visits. Follow-up assessments were conducted by trained assessors using standardized assessment procedures. Assessors were blinded to participants' baseline assessment results during follow-up evaluations.

Baseline DXA and functional assessments were obtained during the original cross-sectional study conducted between October 2020 and January 2022 and were used as baseline measurements for the present follow-up study. All outcome assessments were performed by trained assessors using standardized evaluation procedures. Assessors conducting the follow-up evaluations were blinded to baseline assessment results. Stroke severity and functional status were evaluated using the National Institutes of Health Stroke Scale (NIHSS), upper and lower extremity Motricity Index, Modified Rankin Scale (mRS), Functional Ambulation Category (FAC), and Montreal Cognitive Assessment (MoCA).

Anthropometric measurements included body mass, height, body mass index (BMI) and calf circumference. Muscle strength was assessed using analogue handgrip dynamometry (Takei T.K.K.5001, Takei Scientific Instruments Co., Ltd., Tokyo, Japan). Grip strength was measured separately in both hands. Participants held the dynamometer with the upper arm close to the body, the elbow flexed at 90°, and the forearm in a neutral position. They were instructed to squeeze the device with maximal effort. Three trials were performed with each hand, and the single highest value across the six trials was used for analysis.[18].

Physical performance was assessed using the 10-metre walk test and the Five Times Sit-to-Stand test. For the 10-metre walk test, participants walked a total of 10 metres at their usual walking speed. Timing was recorded for the middle 6 metres, excluding the first and last 2 metres to minimize acceleration and deceleration effects. Participants were permitted to use walking aids or rest as needed. Gait speed (m/s) was calculated, and the average of two trials was reported[19]. For the Five Times Sit-to-Stand test, participants were seated upright on a stable, standard-height chair without armrests, with their feet flat on the floor. With their arms crossed over the chest, they were instructed to stand up and sit down as quickly as possible for five repetitions. The total time required to complete the five movements was recorded[20].

Body Composition Measurements

Lean body mass (LBM), fat mass and bone mineral content (BMC) were measured using dual-energy X-ray absorptiometry (DXA). All scans were performed following standard procedures using a Lunar Prodigy DXA scanner (GE Healthcare; software version 18 SP 4.1). Daily quality assurance procedures included mechanical verification using a black-block phantom and calibration using a spine phantom. Participants were positioned according to a standardized protocol, and regional arm and leg composition was determined using the manufacturer's predefined segmentation procedures. All scans were performed by certified radiologic technologists experienced in DXA assessment and analyzed using the same software and standardized quality-control procedures. Further methodological details regarding DXA are presented in

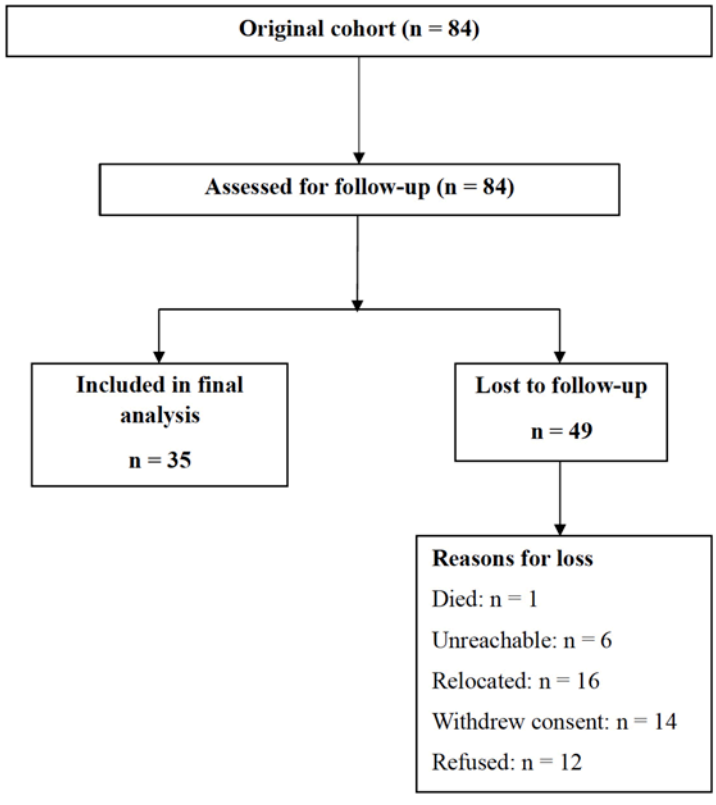

Figure 1. Flow diagram of the study participants.

the Supplementary Material. Participants were instructed to remove their clothing and to wear examination gowns during the scan.

Sample Size Estimation

All participants from the previous study were invited for follow-up[15]. A loss to follow-up of approximately 30 % (due to death, dropout or relocation) was anticipated, yielding an expected sample size of 58 participants. Given the absence of prior longitudinal studies examining these outcomes in this population, this study was intended to characterize longitudinal changes and provide preliminary estimates to inform future research.

Reported sample sizes in exploratory studies with continuous outcomes vary widely, ranging from approximately 8 to 114 participants, with a median of 30 (interquartile range 20–50)[21]. The anticipated sample size for this study therefore fell within the range commonly reported in the literature.

Statistical Analysis

Continuous data are presented as mean and standard deviation (SD) for normally distributed variables and as median (25th and 75th percentiles) for non-normally distributed data. Normality was assessed using the Shapiro–Wilk test. Categorical data are reported as frequency and percentage.

Because this study was designed as an exploratory longitudinal investigation, a broad range of body composition and physical function measures were evaluated to characterize changes over time. No adjustment for multiple comparisons was performed because the objective was to describe longitudinal changes rather than to test a limited set of prespecified hypotheses.

Changes in clinical parameters from baseline to follow-up were examined using paired t-tests for parametric data and Wilcoxon signed-rank tests for non-parametric data. Changes in FAC scores were evaluated using McNemar's test. For continuous outcomes, within-participant mean changes with corresponding 95% confidence intervals were reported.

A paired t-test was used to compare differences in LBM, fat mass, and BMC between baseline and follow-up for each limb. Due to the ambispective design and variability in participant availability for reassessment, follow-up evaluations were not performed at uniform time points across all participants. Therefore, annualized

Table 1. Participant characteristics at the follow-up assessment (n = 35).

Variables	n (%)
Age (years), mean (SD)	63.5 (7.8)
Male	21 (60.0)
Current smoking	1 (2.9)
Marital status	
Married	26 (74.3)
Single/Separated/Divorced	9 (25.7)
Educational level	
Less than or equal to secondary school	12 (34.3)
Higher than secondary school	23 (65.7)
Comorbidities	
Hypertension	27 (77.1)
Diabetes mellitus	24 (68.6)
Dyslipidemia	33 (94.3)
Stroke duration (years), median (P25, P75)	5.7 (4.9, 8.8)
Time interval between baseline to follow-up, median (P25-P75)	3.9 (2.9, 4.2)
Range	2.4 – 4.7
Stroke type	
Infarction	26 (74.3)
Haemorrhage	9 (25.7)
Recurrent stroke during the follow-up interval	4 (11.4)
Hemiparetic side	
Right	11 (31.4)
Left	24 (68.6)
History of fall in previous year	10 (28.6)

Abbreviations: SD, standard deviation; BMI, body mass index; P25, 25th percentile; P75, 75th percentile.

change values were additionally calculated by dividing the individual change score (follow-up minus baseline value) by the elapsed duration between assessments in years. Negative values indicate a decrease from baseline to follow-up.

Missing data were handled using a complete-case approach. Only participants with available baseline and follow-up data for the outcome of interest were included in the corresponding analyses. For all analyses, two-tailed p-values < 0.05 were considered statistically significant[22]. All statistical analyses were performed using Stata (Stata Statistical Software: Release 18. College Station, TX: StataCorp LLC).

Results

Study Population

Of the 84 participants in the baseline cross-sectional study, 35 (41.7%) were successfully contacted and consented to participate in the follow-up study. Among those who did not participate, 1 participant had died, 16 had relocated, 6 could not be contacted, 14 withdrew consent, and 12 declined participation (Figure 1). There were no statistically significant differences in baseline characteristics between participants who participated in this study and those who did not (Supplementary Table S1). The mean age of the participants was 63.5 (7.8) years, and 60 % were men. The median time since stroke at follow-up assessment was 5.7 years. Most participants had ischaemic stroke (74.3 %) and left-sided hemiparesis (68.6 %). The most frequent comorbidity among the participants was dyslipidemia (94.3 %), with other conditions such as diabetes mellitus (68.6 %) and hypertension (77.1 %) also present (Table 1).

Clinical Parameter Outcomes

Over a median follow-up period of 3.9 years, mean body mass increased by 1.9 kg (95 % CI, 0.3 kg to 3.4 kg), accompanied by an increase in mean BMI of 0.8 kg/m^2 (95 % CI, 0.2 kg/m^2 to 1.5 kg/m^2). Comfortable gait

Table 2. Changes in clinical parameters from baseline to follow-up (n = 35).

Variables` Clinical variables	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
NIHSS (score), median (P25, P75)	3 (2, 8)	3 (2, 6)	NA	0.23*	NA
UE Motricity Index	56.1 (36.0)	58.1 (26.1)	-2.0 (-9.0 to 5.0)	0.56	-0.6 (-1.2 to 2.6)
LE Motricity Index	58.0 (34.9)	59.8 (25.6)	-1.8 (-9.3 to 5.7)	0.63	-0.6 (-2.6 to 1.5)
Height (cm)	161.1 (7.7)	161.3 (7.6)	-0.2 (-0.6 to 0.2)	0.25	-0.07 (-0.19 to 0.03)
Body mass (kg)	64.1 (11.8)	62.2 (12.4)	1.9 (0.3 to 3.4)	0.018	0.5 (0.04 to 0.95)
BMI (kg/m^2)	24.6 (3.3)	23.8 (3.6)	0.8 (0.2 to 1.5)	0.017	0.2 (0.02 to 0.41)
Calf circumference (cm)	34.1 (2.9)	33.6 (3.3)	0.5 (-0.3 to 1.2)	0.19	0.2 (-0.04 to 0.36)
Maximal grip strength (kg) (n=30)	26.5 (8.2)	26.2 (9.7)	0.4 (-2.3 to 3.1)	0.78	0.2 (-0.5 to 0.9)
Five Times Sit-to-Stand test (sec), median (P25, P75) (n=20)	20 (16.6, 25.3)	21.6 (15.8, 28.1)	NA	0.081*	NA
Comfortable gait speed (m/s) (n=23)	0.50 (0.30)	0.68 (0.38)	-0.18 (-0.27 to -0.09)	<0.001	-0.04 (-0.07 to -0.02)
MoCA (score), median (P25, P75)* (n=33)	22 (19, 26)	23 (20, 27)	NA	0.37*	NA
FAC, n (%)			Transition		
0-3	21 (60)	18 (51.4)	17 remained in FAC 0-30.38†	NA	
4-5	14 (40)	17 (48.6)	1 improved to FAC 4-5 13 remained in FAC 4-5 and 4 declined to FAC 0-3		
Reported physical activity time in moderate to vigorous intensity (min/week), median (P25, P75)*	15 (0, 42.6)	25 (0, 140)	NA	0.042*	NA

*Continuous variables are presented as mean (SD) unless otherwise indicated. Abbreviations: BMI, body mass index; FAC, Functional Ambulation Category; LE, lower extremity; MoCA, Montreal Cognitive Assessment; NIHSS, National Institutes of Health Stroke Scale; P25, 25th percentile, P75, 75th percentile; UE, upper extremity. FTSTS and gait speed were not performed in participants with functional or mobility limitations or refusal. MoCA was missing due to incomplete documentation. Grip strength change analysis excluded participants without baseline measurements. *Wilcoxon signed-rank test, †McNemar's exact test. For all participants, the maximum grip-strength value used in the analysis was obtained from the non-paretic hand at both baseline and follow-up.*

speed changed by -0.18 m/s (95 % CI, -0.27 m/s to -0.09 m/s). Reported moderate-to-vigorous physical activity also decreased, with the median value declining from 25 to 15 minutes per week. In contrast, changes in upper- and lower-extremity motricity indices, calf circumference, grip strength, cognitive function, Functional Ambulation Category, and Five Times Sit-to-Stand performance showed limited change over time with greater inter-individual variability (Table 2).

Individual changes in comfortable gait speed between baseline and follow-up assessments are shown in Supplementary Figure S1. Four participants experienced a recurrent stroke during the follow-up period. Sensitivity analyses excluding these participants are presented in Supplementary Tables S2–S5. Overall, the direction and magnitude of changes were similar to those observed in the primary analyses. However, the change in the Five Times Sit-to-Stand Test performance reached statistical significance after exclusion of participants with recurrent stroke, whereas it did not in the primary analysis. No other substantive differences were observed.

Table 3. Changes in fat mass and body fat percentage from baseline to follow-up (n = 35).

Fat mass (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
Upper extremity					
Paretic side	1162.2 (298.1)	1085.9 (344.4)	76.3 (-19.7 to 172.3)	0.12	20.0 (-7.2 to 47.1)
Non-paretic side	1148.3 (378.7)	1069.9 (404.4)	78.4 (-1.2 to 157.9)	0.053	20.1 (-4.1 to 44.3)
Lower extremity					
Paretic side	3384.3 (1219.0)	3196.0 (1143.8)	188.3 (-5.2 to 381.9)	0.056	52.9 (-2.0 to 107.8)
Non-paretic side	3306.0 (1175.2)	3114.0 (1090.4)	192.0 (-6.5 to 390.6)	0.058	53.9 (-2.9 to 110.8)
Total body					
Body fat percentage (%)	33.3 (7.6)	31.5 (7.4)	1.8 (0.3 to 3.2)	0.019	0.5 (0.02 to 0.97)

Table 4. Changes in lean body mass and ASMI from baseline to follow-up (n = 35).

Lean body mass (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
Upper extremity					
Paretic side	2127.9 (487.6)	2198.2 (579.5)	-70.3 (-170.3 to 29.7)	0.16	-15.1 (-43.6 to 13.2)
Non-paretic side	2410.5 (583.2)	2430.7 (662.4)	-20.1 (-110.1 to 69.9)	0.65	-4.9 (-30.5 to 20.7)
Lower extremity					
Paretic side	6239.9 (1293.4)	6336.8 (1427.0)	-97.0 (-315.9 to 122.0)	0.37	-27.1 (-87.7 to 33.6)
Non-paretic side	6999.0 (1442.0)	6989.3 (1665.5)	9.7 (-217.4 to 236.8)	0.93	15.6 (-48.8 to 80.0)
Total body					
ASMI (kg/m^2)	6.8 (0.9)	6.8 (1.1)	0.03 (-0.3 to 0.2)	0.82	0.0 (-0.06 to 0.07)
Abbreviations: ASMI, appendicular skeletal muscle mass index.					

Body Composition Outcomes

Body fat percentage increased by 1.8 % (95 % CI, 0.3 % to 3.2 %). Changes in limb fat mass were variable, with mean differences ranging from 76.3 g to 192.0 g across regions (Table 3). For lean body mass (LBM), mean differences ranged from a decrease of 97.0 g in the paretic leg to an increase of 9.7 g in the non-paretic leg, while the appendicular skeletal muscle mass index (ASMI) changed by 0.03 kg/m^2 (Table 4). However, with the exception of body fat percentage, the confidence intervals were wide and included zero, indicating considerable uncertainty regarding the magnitude of the estimated changes. Individual changes in fat mass, body fat percentage, LBM and ASMI between baseline and follow-up assessments are shown in Supplementary Figures S2 and S3.

Bone mineral content decreased in the paretic arm (MD -12.7 g; 95 % CI, -20.3 g to -5.0 g), in the paretic leg (MD -27.4 g; 95 % CI, -41.5 g to -13.4 g), and in the non-paretic leg (MD -14.8 g; 95 % CI, -24.0 g to -5.7 g). In contrast, the non-paretic arm showed a small increase in bone mineral content, but the confidence interval included zero (Table 5). Individual changes in BMC between baseline and follow-up assessments are shown in Supplementary Figure S4.

Table 5. Changes in bone mineral content from baseline to follow-up (n = 35).

Bone mineral content (BMC) (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
Upper extremity					
Paretic side	116.5 (37.7)	129.2 (42.9)	-12.7 (-20.3 to -5.0)	0.002	-3.7 (-6.0 to -1.4)
Non-paretic side	152.5 (40.7)	151.2 (36.3)	1.3 (-5.2 to 7.9)	0.69	0.1 (-1.7 to 1.9)
Lower extremity					
Paretic side	340.0 (103.6)	367.5 (103.8)	-27.4 (-41.5 to -13.4)	<0.001	-8.6 (-13.6 to -3.7)
Non-paretic side	387.4 (95.0)	402.2 (91.4)	-14.8 (-24.0 to -5.7)	0.002	-4.6 (-7.5 to -1.6)

Discussion

This ambispective cohort study aimed to investigate changes in physical function and body composition over time. Our findings revealed significant changes in comfortable gait speed. There were no significant changes in grip strength, cognitive function, Motricity Index and Five Times Sit-to-Stand test. Regarding body composition, significant increases in body mass index and body fat percentage were observed. There were limited changes in LBM. Reductions in BMC were observed in the paretic arm, paretic leg, and non-paretic leg, whereas the non-paretic arm did not show a significant decrease.

With regard to gait speed, most published studies in stroke populations have focused on the early recovery phase, while long-term data are often derived from cross-sectional analyses stratified by time since stroke to infer longitudinal trends[23,24]. Consequently, evidence describing changes in gait speed in individuals with chronic stroke remains limited. In contrast, several longitudinal studies in community-dwelling older adults have consistently demonstrated a progressive decline in gait speed over time, which has been associated with factors such as reduced physical activity, brain volume changes and increased intramuscular fat infiltration[25-27]. The reported rate of gait speed decline varies across studies. Jerome et al. reported an average reduction of 0.004 m/s/year among adults aged 60 years and older[28], whereas Auyeung et al. observed declines of 0.019 m/s/year in men and 0.025 m/s/year in women among community-dwelling Chinese adults aged over 64 years[29]. In addition, Beavers et al. found that gait speed declined by 0.06 m/s over a 4-year period in well-functioning older adults aged 70-79 years, and reported an association between slower gait speed and greater intramuscular fat accumulation[27]. In the present study, participants with chronic stroke demonstrated an annualized gait speed decline of 0.04 m/s/year, which appears greater than the rates reported in the ageing cohorts. However, these comparisons are indirect and our study did not include a non-stroke ageing group. Future longitudinal studies incorporating age- and sex- matched healthy controls are needed to better characterize gait speed trajectories in chronic stroke and to clarify the extent to which they differ from those observed in normal ageing.

Regarding handgrip strength, Schimmel et al. longitudinally assessed grip strength in both upper limbs of individuals with stroke at 0, 6, 12, and 24 months after stroke onset[30]. They found that handgrip strength in the paretic arm was significantly lower than that of the non-paretic arm, with no improvement over time, while grip strength in the non-paretic arm remained unchanged throughout the follow-up period. These findings are consistent with our results, which similarly demonstrated no significant change in maximum grip strength. As with gait speed, data on longitudinal changes in maximum handgrip strength in individuals with chronic stroke remain scarce. Most existing studies focus on the acute to subacute phases after stroke, predominantly assess the paretic arm, and lack extended longitudinal follow-up[31-34]. As a result, the rate and trajectory of grip strength change in chronic stroke populations remain unclear. Nevertheless, studies conducted during acute hospitalization have shown that handgrip strength does not significantly change from the acute phase until 3 months after hospital discharge[35]. In contrast, longitudinal studies in the general older adult population have consistently reported age-related declines in grip strength, with annual reductions of approximately 0.8 kg/year in men and 1.2 kg/year in women[29]. The relative stability of grip strength observed in the non-paretic arm may be attributable to its continued and compensatory use in daily activities.

Regarding cognitive status, Delgado et al. demonstrated that stroke survivors experienced a period of relative cognitive stability lasting up to 8 years post-stroke before a potential decline, as assessed by the Mini-Mental State Examination (MMSE). This finding is consistent with our results, which demonstrated no significant change in cognitive function over this study period[36]. However, it should be noted that the cognitive measures used in both studies were relatively crude and may not have been

sensitive enough to detect subtle changes in specific cognitive domains. More sensitive measures may be better able to identify early or domain-specific cognitive decline.

Evidence regarding longitudinal changes in fat mass after stroke is limited, particularly in the chronic stage. Most available data are derived from studies conducted during the acute and subacute phases after stroke. For example, Lazoura et al. reported significant increases in total-body fat among individuals with stroke, with an approximately 6 % increase between the third and sixth month post-stroke and an 8 % increase between the sixth and twelfth month, representing an overall increase of 14 % during the first year after stroke[4]. Similarly, English et al. reported greater adiposity in individuals with stroke than in healthy controls[37]. Although these studies provide evidence of fat accumulation after stroke, it remains unclear whether similar trajectories persist into the chronic stage.

In the present study, the point estimate suggests higher fat mass at follow-up across all extremities, with similar estimated changes in the paretic and non-paretic limbs. However, these findings should be interpreted with caution because the confidence intervals were wide and included zero (no change). Therefore, the results do not provide conclusive evidence of regional fat mass increases. Nevertheless, the direction of the estimates is broadly consistent with previous studies describing increased adiposity after stroke[4]. The upper extremities demonstrated larger relative changes than the lower extremities. However, because these estimates were not statistically significant and no previous studies have directly compared longitudinal fat mass changes between the upper and lower extremities after stroke, this apparent difference should be considered hypothesis-generating. Most previous studies have focused on comparisons between paretic and non-paretic limbs rather than between-limb regions. Consequently, the mechanisms underlying potential differences in fat accumulation across limb regions remain uncertain. One possible explanation is the reduced use of the upper extremity after stroke[38], given that approximately 70–80 % of stroke survivors experience upper-extremity impairment[39]. Moreover, many individuals retained better motor control in the proximal segments such as the shoulder and elbow than distal segments[40], which may limit performance of activities of daily living (ADLs)[41]. Reduced limb use may contribute to disuse-related changes in body composition, including fat accumulation and fatty infiltration of skeletal muscle over time[2]. Future studies with larger samples are needed to determine whether regional differences in fat mass change truly exist in chronic stroke populations.

A meta-analysis by English et al. reported significantly lower lean tissue mass in paretic compared with non-paretic limbs among individuals ≥6 months post-stroke, consistent with the between-limb differences observed in our cohort at both baseline and follow-up[7]. However, longitudinal evidence describing changes in LBM during the chronic stage of stroke remains scarce. Most available longitudinal studies have been conducted during the acute and subacute phases after stroke. For example, Carin-Levy et al. reported no significant change in appendicular skeletal muscle mass (ASMM) over time during the first year after stroke[5]. Similarly, Jorgensen et al. observed 5–6 % reductions in lower extremity LBM in both the paretic and non-paretic legs within the first two months after stroke, a magnitude of loss comparable to that typically observed over approximately 20 years in healthy older adults[8]. While the paretic leg showed limited recovery of lean mass, the non-paretic leg could regain lean mass over time, particularly among individuals who recovered walking ability. Recovery was also observed in some participants who had not regained ambulation by 2 months post-stroke[8]. The authors proposed that increased loading and compensatory use of the non-paretic leg during transfers and gait may have contributed to this recovery[6]. Although these findings provide important insights into muscle adaptations during the early stages after stroke, they may not reflect long-term body-composition trajectories in individuals with chronic stroke. In the present study, the point estimates suggest a decrease in LBM in the paretic leg and an increase in the non-paretic leg over the follow-up period; however, the confidence intervals were wide and included zero, indicating considerable uncertainty regarding the magnitude of the estimated changes.

Most evidence describing post-stroke bone loss originates from studies conducted during the acute and subacute phases after stroke. These studies suggest that bone loss begins within the first few months after stroke, particularly on the paretic side, progresses over the subsequent year[10,42], and appears to slow thereafter[13]. For example, Ramnemark et al. reported reductions in BMD in the paretic limbs and non-paretic leg, accompanied by an increase in BMD in the non-paretic arm[14]. The greatest losses were observed in the paretic humerus (17.4 %) and proximal femur (12.2 %). Similarly, Lazoura et al. found that between 3 and 12 months after stroke, BMD decreased by 4.6 % in the paretic limb and by 1 % in the non-paretic limb[4], whereas Jorgensen et al. reported one-year reductions of 7 % and 2 % in the paretic and non-paretic legs, respectively[8]. In contrast, longitudinal evidence describing skeletal changes during the chronic stage of stroke remains limited. Although previous studies have primarily assessed BMD, evaluation of BMC provides complementary information regarding absolute bone mineral mass. Because BMD is derived from BMC divided by projected bone area, it may be influenced by bone size, whereas BMC reflects absolute bone mineral mass[43]. Despite these methodological differences, the pattern observed in our chronic stroke cohort was broadly consistent with previous BMD-based studies. Specifically, we observed a decline in BMC in the paretic arm and the paretic leg, whereas smaller reductions were observed in the non-paretic leg. The point estimate suggests a small increase in BMC in the non-paretic arm, although

the confidence interval included zero. Nevertheless, the direction of this change is similar to the increase in the non-paretic arm BMD reported by Ramnemark et al.[14].

One of the key strengths of this study is its longitudinal assessment of individuals with chronic stroke over a median follow-up period of 3.9 years. Long-term follow-up allowed us to observe longitudinal patterns in physical activity, cognitive function, and other functional domains. Understanding long-term alterations in physical performance and body composition can help guide rehabilitation planning and potentially prevent later complications. Notably, our study observed significant reductions in BMC in the paretic limb as well as in the non-paretic leg. This finding underscores the need for greater attention to bone health after stroke, an area that remains understudied. Additionally, although gait speed declined significantly, LBM in individuals with chronic stroke did not change significantly over the follow-up period. This may be due to limitations of DXA, which cannot detect intramuscular fat or fibrosis[44], potentially leading to overestimation of LBM. These findings highlight that assessments of sarcopenia in individuals with chronic stroke may underestimate the true extent of muscle deterioration.

Several limitations should be acknowledged. First, although the estimated sample size was 58 participants, only 35 of the original 84 participants (41.7 %) completed the study. Attrition occurred because of death, relocation, inability to contact, withdrawal of consent or refusal to participate, which may lead to selection bias and limit the generalizability of the findings. Furthermore, because some participants did not participate owing to clinical improvement whereas others were unable to complete follow-up assessments because of disability or health-related limitations, the missing data mechanism was unlikely to be completely random. Consequently, the observed changes in body composition and physical function may not fully represent those of the original cohort. In particular, mobility-related outcomes may have been biased towards participants with better functional status, potentially leading to an underestimation of functional decline in the broader population of individuals with chronic stroke. Second, the absence of a non-stroke comparator group limits interpretation of the observed changes. Ageing-related declines in physical function, alterations in body composition, and reductions in bone health occur in the general population; therefore, the present study can only describe longitudinal changes observed within this cohort and cannot determine the extent to which these changes are attributable specifically to chronic stroke. Third, the relatively long interval between baseline and follow-up assessments limited our ability to characterize the temporal pattern of change. The study was not designed to establish causal trajectories of chronic stroke recovery or decline. In addition, recurrent stroke experienced by some participants, changes in rehabilitation exposure, medication, nutritional status, osteoporosis treatment, and other health events occurring during follow-up were non-systematically collected or adjusted for and may have influenced the findings. Therefore, the findings should be interpreted as descriptive longitudinal observations rather than as evidence of causal trajectories or attributable effects of specific clinical or lifestyle factors. Fourth, the analyses were exploratory in nature. Multiple outcomes were evaluated, including measures of mobility, strength, lean mass, fat mass and bone mineral content. No adjustment for multiple comparisons was performed because the objective was to describe longitudinal changes across a wide range of outcomes rather than to test a limited set of prespecified hypotheses. Therefore, the findings should be interpreted with caution with emphasis placed on the magnitude and precision of the observed changes rather than on statistical significance alone. Finally, although standardized DXA acquisition and quality-control procedures were used, measurement error remains possible, particularly for regional body composition measures where observed changes may be small relative to the precision of the technique. Future studies should recruit larger cohorts with improved retention strategies, include age- and sex-matched non-stroke comparison groups, collect longitudinal information on rehabilitation exposure, medical comorbidities, medication use, nutrition, and recurrent vascular events, and incorporate more frequent assessments to better characterize patterns of change over time. Such studies would help distinguish stroke-related changes from normal ageing processes and improve understanding of long-term physical function and body composition after stroke.

Conclusion

Individuals with chronic stroke demonstrated declining gait speed, reduced bone mineral content, and increased total body adiposity over a median duration of 3.9 years, whereas lean body mass exhibited limited change.

Ethics Approval

This study was reviewed and approved by the Khon Kaen University Ethics Committee for Human Research (Ref. HE671416).

Consent to Participate

All participants signed a written informed consent form prior to participation.

Authors' Contributions

Piyathida Orantanasan, Patpiya Sirasaporn, Charoonsak Somboonporn and Jittima Saengsuwan contributed to the conceptualization, research design, analysis, and interpretation of the study, as well as drafting, revising, and reviewing the manuscript. Jittima Saengsuwan and Piyathida Orantanasan participated in the study implementation, data collection and confirmed the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

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Supplementary Material

Detailed description of the DXA acquisition procedures, quality-control processes, participant positioning, regional analysis methods, and scanner precision characteristics used in the study.

DXA Acquisition and Analysis Protocol

Body composition was assessed using a Lunar Prodigy dual-energy X-ray absorptiometry (DXA) scanner (GE Healthcare; software version 18 SP 4.1, serial number 510981). All scans were performed at the same facility using the same scanner throughout the study period.

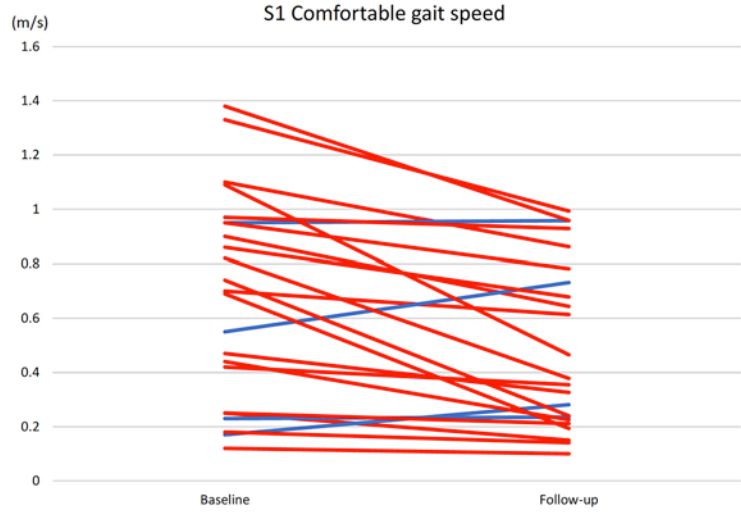
Quality assurance procedures were conducted according to institutional protocols. Daily quality assessment included mechanical verification using a black-block phantom. Daily quality control was performed using a spine phantom to assess measurement accuracy and precision before participant scanning. Preventive maintenance and major calibration were performed quarterly by manufacturer-certified engineers.

Participants were scanned in the supine position according to the manufacturer's standard whole-body scanning protocol. The body was positioned centrally on the scanning table using the scanner centreline as a reference. The arms were placed alongside the trunk with the thumbs facing upwards and palms facing the legs. A small gap (approximately 1 cm) was maintained between the arms and trunk to minimize tissue overlap. Lower limbs were positioned according to standard whole-body DXA procedures.

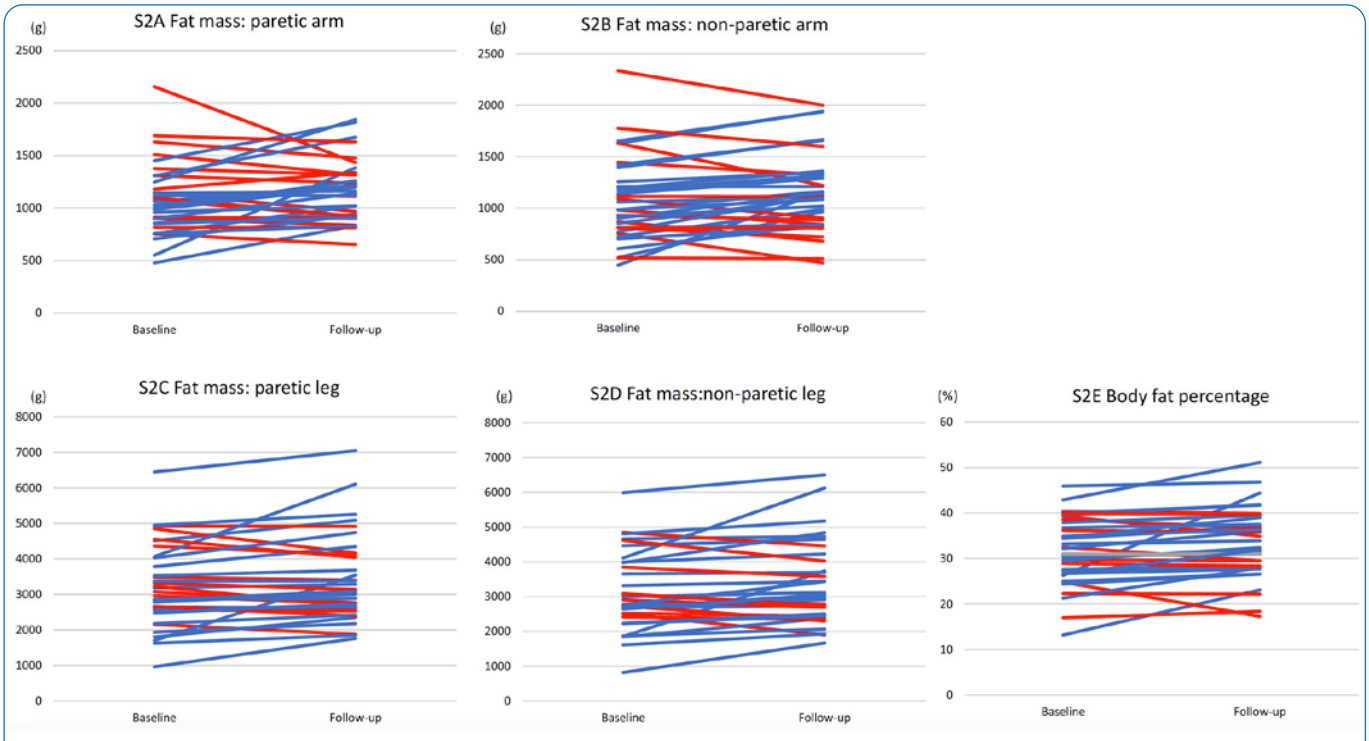
Regional body composition analyses were performed using the manufacturer's predefined segmentation protocol. For arm regions, cut lines were placed through the shoulder joints as close to the trunk as possible. For leg regions, cut lines extended from the pelvic cut through the femoral necks without intersecting the pelvis. Lean mass, fat mass, and bone mineral content were obtained for the whole body and regional limb compartments.

All scans were acquired by certified radiologic technologists with prior experience in DXA-based body composition and bone mineral density assessment. Regions of interest were reviewed during analysis to ensure consistency with standard segmentation procedures.

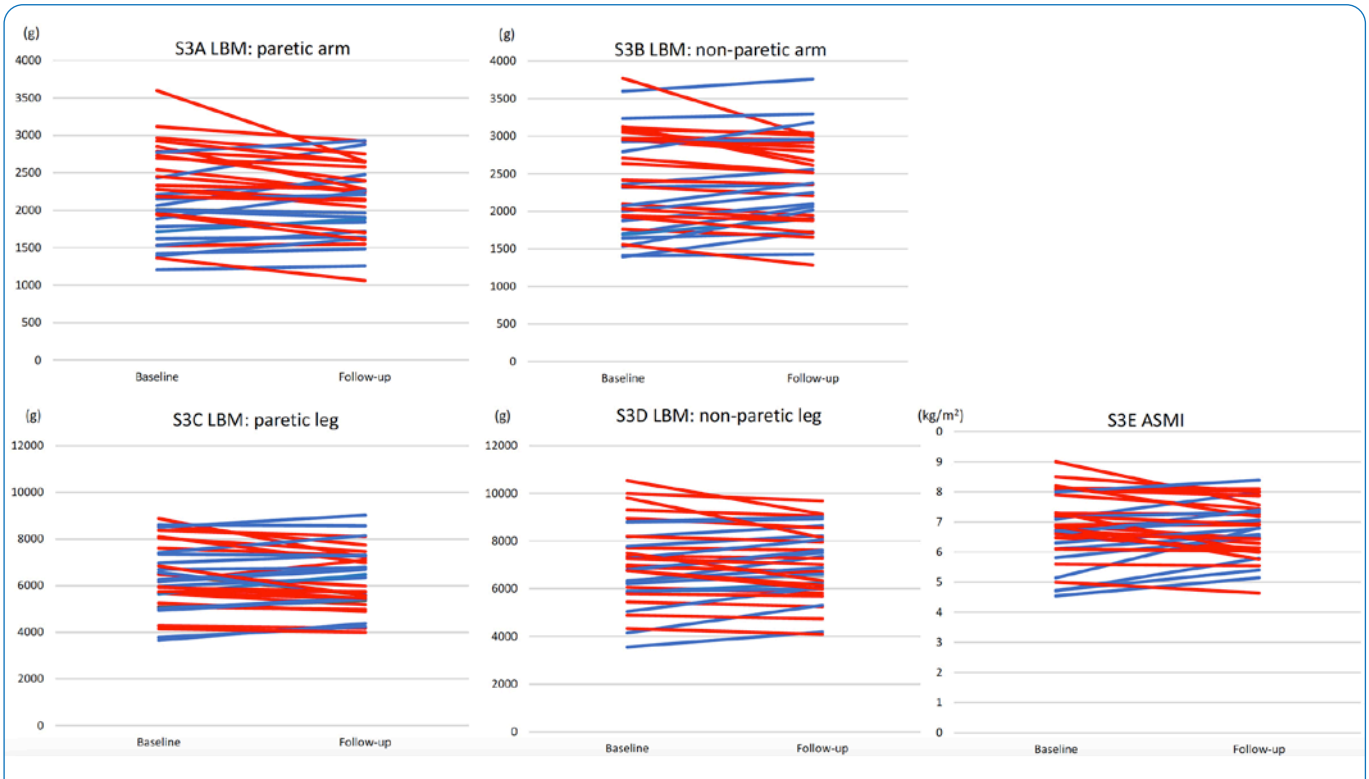
Scanner precision testing demonstrated a coefficient of variation of approximately 1 % and a standard deviation of 0.01 g/cm^2. The least significant change values were 2.2 % for body fat percentage and 0.028 g/cm^2 for total body bone mineral density. Additional precision metrics for lean mass and fat mass are provided according to manufacturer and institutional quality-control records.



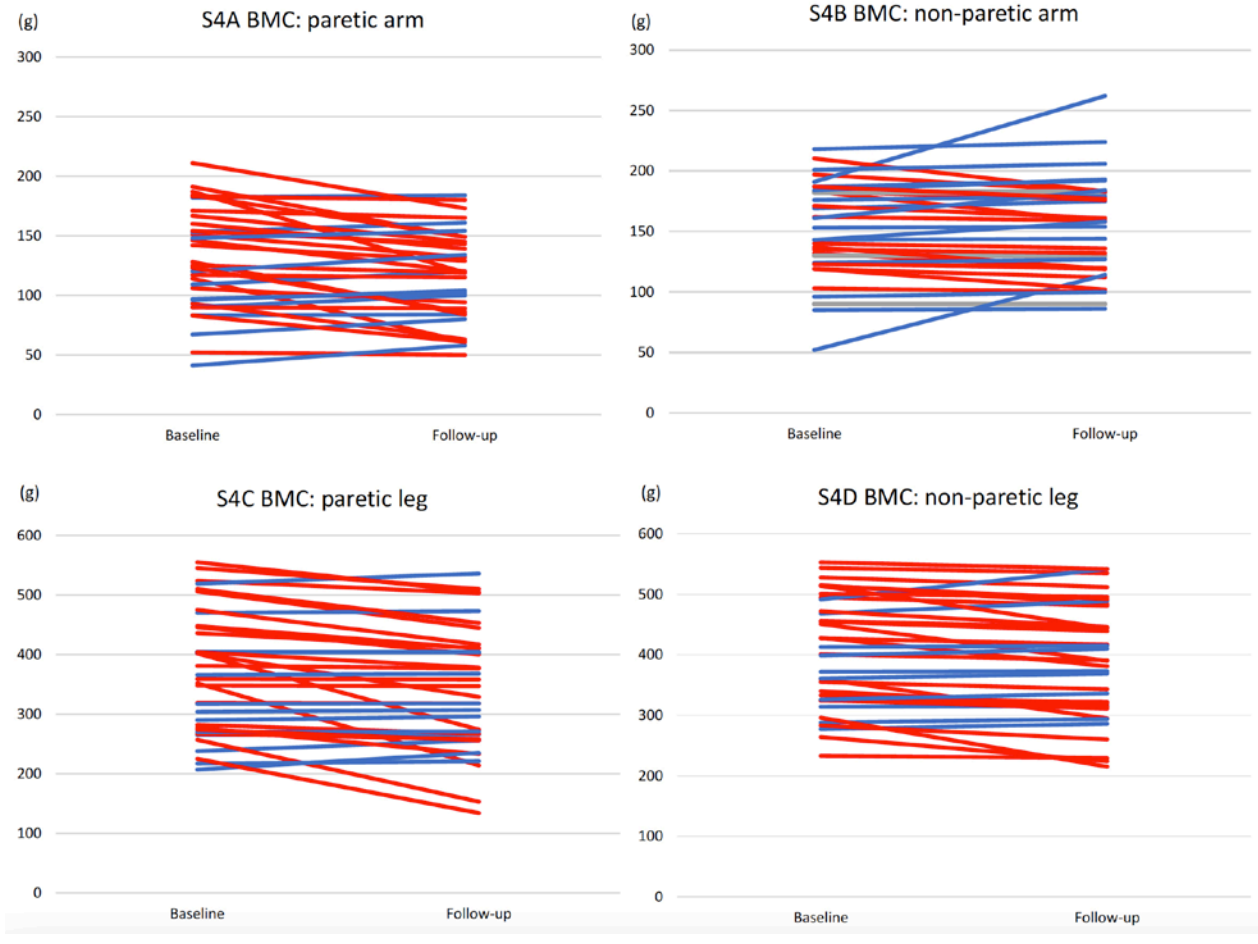
Supplementary Figure S1. Individual changes in comfortable gat speed between baseline and follow-up assessments. Red lines denote decrease; blue lines denote increase.



Supplementary Figure S2. Individual participant baseline and follow-up data for fat mass and body fat percentage. (A) Paretic arm fat mass; (B) non-paretic arm fat mass; (C) paretic leg fat mass; (D) non-paretic leg fat mass; and (E) body fat percentage. Red lines denote decrease; blue lines denote increase; and grey lines represent unchanged values.



Supplementary Figure S3. Individual participant baseline and follow-up data for lean body mass (LBM) and appendicular skeletal muscle mass index (ASMI). (A) Paretic arm LBM; (B) non-paretic arm LBM; (C) paretic leg LBM; (D) non-paretic leg LBM; and (E) ASMI. Red lines denote decrease; blue lines denote increase.



Supplementary Figure S4. Individual participant baseline and follow-up data for bone mineral content (BMC). (A) Paretic arm BMC; (B) non-paretic arm BMC; (C) paretic leg BMC; and (D) non-paretic leg BMC. Red lines denote decrease; blue lines denote increase.

Supplementary Table 1. Baseline demographic and clinical characteristics of participants who completed follow-up versus those lost to follow-up in the study cohort.

Variables	Lost to follow-up (n=49) Mean (SD)	Completed follow-up (n=35) Mean (SD)	p-value
Age, years	57.7 (10.7)	59.1 (7.8)	0.50
Female, n (%)	12 (24.5)	14 (40)	0.13
BMI (kg/m^2)	24.6 (3.7)	23.8 (3.6)	0.32
NIHSS (score), median (P25, P75)	4 (3, 7)	3 (2, 6)	0.15
UE motricity index	48.9 (31.2)	58.1 (26.1)	0.16
LE motricity index	54.1 (28.1)	59.8 (25.6)	0.34
Maximum handgrip strength (kg) (n = 75)	24.6 (9.2) (n = 45)	26.2 (9.7) (n = 30)	0.51
Gait speed (m/s) (n = 69)	0.52 (0.33) (n = 42)	0.60 (0.40) (n = 27)	0.41
ASMI (kg/m^2)	7.0 (1.2)	6.8 (1.1)	0.44
Sarcopenia	16 (32.7)	12 (34.3)	0.88

Abbreviations: ASMI, appendicular skeletal muscle mass index; BMI, body mass index; LE, lower extremity, NIHSS, National Institutes of Health Stroke Scale; P25, 25th percentile, P75, 75th percentile; UE, upper extremity.

Supplementary Table 2. Changes in clinical parameters from baseline to follow-up after excluding cases with recurrent stroke (n=31).

Variables Clinical variables	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
NIHSS (score), median (P25, P75)	4 (2, 8)	3 (2, 6)	NA	0.14*	NA
UE Motricity Index	55.4 (37.5)	57.2 (27.5)	-1.7 (-9.5 to 6.0)	0.65	-0.6 (-2.8 to 1.6)
LE Motricity Index	57.2 (37.0)	60.2 (27.0)	-3.0 (-11.3 to 5.4)	0.47	-0.8 (-3.2 to 1.5)
BW (kg)	65.4 (11.5)	63.3 (12.3)	2.0 (0.3 to 3.7)	0.020	0.5 (0.0 to 1.0)
BMI (kg/m^2)	25.0 (3.2)	24.1 (3.6)	0.9 (0.2 to 1.6)	0.019	0.24 (0.02 to 0.46)
Calf circumference (cm)	34.4 (3.0)	33.7 (3.4)	0.6 (-0.1 to 1.4)	0.099	0.19 (-0.02 to 0.40)
Maximal grip strength (kg) (n=26)	27.0 (8.3)	26.1 (10.1)	0.9 (-1.8 to 3.7)	0.50	0.3 (-0.4 to 1.0)
Five Times Sit-to-Stand test (sec), median (P25, P75) (n=17)	19.4 (15.4, 27.5)	21.3 (13.4, 28.1)	NA	0.012*	NA
Comfortable gait speed (m/s), (n=20)	0.54 (0.31)	0.71 (0.38)	-0.17 (-0.27 to -0.07)	0.002	-0.04 (-0.07 to -0.02)
MoCA (score), median (P25, P75) (n=29)	22 (19, 26)	23 (20, 27)	NA	0.32*	NA
FAC, n (%)			Transition		
0-3	15 (48.4)	17 (54.8)	14 remained in FAC 0-3	0.63†	NA
4-5	16 (51.6)	14 (45.2)	1 improved to FAC 4-5 13 remained in FAC 4-5 and 3 declined to FAC 0-3		
Reported physical activity time in moderate to vigorous intensity (min/week), median (P25, P75)	15 (0, 42.6)	35 (0, 175)	NA	0.011*	NA

*Continuous variables are presented as mean (SD) unless otherwise indicated. Abbreviations: BMI, body mass index; FAC, Functional Ambulation Category; LE, lower extremity; MoCA, Montreal Cognitive Assessment; NIHSS, National Institutes of Health Stroke Scale; P25, 25th percentile, P75, 75th percentile; UE, upper extremity. FTSTS and gait speed were not performed in participants with functional or mobility limitations or refusal. MoCA was missing due to incomplete documentation. Grip strength change analysis excluded participants without baseline measurements. *Wilcoxon signed-rank test, †McNemar's exact test.*

Supplementary Table 3. Changes in fat mass from baseline to follow-up after excluding cases with recurrent stroke (n=31).

Fat mass (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
Upper extremity					
Paretic side	1184.7 (307.8)	1110.8 (357.4)	73.8 (-35.5 to 183.1)	0.18	19.7 (-11.3 to 50.7)
Non-paretic side	1171.1 (393.5)	1101.3 (414.7)	69.8 (-17.7 to 157.3)	0.11	18.4 (-8.6 to 45.3)
Lower extremity					
Paretic side	3383.6 (1262.3)	3290.6 (1182.3)	193.0 (-26.6 to 412.6)	0.083	55.3 (-7.0 to 117.6)
Non-paretic side	3394.9 (1218.6)	3205.3 (1125.9)	189.6 (-34.8 to 414.0)	0.095	54.7 (-9.7 to 119.1)
Total body					
Body fat percentage (%)	33.6 (8.0)	31.9 (7.7)	1.7 (0.1 to 3.4)	0.041	0.49 (-0.04 to 1.03)

Supplementary Table 4. Changes in lean body mass and ASMI from baseline to follow-up after excluding cases with recurrent stroke (n=31).

Lean body mass (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean difference (95 % CI)
Upper extremity					
Paretic side	2200.0 (457.7)	2241.3 (593.5)	-41.3 (-151.2 to 68.5)	0.45	-8.4 (-40.2 to 23.3)
Non-paretic side	2461.5 (543.3)	2479.2 (669.7)	-17.7 (-113.9 to 78.5)	0.71	-4.6 (-32.5 to 23.3)
Lower extremity					
Paretic side	6333.2 (1298.0)	6407.7 (1458.9)	-74.5 (-306.4 to 157.4)	0.52	-23.1 (-89.2 to 42.9)
Non-paretic side	7112.9 (1405.0)	7085.0 (1687.7)	27.9 (-211.6 to 267.3)	0.81	20.8 (-49.0 to 90.6)
Total body					
ASMI (kg/m^2)	6.9 (0.9)	6.9 (1.2)	0.0 (-0.2 to 0.3)	0.95	0.01 (-0.06 to 0.09)
Abbreviations: ASMI, appendicular skeletal muscle mass index.					

Supplementary Table 5. Changes in bone mineral content from baseline to follow-up after excluding cases with recurrent stroke (n=31).

Bone mineral content (BMC) (g)	Follow-up Mean (SD)	Baseline Mean (SD)	Mean differences (95 % CI)	p-value	Annualized mean differences (95 % CI)
Upper extremity					
Paretic side	118.1 (38.1)	130.3 (44.6)	-12.1 (-20.4 to -3.8)	0.006	-3.7 (-6.2 to -1.2)
Non-paretic side	153.5 (41.1)	152.4 (39.7)	1.0 (-6.3 to 8.2)	0.78	0.03 (-1.98 to 2.04)
Lower extremity					
Paretic side	346.0 (102.3)	372.2 (104.4)	-26.2 (-41.3 to -11.2)	0.001	-8.7 (-14.2 to -3.2)
Non-paretic side	390.9 (93.6)	405.1 (91.0)	-14.2 (-24.3 to -4.2)	0.007	-4.6 (-7.9 to -1.3)