Research Square

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High Early Burden of Post-Stroke Spasticity and Persistent Treatment Gaps in Structured Follow-up Care

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Abstract

Background

Post-stroke spasticity (PSS) is a frequent and disabling complication after stroke. Real-world data on the implementation of guideline-recommended antispastic treatment within structured follow-up programs are limited.

Methods

In this prospective cohort study, patients with persistent motor deficits after stroke were followed within a structured post-stroke care program. Spasticity was assessed using the Modified Ashworth Scale (MAS) at predefined follow-up intervals (3 and 12 months after stroke onset). Clinically relevant spasticity was defined as MAS ≥ 2 in at least one joint. Antispastic treatments, including botulinum toxin type A (BoNT-A), were recorded. The implementation gap for BoNT-A was defined as the proportion of patients with a treatment recommendation who had not initiated therapy.

Results

Using a cumulative 12-month MAS-based definition among patients with persistent paresis and available follow-up assessments, clinically relevant spasticity was observed in 40/55 patients (72.7%, 95%CI 59-83.9), while 51/55 patients (92.7%, 95%CI 82.4–97.9) showed any increase in muscle tone (MAS > 0). Clinically relevant spasticity was present in 34/53 patients at 3 months (64.2%, 95%CI 49.8–76.9) and in 26/46 patients at 12 months (56.5%, 95%CI 41.1–71.1). Among patients with clinically relevant spasticity, BoNT-A treatment was recommended in 11.8% at 3 months and 26.9% at 12 months, while no patient with a documented recommendation had initiated therapy.

Conclusions

PSS occurs early and affects a substantial proportion of patients with persistent motor deficits after stroke. Despite structured follow-up and systematic screening, implementation of guideline-recommended BoNT-A therapy remained absent in patients with documented treatment recommendations, indicating substantial barriers to evidence-based outpatient spasticity care.

Introduction

Stroke is a major global health burden and a leading cause of long-term disability in adults [1]. Advances in acute stroke care have improved survival, resulting in a growing number of patients living with persistent neurological deficits. One of the most frequent and disabling motor complications is post-stroke spasticity (PSS), which negatively affects functional recovery, quality of life and healthcare costs.

Approximately 25–40% of stroke survivors develop clinically relevant spasticity within the first year after stroke [2, 3].

PSS is characterized by increased muscle tone and involuntary muscle activation following upper motor neuron injury [4–6]. Clinically, PSS may lead to pain, contractures, impaired mobility and secondary musculoskeletal complications, thereby substantially contributing to long-term disability after stroke.

Several treatment options for PSS are available, including physiotherapy, occupational therapy, oral antispastic medication and intramuscular botulinum toxin type A (BoNT-A). In particular, BoNT-A is recommended as a first-line treatment for focal PSS in current guidelines and consensus statements and has been shown to reduce muscle overactivity, improve limb function, reduce pain and facilitate care [5, 6]. Despite these recommendations, real-world utilization of BoNT-A therapy remains low and many patients with clinically relevant spasticity do not receive evidence-based treatment [7, 8]. Limited outpatient screening, delayed recognition, restricted access to specialized treatment and fragmented referral pathways may all contribute to this treatment gap.

Structured post-stroke follow-up programs may provide an opportunity to systematically identify patients with spasticity and unmet treatment needs. However, whether treatment recommendations are subsequently translated into evidence-based outpatient care remains unclear. The aim of this prospective study was therefore to assess the occurrence of PSS and the real-world implementation of antispastic treatments within a structured post-stroke follow-up program.

Methods

Study design and population

This prospective study was conducted within the SOS-Care program, a structured post-stroke follow-up program as previously described [9]. The program provides coordinated outpatient care for patients after discharge to home or rehabilitation over a 12-month period and is delivered by trained stroke case managers. Eligible patients included those hospitalized for ischemic stroke or intracerebral hemorrhage. Patients with pre-stroke functional dependence, severe cognitive impairment or receiving palliative care were not eligible for participation in the SOS-Care program. For the present study, systematic spasticity screening was integrated into the follow-up program. Patients with motor deficits at hospital discharge, defined as ≥ 1 point in the motor items of the National Institutes of Health Stroke Scale (NIHSS) score were considered at risk for PSS and prospectively followed. Spasticity screening and treatment recommendations were performed by trained case managers with a physiotherapy background. The SOS-Care program itself did not include initiation or delivery of antispastic treatments. Treatment implementation including initiation of BoNT-A therapy, remained part of routine outpatient care and was carried out by treating physicians outside the program, typically involving general practitioners and office-based neurologists. The screening algorithm is shown in Fig. 1A.

Baseline variables included demographic characteristics, stroke type (ischemic stroke or intracerebral hemorrhage), stroke severity and disability, including NIHSS and modified Rankin Scale (mRS) scores at discharge, vascular risk factors and acute stroke therapies.

The study was approved by the local ethics committee and all participants provided informed consent (EK-214062013). The study was conducted and reported in accordance with the STROBE guidelines [10].

Follow-up assessments

Outcome assessments were scheduled around predefined 3-month and 12-month follow-up visits (Fig. 1A). Assessments were assigned according to the time elapsed since stroke onset. If multiple assessments were available within a given follow-up window, the assessment closest to the target time point was used. At 3 months, the patient-reported Zorowitz Spasticity Screening Tool was administered to the overall at-risk cohort as part of an early screening approach for clinically relevant PSS. In addition, Modified Ashworth Scale (MAS)-based clinical spasticity assessments were performed in patients with persistent motor deficits. At 12 months, follow-up assessments were restricted to patients with persistent motor deficits and included repeated MAS assessments as well as a reassessment of patient-reported spasticity-related functional impairment using the Zorowitz questionnaire. Patients without residual paresis during follow-up were generally not subjected to further structured spasticity-specific assessments but remained in the general SOS-Care follow-up program.

Outcome definitions

Clinical spasticity was assessed during in-person home visits using the MAS. Assessments included predefined upper- and lower-limb joint movements across 12 joint regions involving the shoulder, elbow, wrist, hip, knee and ankle, representing muscle groups commonly affected by PSS (Fig. 1B). The primary outcome was clinically relevant PSS, defined as a MAS score ≥ 2 in at least one joint. Secondary MAS-based outcomes included MAS ≥ 2 in at least two joints and any increase in muscle tone (MAS > 0). An additional patient-reported functional outcome was assessed using the Zorowitz questionnaire, with any total score > 0 indicating spasticity-related functional impairment [11]. For cumulative analyses, patients were classified as having developed spasticity if the respective criterion was met at any assessment during first-year follow-up.

In addition to clinical spasticity outcomes, patterns of antispastic treatment were assessed, including physiotherapy, occupational therapy, oral antispastic medication and BoNT-A therapy. Treatments were recorded at each follow-up assessment. The implementation gap for BoNT-A therapy was defined as the proportion of patients with a documented treatment recommendation who had not initiated therapy.

Statistical analysis

Continuous variables are presented as mean ± standard deviation (SD) or median with interquartile range (IQR), as appropriate, and categorical variables as counts and percentages. Exact binomial 95% confidence intervals (95% CIs) were calculated for key proportions. Multivariable logistic regression models were used to identify factors associated with clinically relevant PSS within 12 months. The primary model included age, sex, stroke type (intracerebral hemorrhage vs. ischemic stroke) and mRS score at discharge as prespecified covariates. In an alternative model, NIHSS score at discharge was entered instead of mRS. Odds ratios (ORs) with 95%CIs were reported and robust standard errors were applied. Correlation between Zorowitz total score and MAS sum score at 3 months was assessed using Pearson correlation analysis. A two-sided p-value < 0.05 was considered statistically significant. No imputation of missing data was performed and analyses were based on available cases. All analyses were performed using Stata version 16 (StataCorp, College Station, TX, USA).

Results

Study population

Between July 2022 and December 2024, 151 patients participating in SOS-Care were screened for inclusion in the PSS follow-up program. Of these, 149 patients (98.7%) had a documented motor deficit at discharge and were therefore considered at risk for the development of PSS, while 2 patients (1.3%) had no residual paresis. Among the 149 at-risk patients, 75 individuals (50.3%) declined participation in the follow-up program. Consequently, 74 patients (49.7%) were enrolled and constituted the final study population for longitudinal follow-up (Fig. 2). Baseline characteristics are summarized in Table 1.

Table 1
Baseline characteristics of the study population

Variable	Study population (n = 74)
Demographics	
Age, years, median (IQR)	67 (18)
Female, n (%)	25 (33.8)
Risk factors, n (%)	
Arterial Hypertension	65 (87.8)
Diabetes mellitus	23 (31.1)
Nicotine abuse	15 (20.3)
Hyperlipidemia	68 (91.9)
Previous stroke/TIA	17 (23.0)
Atrial fibrillation	19 (25.7)
Sleep apnea	5 (6.8)
Stroke-related data	
NIHSS, median (IQR)	
Total NIHSS score	12 (11)
Motor items of NIHSS	5 (5)
mRS, median (IQR)	5 (1)
Intracerebral Hemorrhage, n (%)	12 (16.2)
Ischemic stroke, n (%)	62 (83.8)
Acute therapy for ischemic stroke	
Intravenous therapy, n (%)	20 (32.3)
DTN time, median (min)	31 (27)
Endovascular therapy, n (%)	21 (33.9)
DTG time, median (min)	56 (43)
TICI Scale Grade 0, n (%)	3 (14.3)
TICI Scale Grade 2b-3, n (%)	17 (81.0)
Combinatory treatment, n (%)	10 (16.1)
Ischemic stroke etiology, TOAST-classification, n (%)	

Variable	Study population (n = 74)
Large-artery atherosclerosis	20 (32.3)
Cardio embolism	17 (27.4)
Small-vessel occlusion	5 (8.1)
Stroke of other determined etiology	2 (3.2)
Stroke of undetermined etiology	7 (11.3)
ESUS, n (%)	9 (14.5)

Prevalence and temporal evolution of post-stroke spasticity

Using the cumulative 12-month MAS-based definition among patients with persistent paresis and available MAS follow-up assessments, clinically relevant spasticity (MAS ≥ 2 in at least one joint) was observed in 40 of 55 patients (72.7%, 95%CI 59-83.9), while involvement of at least two joints was present in 30 of 55 patients (54.5%, 95%CI 40.6–68). When applying a more sensitive definition of any increase in muscle tone (MAS > 0), spasticity was observed in 51 of 55 patients (92.7%, 95%CI 82.4–97.9).

At 3 months, persistent paresis with corresponding MAS follow-up assessment was present in 53 patients and at 12 months in 46 patients. Clinically relevant spasticity (MAS ≥ 2) was present in 34 of 53 patients at 3 months (64.2%, 95%CI 49.8–76.9) and in 26 of 46 patients at 12 months (56.5%, 95%CI 41.1–71.1). Moderate-to-severe spasticity involving at least two joints was present in 26 of 53 patients at 3 months (49.1%, 95%CI 35.1–63.2) and in 17 of 46 patients at 12 months (37%, 95%CI 23.2–52.5). When considering any increase in muscle tone (MAS > 0), proportions were 48 of 53 patients at 3 months (90.6%, 95%CI 79.3–96.9) and 42 of 46 patients at 12 months (91.3%, 95%CI 79.2–97.6).

Using the cumulative 12-month Zorowitz-based definition, spasticity-related functional impairment was observed in 55 of 72 patients (76.4%, 95%CI 64.9–85.6). Functional impairment was present in 36 of 70 patients at 3 months (51.4%, 95%CI 39.2–63.6) and in 39 of 49 patients at 12 months (79.6%, 95%CI 65.7–89.8). Among patients with both Zorowitz and MAS data available at 3 months, the correlation between Zorowitz total score and MAS sum score was weak and not statistically significant ($r = 0.18$; $p = 0.20$).

Predictors of spasticity within 12 months

In multivariable logistic regression analysis using cumulative MAS-defined spasticity (MAS ≥ 2 in at least one joint) as the dependent variable, disability at discharge emerged as the only independent predictor of clinically relevant spasticity. Each one-point increase in mRS was associated with a 2.62-fold increase in the odds of developing clinically relevant spasticity within 12 months (OR 2.62, 95%CI 1.45–4.75; p =

0.001). No significant associations were observed for age (OR 1.01, 95%CI 0.96–1.07; p = 0.61), female sex (OR 1.15, 95%CI 0.25–5.35; p = 0.86) or intracerebral hemorrhage (OR 0.26, 95%CI 0.05–1.30; p = 0.10). In an alternative model including NIHSS at discharge instead of mRS, NIHSS was not independently associated with spasticity (OR 1.07 per point, 95%CI 0.94–1.21; p = 0.31).

Antispastic treatment and implementation gap

Among patients with clinically relevant spasticity (MAS ≥ 2) at 3 months (n = 34), BoNT-A treatment was recommended in 4 patients (11.8%; 95%CI 3.3–27.5) and none had initiated therapy. At 12 months, among 26 patients with clinically relevant spasticity, BoNT-A treatment was recommended in 7 patients (26.9%; 95% CI 11.6–47.8) and none had initiated therapy.

Oral antispastic medication was documented in 2 of 34 patients with clinically relevant spasticity at 3 months (5.9%; 95%CI 0.7–19.7) and in 7 of 26 patients at 12 months (26.9%; 95%CI 11.6–47.8). At 12 months, physiotherapy and occupational therapy were documented in 25 of 26 patients (96.2%; 95%CI 80.4–99.9) and 21 of 26 patients (80.8%; 95%CI 60.6–93.4), respectively.

Discussion

This prospective study provides novel real-world insights into the burden and outpatient management of PSS within a structured post-stroke follow-up program: (1) clinically relevant PSS occurred early and affected a substantial proportion of patients with persistent motor deficits, and (2) despite systematic screening and structured follow-up care, implementation of guideline-recommended BoNT-A therapy remained nearly absent. Notably, a high proportion of patients already exhibited moderate-to-severe spasticity within the early follow-up phase, while evidence-based focal spasticity treatment was rarely implemented despite documented treatment recommendations.

Reported prevalence rates of PSS vary widely depending on diagnostic definitions, study populations and timing of assessment [2]. Meta-analyses suggest that approximately one-third of unselected stroke survivors develop spasticity within the first months after stroke [2, 12]. In patients with paresis after a first stroke, an incidence of 39.5% has been reported, with some studies showing rates as high as 46% [3]. The higher cumulative prevalence observed in the present study likely reflects the predefined inclusion of patients with persistent motor deficits, representing a population at increased risk. In line with previous prospective studies demonstrating early development of PSS after stroke [13], a substantial proportion of patients in our cohort already exhibited clinically relevant spasticity within the 3-month follow-up window. The persistently high prevalence at 12 months further suggests that, once established, PSS often persists and requires ongoing management.

The most relevant finding of this study was the near-complete absence of BoNT-A implementation despite documented treatment recommendations. Although treatment was recommended in a subset of patients with clinically relevant spasticity, none of the patients initiated therapy during follow-up. Notably, this treatment gap persisted even within a structured follow-up program with active case management

and systematic spasticity screening, suggesting that barriers extend beyond under-recognition alone. While previous real-world studies have reported low utilization rates of BoNT-A therapy in routine care [14, 15], our findings extend these observations by demonstrating that even structured identification of treatment needs does not necessarily translate into treatment implementation. Several factors likely contribute to this gap, including limited availability of experienced injectors or specialized outpatient services, administrative barriers, uncertainty regarding referral pathways and fragmentation of care across multiple providers [12, 16–21]. In addition, stroke survivors are frequently transferred between rehabilitation facilities, primary care physicians, office-based neurologists, therapists and caregivers, increasing the risk of delayed or lost treatment recommendations. Our findings suggest that improving detection alone may be insufficient and needs to be complemented by structured referral pathways and accessible specialist treatment.

In line with previous studies, functional impairment emerged as the main predictor of PSS in our analysis. Meta-analytic data identified moderate to severe paresis as the strongest predictor, while sensory deficits showed weaker and time-dependent associations, reaching significance mainly at 3 to 6 months [3]. In contrast to some previous studies, we did not observe an association between hemorrhagic stroke and PSS. However, only a minority of patients in our cohort had intracerebral hemorrhage as stroke etiology. Previous studies have further identified early increased muscle tone, greater severity of paresis, hemihypesthesia and low Barthel Index score as predictors of early PSS development [22]. In this context, mRS at discharge may act as a global surrogate capturing the overall severity of neurological and functional impairment.

Beyond spasticity management, our findings may also inform the broader concept of structured post-stroke care. Previous analyses have shown that case management-based follow-up is associated with improved vascular risk factor control and reduced rates of recurrent vascular events [9]. Our findings suggest that structured follow-up programs may effectively identify unmet needs and optimize secondary prevention, but additional integration of specialized outpatient services may be required to translate treatment recommendations into effective care for disabling long-term stroke sequelae such as PSS.

This study has several limitations. First, the single-center design may limit generalizability. Second, inclusion was restricted to patients participating in a structured follow-up program, which may introduce selection bias. Third, missing data at different time points and the restriction of MAS-based assessments primarily to patients with persistent motor deficits may have influenced prevalence estimates. In addition, clinical spasticity assessment relied primarily on the MAS, whereas comprehensive evaluation of PSS may also require integration of functional impairment, activity limitations, quality of life and patient-reported outcomes [8]. Strengths of this study include its prospective design, systematic longitudinal screening for spasticity and detailed characterization of real-world treatment patterns within a structured post-stroke care program. These features provide clinically relevant insights into both the burden of PSS and current barriers to evidence-based outpatient management after hospital discharge.

Conclusions

This study demonstrated a high burden of PSS among patients with persistent motor deficits and reveals a substantial gap between recognition and implementation of guideline-recommended BoNT-A therapy, even within a structured follow-up program. These findings suggest that modern post-stroke care should not be limited to recurrent event prevention and incorporate structured pathways for detection, referral and treatment of disabling long-term sequelae such as spasticity.

Abbreviations

PSS
Post-stroke spasticity
MAS
Modified Ashworth Scale
BoNT-A
botulinum toxin type A
CI
Confidence Interval
SOS-Care
“Schlaganfallversorgung Ostsachsen“ (translated:Stroke Care in Eastern Saxony)
NIHSS
National Institutes of Health Stroke Scale
mRS
modified Rankin Scale
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology
SD
Standard deviation
OR
Odds Ratio
IQR
Interquartile Range
n
number
DTN time
Door-to-Needle time
DTG time
Door-to-Groin time
TICI
Thrombolysis in Cerebral Infarction (classification)
TOAST

Trial of Org 10172 in Acute Stroke Treatment (classification)
ESUS
Embolic Stroke of Undetermined Source

Declarations

Availability of data

Upon reasonable request by a qualified investigator, anonymised datasets not included in the article may be shared by the corresponding author.

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Author information

Contributions

Conceptualization: TS, JB, KB; Investigation: IS, SW, NP, UH, LF; Data curation: IS, DS, NP; Formal analysis: IS, KB; Supervision: TS, JB, KB; Writing - original draft: IS, DS, KB; Writing - review & editing: EA, NP, UH, LF, SW, TS, JB, HBH, KB.

All authors read and approved the final manuscript.

Ethics declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the TUD Dresden University of Technology (approval number EK-214062013). All participants provided written informed consent. The study was conducted in accordance with the guidelines and regulations of the approving ethics committee and the principles of the Declaration of Helsinki and was reported in accordance with the STROBE guidelines [10].

Consent for publication

Not applicable.

Conflict of Interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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Figures

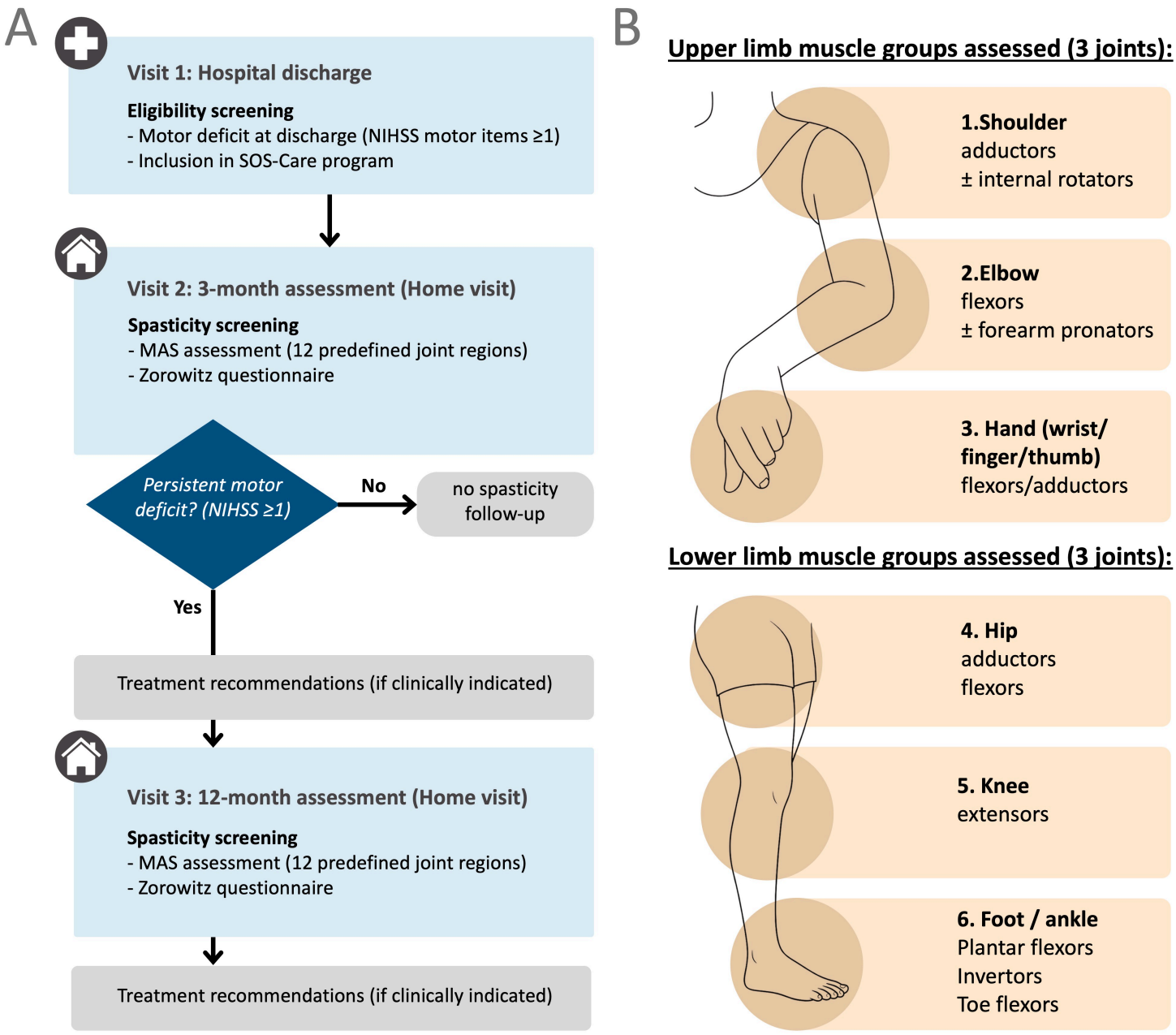


Figure 1

A: Screening algorithm, B: Joint movements assessed by MAS and corresponding muscle groups commonly affected by post-stroke spasticity

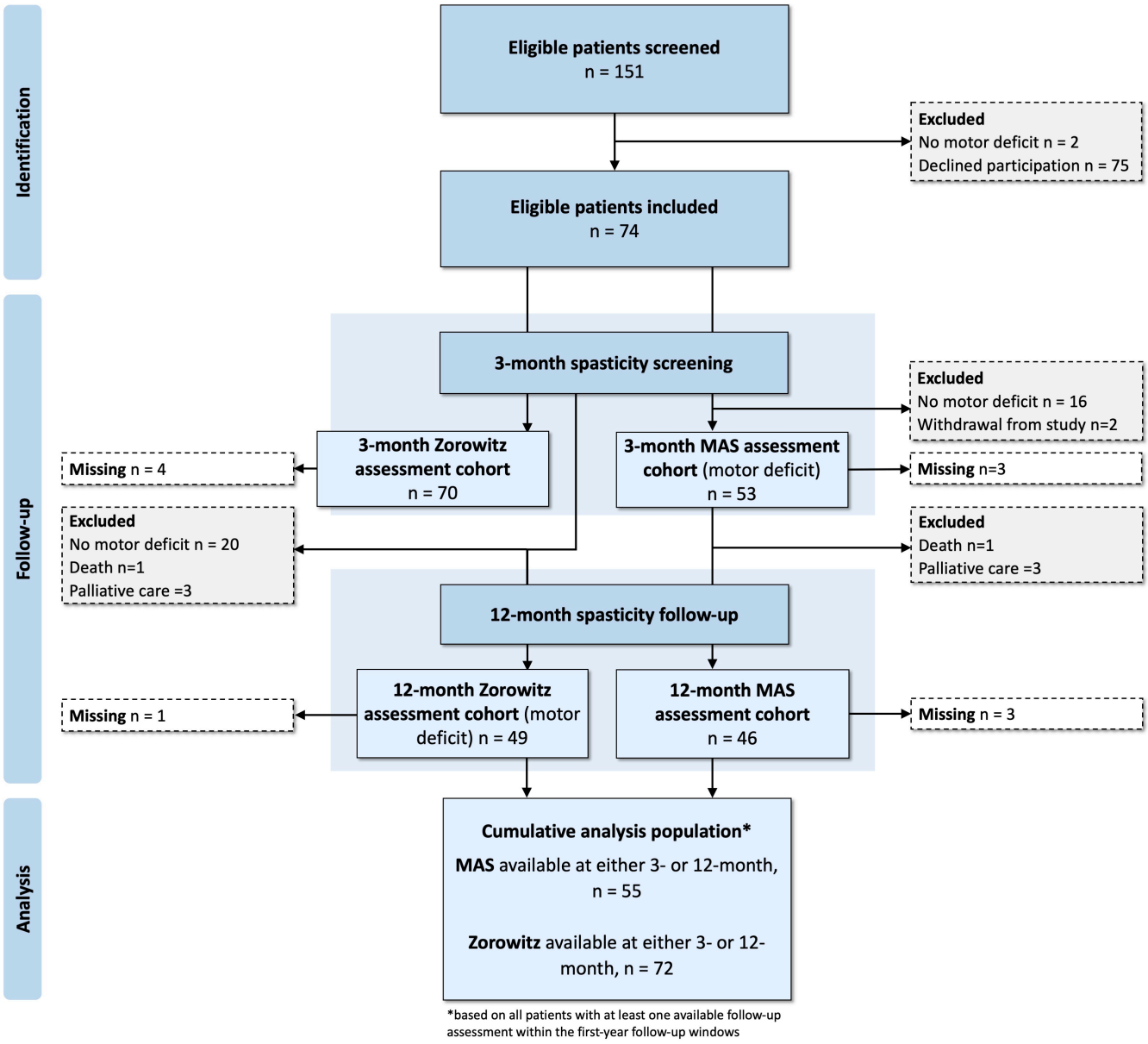


Figure 2

STROBE flow chart showing the development of the patient population over the 12-month follow-up

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- PSSSTROBEchecklist.docx