Medical Rehabilitation (Med Rehabil) 2023, 27 (4): 15-25 DOI: 10.5604/01.3001.0054.0117 eISSN 1896–3250

Pain and Sleep Disturbances are Associated with Post-stroke Anger Proneness and Emotional Incontinence

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Keywords
stroke; sleep quality; post-stroke headache; emotional disorders; post-stroke depression

Abstract
Background: Emotional disorders are common findings after a stroke episode. Despite evidence linking pain and sleep disorders to various post-stroke emotional conditions, their roles in the prevalence of post-stroke anger proneness (PSAP) and post-stroke emotional incontinence (PSEI) remain unclear. We investigated the influence of composite and different components of post-stroke pain (PSP) as well as post-stroke sleep disorders (PSSD) on PSAP and PSEI.
Methods: Cross-sectional data on PSAP, PSEI, PSP and PSSD were evaluated through validated instruments and structured interviews for a total of 185 community-dwelling stroke survivors attending two Nigerian tertiary health facilities. Data on potential confounding variables were also assessed.
Results: The rates of PSSD, PSP, PSAP and PSEI among Nigerian stroke survivors were 36.8%, 63.3%, 23.2% and 44.9%, respectively. The results of logistic regression models showed that composite PSP was associated with PSEI (adjusted odd ratio (aOR): 0.492; 95% confidence interval (CI): 0.251-0.965). While assessing the different components of PSSD and PSP, the results showed that sleep disturbances (aOR: 1.855; 95% CI: 1.096-3.140) and post-stroke headache (aOR: 0.364; 95% CI: 0.153-0.864) were associated with PSEI. In addition, being a domain of PSP, post-stroke headache was associated with PSAP (aOR: 0.052; 95% CI: 0.011-0.238).
Conclusion: There is high prevalence of PSSD, PSP, PSAP and PSEI among Nigerian stroke survivors. Post-stroke headache is associated with both PSAP and PSEI, while sleep disturbances are associated with PSEI. Post-stroke headache and sleep disturbances are potential targets for interventions in patients with stroke to lessen the burden of PSAP and PSEI.

INTRODUCTION

Stroke, a major cause of disability globally, often results in emotional health disorders[1]. The common emotional health disorders observed among stroke survivors include post-stroke depression (PSD), post-stroke anxiety (PSA), post-stroke fatigue (PSF), post-stroke anger proneness (PSAP) and post-stroke emotional incontinence (PSEI)[2]. Among these emotional disorders, PSD, PSA and PSF have been most investigated unlike PSAP and PSEI, whereas the occurrence of PSAP and PSEI is said to be high and equally detrimental to stroke survivors[3-8]. Some of the reported symptoms of PSAP are impulsive behaviours, aggression, irritability and hostility, while those of PSEI include excessive, inappropriate and uncontrollable display of emotions (e.g. laughing or crying) without apparent reason or stimuli to evoke such feelings[4,5]. The presence of PSAP and PSEI is said to be debilitating to the stroke survivors and caregivers alike. Some of the reported negative effects of PSAP and PSEI include low quality of life, poor rehabilitation outcomes, increasing burden on caregivers, distress and embarrassment to patients, families, friends and caregives or carers[9-14].

Neurological, socio-ecological and genetic factors are known to be associated with PSAP and PSEI, however, the evidence is still inconclusive[15,16]. Meanwhile, it appears that post-stroke sleep disorders (PSSD) and post-stroke pain (PSP) are predictors of many psychological disorders fol-

The individual division of this paper was as follows: A – research work project; B – data collection; C – statistical analysis; D – data interpretation; E – manuscript compilation; F – publication search

Article received: 08 Oct. 2022 / Accepted: 19 Nov. 2023

Please cite as: Ademoyegun A.B., Rasaq W.A., Adelowokan O.I., Afolabi V.A., Ibitoye A.G., Awotidebe T.O., Mbada Ch.M. Pain and Sleep Disturbances are Associated with Post-stroke Anger Proneness and Emotional Incontinence. Med Rehabil 2023; 27(4): 15-25. DOI: 10.5604/01.3001.0054.0117

Internet version (original): www.rehmed.pl

lowing stroke, as it has been shown in several studies that both sleep disorders and pain are consistent and independent factors associated with PSD, PSA and PSF[2,17-33]. Nonetheless, to our knowledge, there are no studies to date in which the roles of PSSD and PSP on PSAP and PSEI have been investigated.

Similar to PSAP and PSEI, post-stroke sleep disorders (PSSD) are highly prevalent among stroke survivors[34,35]. Stroke is a known cause of sleep disorders, and in fact, stroke has been reported to worsen underlying sleep disorders if present pre-stroke[36]. Evidence has shown that more than half of stroke survivors often present with PSSD[37], while the results of a meta-analysis investigating the polysomnographic characteristics of sleep in stroke revealed that stroke survivors have poorer sleep quality than the controls[38]. Some of the traits of sleep-related issues that have been reported among stroke survivors include sleep apnea, nighttime sleep disturbances, excessive daytime sleepiness and fatigue[34,39,40]. Alike PSSD, PSP is a debilitating co-morbidity of stroke and it is reportedly common among stroke survivors with data showing that about 11-66% of stroke survivors have stroke-related pain[26]. Various types of PSP identified in the literature include headache, shoulder pain, pain as a result of muscle stiffness, spasm, complex regional pain syndrome and central PSP[41-43], which can manifest singularly or in combination at acute or chronic stage after stroke, even up to 5 years post-stroke[32,33].

Apparently, factors associated with mental health disorders in stroke survivors tend to overlap and all these emotional disorders appear to pathophysiologically interconnect[2]. In fact, it has been shown that antidepressant usage reduces not only symptoms of depression but also aggression and emotional incontinence in a stroke cohort[44], while the presence of sleep disturbances is consistent with the incidences and prevalence of post-stroke depression, anxiety and fatigue[2]. Furthermore, reports have emerged in which it has been stated that treatment of sleep disorders and pain after stroke improves quality of life and reduces mortality, morbidity and emotional disorders among stroke survivors[30-33,37].

Consequent to the foregoing, it is important to investigate the impact of sleep disorders and pain on the high prevalence of emotional incontinence and aggression in stroke survivors since they both appear to show consistent patterns of association with other psychological disorders in stroke survivors. Understanding the effects of sleep disorder and pain on aggression as well as emotional incontinence in stroke may help deepen the knowledge and management protocols of these emotional disorders. Meanwhile, pain after stroke is reported to present with different characteristics having varying and overlapping etiological mechanisms[30,32,33,45-47]. Furthermore, the assessment of sleep disorders as composite variables has been reported to be misleading[48], while the dearth of data on the specific link between different components of sleep disorders and stroke outcome may hamper its robust understanding[49]. Thus, in this study, the prevalence and correlates of PSSD, PSP, PSAP and PSEI were examined among Nigerian stroke survivors, and the impact of PSSD and PSP as composite variables was investigated, as well as the different components of PSSD (sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime sleep dysfunction) and PSP (post-stroke headache, central post-stroke pain, peripheral post-stroke pain, other pain, and pain severity) on PSAP and PSEI.

METHODS

Participants

The participants for this cross-sectional observational study, recruited from February 2021 to July 2022, were community-dwelling stroke survivors who were receiving treatment at the Physiotherapy Department, Osun State University Teaching Hospital and State Specialist Hospital, Osogbo, Nigeria. Stroke survivors aged 18 years and older, with first-ever stroke and who had a stroke diagnosis confirmed by computed tomography or magnetic resonance imaging examination of the head were included in the study. Exclusion criteria were severe aphasia or dysarthria preventing reliable interview, the presence of other neurological ailment apart from stroke, e.g. Parkinson disease, current use of antidepressants or any other psychiatric medications, having ≤ 23 scores for the Mini Mental State Examination, and those who came to the clinics alone preventing easy access to information from caregivers to confirm the presence or absence of PSAP and PSEI [8]. By using the sample size formula for cross-sectional epidemiological studies proposed by Kasiulevicius et al.[50], with a 95% confidence interval, 0.05 precision level and 10% stroke prevalence, a minimum of 125 participants were required for this study. However, a total of 185 stroke survivors participated in this study. The Ethics Review Committee of the Osun State University Teaching Hospital, Osogbo, Nigeria gave approval for the study (LTH/EC/2021/01/496). Also, written informed consent was obtained from each participant before commencement of the study.

Measures

Measurement of post-stroke anger proneness and emotional incontinence

Post-stroke anger proneness (PSAP) and post-stroke emotional incontinence (PSEI) were examined through interviews with stroke survivors and their caregivers/relatives, as well as the use of appropriate outcome measures. The Spielberger Trait Anger Scale (STAS) was implemented to assess PSAP of the participants. The subjects were considered as being prone to anger if the post-stroke aggregate score on the STAS was higher than that for pre-stroke. The STAS is a 10-item instrument eliciting information on aggression and anger proneness. Each of the questions from the STAS was answered on a numerical scale 1 (almost never), 2 (sometimes), 3 (often) and 4

(almost always) with the aggregate scores indicating pre- and post-stroke anger status[8,51]. In addition, the caregiver/relative living with each participant must agree that the stroke survivors developed anger proneness after stroke onset for it to be considered positive in any participant[5]. Furthermore, the participants were considered positive with PSEI if the stroke survivors had exhibited excessive or inappropriate laughing, crying or both more than in pre-stroke states. The patients must have exhibited this behaviour at least twice in the past month as confirmed by the caregiver/relatives of the stroke survivors[4,52]. The assessment of PSAP and PSEI was undertaken the same day by one of the researchers (ABA) who had more than a decade of experience in stroke rehabilitation and was blinded to the other measures.

Measurement of post-stroke sleep disorders and post-stroke pain

The post-stroke sleep disorders (PSSD) of the participants were evaluated by the Pittsburgh Sleep Quality Index (PSQI). The PSQI is an instrument consisting of 19 items assessing subjective sleep quality for the previous month[53]. The PSQI items are scored on a 4-point scale from 0 to 3. Each of the seven components of the PSQI (sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime sleep dysfunction) was calculated based on specific scoring keys and were summed to achieve a global PSQI score. The cumulative PSQI score ranges from 0 to 21. A higher PSQI global and component score indicates poorer sleep quality. A global PSQI score of <7 was considered good sleep quality while PSQI scores of ≥ 7 were assumed as poor sleep quality or sleep disorder[54].

The post-stroke pain (PSP) of the participants was evaluated by a proprietary questionnaire/interview requiring information on whether the participants experience pain after the stroke episode. The types of PSP evaluated in this study were central post-stroke pain (CPSP), peripheral post-stroke pain (PPSP), post-stroke headache (PSH) and other pain. Stroke survivors, with the presence of at least 4 out of 10 central pain symptoms (pins and needles, numbness, itching, burning, painful cold, electric shocks, tingling, touch hypoesthesia, allodynia, and pinprick hypoesthesia), assessed using the Douleur Neuropathique 4 questionnaire, were considered as having CPSP[31,55,56]. The PPSP was confirmed positive in the presence of any of hemiplegic shoulder pain, pain due to spasticity or muscle spasm/stiffness and complex regional pain syndrome[46]. The presence and confirmation of headache after stroke onset at the time of interview was considered as PSH, while pain in the back, neck, abdomen or chest was categorised as other pain[45]. The participants with a record of pain were asked to rate their pain severity via the 100-mm horizontal Visual Analogue Scale for pain (VAS). The participants rated their pain severity on a 100-mm horizontal fixed line from left to right with higher scores indicating greater pain[57].

Covariates

The potential confounding variables including social support, functional independence, stroke severity, stroke type, laterality, stroke duration, body mass index, number of comorbidities/stroke risk factors (hypertension, diabetes, cancer, obesity, arthritis, cigarette smoking, alcohol consumption, respiratory disease, heart disease, kidney disease) and socio-demographic characteristics (age, gender, years of education, monthly income, and employment status) were assessed. The Duke-University of North Carolina Functional Social Support Questionnaire (DUFSS) was used to evaluate the social support available to participants. The DUFFS is a measure of perceived social support with 11 items scored on a 5-point Likert scale. The aggregate scores range from 11 to 55, with higher score suggesting higher social support availability[58,59]. The level of functional independence was measured by the Modified Barthel Index (MBI). The MBI, which is well-validated in stroke patients, is used to assess patients' ability to perform 10 types of activities of daily living and has a maximum of 100 scores, with higher scores indicating a greater level of functional independence[60,61]. The National Institutes of Health Stroke Scale (NIHSS) was used to evaluate stroke severity. The NIHSS is a validated 11-item instrument with each item scoring between a 0 and 4. A higher score is indicative of higher stroke severity level. The maximum possible score is 42, with the minimum being 0[62]. Stroke duration was categorised as early subacute (7 days-3 months), late subacute ($>3\leq 6$ months) and chronic (>6 months) stroke[63].

Data analysis

Descriptive statistics of frequency, percentage, median, mean and standard deviation were used to summarize data. The independent *t*-test and chi-square test of association were used to investigate factors associated with PSEI, PSAP, PSP and PSSD. The binary logistic regression models expressed in odds ratio (OR) and 95% confidence interval (CI) were employed to assess the impact of composite PSSD and PSP, and the different domains of PSSD (sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, sleep medication, daytime sleep and PSP (post-stroke headache, central post-stroke pain, peripheral post-stroke pain, other pain and pain severity) on PSEI and PSAP. Age and gender were adjusted for all the logistic regression models including other factors significantly associated with PSEI and PSAP in bivariate analyses. Thus, age, gender and stroke severity were adjusted in PSEI models, while PSAP models were adjusted for age, gender, body mass, income, education, employment, stroke severity, social support and functional independence. The alpha level was set to $p<0.05$. Data analysis was conducted with the SPSS 21.0 version (SPSS Inc., Chicago, Illinois, USA).

RESULTS

The general characteristics of the participants are presented in Table 1. The mean age was 58.45 ± 10.16 years,

Table 1

General characteristics of the participants (n = 185)	
Variable	Mean (SD) or N/ %
Age (years)	58.45 (10.16)
Gender (Male)	112/60.50
BMI (Kgm^{-2})	26.54 (4.09)
Marital status (Married)	162/87.60
Income level (High)	118/63.80
Stroke type (Ischaemic)	140/75.7
Laterality (Right)	98/53.0
Stroke duration (months)	6 (19)[a]
Early sub-acute	57/30.80
Late sub-acute	38/20.50
Chronic	90/48.60
Years of education	12.89 (4.64)
Employment status (Employed)	137/74.10
Number of morbidities	2.14 (1.23)
NIHSS	2.66 (2.61)
DUFSS	41.85 (10.33)
MBI	78.06 (24.86)
PSP status (Yes)[b]	117/63.25
Post-stroke headache (Yes)	46/24.90
Central post-stroke pain (Yes)	16/8.60
Peripheral post-stroke pain (Yes)	99/53.50
Other pain (Yes)	12/6.50
VAS (Pain) (mm)	30.68 (28.98)
Global PSQI	5.84 (4.63)
PSQI sleep quality	0.70 (0.84)
PSQI sleep latency	1.09 (1.06)
PSQI sleep duration	1.02 (1.01)
PSQI sleep efficiency	0.78 (0.90)
PSQI sleep disturbances	0.96 (0.80)
PSQI sleep medication	0.41 (0.88)
PSQI daytime sleep	0.93 (1.03)
Sleep disorder (Yes)	68/36.80
PSEI status (Yes)	83/44.9
PSAP status (Yes)	43/23.2

SD – standard deviation; N – number; % – percentage; NIHSS – National Institute of Health Stroke Scale; DUFSS – Duke University of North Carolina Functional Social Support Questionnaire; MBI – Modified Barthel Index; PSP – Post-stroke Pain; VAS – Visual Analogue Scale; PSQI – Pittsburgh Sleep Quality Index; PSEI – post-stroke emotional incontinence; PSAP – post-stroke anger proneness; [a] – median (interquartile range); [b] – some stroke survivors reported more than one type of post-stroke pain

with majority of the participants being male (60.5%). A total of 75.7% had ischaemic stroke, others were in chronic phase (48.6%) and had 2.66±2.61 mean NIHSS scores. The prevalence of PSP, PSSD, PSEI and PSAP was 63.3%, 36.8%, 44.9% and 23.2%, respectively. While considering the prevalence of different PSSD and PSP domains, sleep latency (mean; standard deviation: 1.09±1.06) and peripheral post-stroke pain (53.50%) were the most prevalent. The prevalence of central post-stroke pain was 8.60%, while 44 (23.8%) reported more than one type of PSP.

As shown in Table 2, PSP was significantly associated with PSEI (χ^2=8.498; p=0.004), while participants with PSEI had significantly more stroke severity than those without, as measured by NIHSS (3.07±2.5 vs. 2.33±2.7; t=-1.935; p=0.049). Meanwhile, PSAP was significantly associated with income level (χ^2=5.667; p=0.017) and employment status (χ^2=8.077; p=0.004), and those with the presence of PSAP had significantly higher levels of body mass (BMI: 27.8±4.2 vs. 26.2±4.0; t=-2.270; p=0.024), more years of education (14.5±4.4 vs. 12.4±4.6; t=-2.581; p=0.011), higher stroke severity (NIHSS: 3.72±2.8 vs. 2.35±2.5; t=-3.091; p=0.002), less social support (DUFSS: 38.3±12.4 vs. 42.9±9.4; t=2.585; p=0.011) and lower functional independence (MBI: 71.4±28.6 vs. 79.6±20.4; t=1.966; p=0.048) (Table 2).

Furthermore, PSP was significantly associated with gender (χ^2=5.971; p=0.015), marital status (χ^2=15.625; p<0.001), income level (χ^2=13.608; p<0.001), stroke duration (χ^2=24.372; p<0.001), employment status (χ^2=5.341; p=0.021), less years of education (12.1±4.5 vs. 14.3±4.5; t=3.219; p=0.002), higher stroke severity (NIHSS: 3.20±2.8 vs. 1.75±2.0; t=-3.753; p<0.001) and lower functional independence (MBI: 75.4±24.6 vs. 82.7±17.3; t=1.935; p=0.049) (Table 2). In Table 2, it is shown that PSSD was significantly associated with stroke duration (χ^2=24.372; p<0.001), PSP (χ^2=9.992; p=0.002), higher body mass (BMI: 27.4±4.8 vs. 26.0±3.5; t=-2.276; p=0.024), higher number of co-morbidities (2.44±1.2 vs. 1.96±1.2; t=-2.615; p=0.010) and lower social support (DUFSS: 38.8±10.8 vs. 43.6±9.6; t=3.096; p=0.002).

After adjustment, the results of binary logistic regression models demonstrated that PSP as a composite variable had significant impact on PSEI (adjusted odds ratio (aOR): 0.492; 95% CI: 0.251-0.965; p=0.039). However, PSSD as a composite variable had no significant impact on PSEI (p>0.05). While assessing the impact of different PSSD and PSP domains on PSEI, the results showed that sleep disturbances (aOR: 1.855; 95% CI:

Table 2

Factors associated with post-stroke emotional incontinence, post-stroke anger proneness, post-stroke pain and post-stroke sleep disorders (N =185)

Variables	PSEI			PSAP			PSP			PSSD		
	Yes	No	Test-statistic	Yes	No	Test-statistic	Yes	No	Test-statistic	Yes	No	Test-statistic
Age (years) [a]	59.1(10.1)	57.8(10.2)	-0.840 [ns]	57.3(9.58)	58.8(10.3)	0.844 [ns]	58.8(10.2)	57.9(10.2)	-0.562 [ns]	59.7(9.3)	57.8(10.5)	-1.224 [ns]
Gender[b]												
Male	47/56.6	65/63.7	0.965 [ns]	23/53.5	89/62.7	1.166 [ns]	63/53.8	49/72.1	5.971*	44/64.7	68/58.1	0.781 [ns]
Female	36/43.4	37/36.3		20/46.5	53/37.3		54/46.2	19/27.9		24/35.3	49/41.9	
BMI (Kgm^{-2}) [a]	26.8(4.2)	26.3(4.0)	-0.794 [ns]	27.8(4.2)	26.2(4.0)	-2.270*	26.8(4.0)	26.1(4.1)	-1.010 [ns]	27.4(4.8)	26.0(3.5)	-2.276*
Marital status[b]												
Married	75/90.4	87/85.3	1.079 [ns]	40/93.0	122/85.9	1.532 [ns]	94/80.3	68/100.0	15.265***	59/86.8	103/88.0	0.064 [ns]
Single[c]	8/9.6	15/14.7		3/7.0	20/14.1		23/19.7	0/0.0		9/13.2	14/12.0	
Income level[b]												
Low	31/37.3	36/35.3	0.084 [ns]	9/20.9	58/40.8	5.667*	54/46.2	13/19.1	13.608***	24/35.3	43/36.8	0.040 [ns]
High	52/62.7	66/64.7		34/79.1	84/59.2		63/53.8	55/80.9		44/64.7	74/63.2	
Stroke duration[b]												
Early subacute	20/24.1	37/36.3	4.828 [ns]	13/30.2	44/31.0	0.181 [ns]	22/18.8	35/51.5	24.372***	15/22.1	42/35.9	22.415***
Late subacute	22/26.5	16/15.7		8/18.6	30/21.1		24/20.5	14/20.6		5/7.4	33/28.2	
Chronic	41/49.4	49/48.0		22/51.2	68/47.9		71/60.7	19/27.9		48/70.5	42/35.9	
Years of education[a]	13.2(5.0)	12.7(4.3)	-0.712 [ns]	14.5(4.4)	12.4(4.6)	-2.581*	12.1(4.5)	14.3(4.5)	3.219**	13.1(4.8)	12.8(4.6)	-0.449 [ns]
Employment status[b]												
Employed	63/75.9	74/72.5	0.268 [ns]	39/90.7	98/69.0	8.077**	80/68.4	57/83.8	5.341*	47/69.1	90/76.9	1.364 [ns]
Unemployed	20/24.1	28/27.5		4/9.3	44/31.0		37/31.6	11/16.2		21/30.9	27/23.1	
Number of Co-morbidities[a]	2.17(1.2)	2.11(1.2)	-0.333 [ns]	2.09(1.5)	2.15(1.2)	0.255 [ns]	2.16(1.1)	2.09(1.5)	-0.394 [ns]	2.44(1.2)	1.96(1.2)	-2.615*
Stroke severity[a] (NIHSS)	3.07(2.5)	2.33(2.7)	-1.935*	3.72(2.8)	2.35(2.5)	-3.091**	3.20(2.8)	1.75(2.0)	-3.753***	3.01(2.5)	2.46(2.7)	-1.390 [ns]
Social support[a] (DUFSS)	40.7(10.2)	42.9(10.4)	1.431 [ns]	38.3(12.4)	42.9(9.4)	2.585*	41.5(10.6)	42.4(9.9)	0.540 [ns]	38.8(10.8)	43.6(9.6)	3.096**
Functional independence[a] MBI	75.5(25.5)	80.1(19.9)	1.235 [ns]	71.4(28.6)	79.6(20.4)	1.966*	75.4(24.6)	82.7(17.3)	1.935*	78.9(25.4)	77.6(24.6)	-0.365 [ns]
PSP[b]												
Yes	62/74.7	55/53.9	8.498**	26/60.5	91/64.1	0.186 [ns]	----------	----------	----------	53/77.9	64/54.7	9.992**
No	21/25.3	47/46.1		17/39.5	51/35.9		----------	----------		15/22.1	53/45.3	
PSSD[b]												
Yes	33/39.8	35/34.3	0.584 [ns]	15/34.9	53/37.3	0.085 [ns]	----------	----------	----------	----------	----------	----------
No	50/60.2	67/65.7		28/65.1	89/62.7		----------	----------	----------	----------	----------	----------

[a] – independent *t*-test in mean and standard deviation; [b] – chi square in number and percentages; [c] – included the unmarried, divorced, separated or widowed; PSEI-post-stroke emotional incontinence; PSAP post-stroke anger proneness; PSP post-stroke pain; PSSD post-stroke sleep disorders; NIHSS National Institute of Health Stroke Scale; DUFSS Duke University of North Carolina Functional Social Support Questionnaire; MBI Modified Barthel I ndex; ns non-significance; * $p<0.05$; ** $p<0.01$; *** $p<0.001$.

Medical Rehabilitation (Med Rehabil) 2023, 27 (4): 15-25 DOI: 10.5604/01.3001.0054.0117 eISSN 1896–3250

Table 3

Binary regression model relating the impact of composite post-stroke sleep disorders and post-stroke pain on post-stroke emotional incontinence

Variable	B	β	95% CI of β (lower-upper)	*p*-value
Age	0.008	1.008	0.978-1.039	0.598
Gender	0.092	1.097	0.587-2.049	0.772
Stroke severity	0.052	1.053	0.933-1.188	0.402
PSQI	0.049	1.050	0.980-1.125	0.164
PSP	-0.708	0.492	0.251-0.965	0.039*

B unstandardised beta; β standardised beta; CI confidence interval; PSQI Pittsburgh Sleep Quality Index; PSP post-stroke pain; *significance at $p<0.05$.

Table 4

Binary regression model relating the impact of different domains of post-stroke sleep disorders and post-stroke pain on post-stroke emotional incontinence

Variable	B	β	95% CI of β (lower-upper)	*p*-value
Age	0.004	1.004	0.972-1.036	0.831
Gender	-0.078	0.925	0.458-1.870	0.828
Stroke severity	0.049	1.051	0.918-1.202	0.473
Post-stroke sleep disorders				
Sleep quality	-0.071	0.932	0.550-1.579	0.793
Sleep latency	-0.324	0.723	0.483-1.082	0.115
Sleep duration	0.178	1.194	0.695-2.052	0.520
Sleep efficiency	0.023	1.023	0.593-1.765	0.935
Sleep disturbances	0.618	1.855	1.096-3.140	0.021*
Sleep medication	-0.171	0.843	0.564-1.260	0.406
Daytime sleep	0.129	1.137	0.747-1.731	0.548
Post-stroke pain				
Post-stroke headache	-1.010	0.364	0.153-0.864	0.022*
Central post-stroke pain	-0.214	0.808	0.382-1.708	0.576
Peripheral post-stroke pain	-0.032	0.968	0.269-3.485	0.961
Other pain	1.381	3.978	0.903-17.529	0.068
Pain severity	0.001	1.000	0.981-1.020	0.996

B – unstandardised beta; β – standardised beta; CI – confidence interval; * – significance at $p<0.05$

1.096-3.140; $p=0.021$) and headache (aOR: 0.364: 95% CI: 0.153-0.864; $p=0.022$) were the only domains of PSSD and PSP that had significant impact on PSEI (Tables 3 and 4). Furthermore, PSH (aOR: 0.052; 95% CI: 0.011-0.238; $p<0.001$) was the only domain of PSP that was significantly associated with PSAP. In addition, education (aOR: 1.223; 95% CI: 1.309-1.439; $p=0.015$), employment (aOR: 0.122; 95% CI: 0.023-0.653; $p=0.014$) and stroke severity (aOR: 1.467; 95% CI: 1.111-1.936; $p=0.007$) were significantly associated with PSAP. Nonetheless, PSSD and PSP treated as composite variables and none of domains of PSSD were associated with PSAP in the logistic regression analyses ($p>0.05$) (Tables 5 and 6).

DISCUSSION

In this cross-sectional observational study, the prevalence and correlates of PSSD, PSP, PSAP and PSEI were examined among Nigerian stroke survivors. It was further investigated whether PSSD and PSP, as composite variables, and the different components of PSSD (sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication and daytime sleep dysfunction) as well as PSP (post-stroke headache, central post-stroke pain, peripheral post-stroke pain, other pain and pain severity) were associated with PSAP and PSEI. The findings revealed a prevalence of 36.8%, 63.3%, 23.2% and 44.9%, respectively, for PSSD, PSP, PSAP and PSEI. The most prevalent domains of PSSD and PSP in the studied cohort of stroke survivors were sleep latency and peripheral post-stroke pain.

Table 5

Binary regression model relating the impact of composite post-stroke sleep disorders and post-stroke pain on post-stroke anger proneness

Variable	B	β	95% CI of β (lower-upper)	*p*-value
Age	0.042	1.043	0.983-1.106	0.162
Gender	0.339	1.403	0.597-3.298	0.437
BMI	0.030	1.030	0.922-1.150	0.600
Income	-0.278	0.758	0.256-2.238	0.615
Education	0.105	1.110	0.994-1.241	0.064
Employment	-2.111	0.121	0.027-0.540	0.006*
Stroke severity	0.249	1.282	1.050-1.566	0.015*
Social support	-0.032	0.969	0.929-1.011	0.146
Functional independence	0.002	1.002	0.983-1.002	0.820
PSQI	-0.034	0.966	0.872-1.072	0.512
PSP	0.015	1.015	0.398-2.589	0.975

B – unstandardised beta; β – standardised beta; CI – confidence interval; BMI – body mass index; PSQI – Pittsburgh Sleep Quality Index; PSP – post-stroke pain; * –significance at $p<0.05$

Table 6

Binary regression model relating the impact of different domains of post-stroke sleep disorders and post-stroke pain on post-stroke anger proneness

Variable	B	β	95% CI of β (lower-upper)	*p*-value
Age	0.036	1.037	0.973-1.105	0.267
Gender	-0.064	0.938	0.329-2.672	0.924
BMI	0.115	1.122	0.982-1.282	0.091
Income	0.637	1.891	0.441-8.109	0.391
Education	0.201	1.223	1.309-1.439	0.015*
Employment	-2.105	0.122	0.023-0.653	0.014*
Stroke severity	0.383	1.467	1.111-1.936	0.007*
Social support	-0.028	0.972	0.925-1.022	0.273
Functional independence	0.007	1.007	0.983-1.031	0.588
Post-stroke sleep disorders				
Sleep quality	-0.055	0.947	0.471-1.903	0.878
Sleep latency	-0.098	0.906	0.532-1.544	0.718
Sleep duration	0.554	1.741	0.829-3.655	0.143
Sleep efficiency	-0.810	0.445	0.180-1.099	0.079
Sleep disturbances	0.322	1.380	0.662-2.879	0.390
Sleep medication	-0.448	0.639	0.329-1.240	0.185
Daytime sleep	-0.302	0.739	0.367-1.489	0.398
Post-stroke pain				
Post-stroke headache	-2.957	0.052	0.011-0.238	0.000**
Central post-stroke pain	0.405	1.499	0.282-7.591	0.635
Peripheral post-stroke pain	0.261	1.299	0.160-10.553	0.807
Other pain	2.526	12.507	0.890-175.727	0.061
Pain severity	-0.009	0.992	0.959-1.025	0.611

B – unstandardised beta; β – standardised beta; CI – confidence interval; BMI – body mass index; PSQI – Pittsburgh Sleep Quality Index; PSP – post-stroke pain; * – significance at $p<0.05$; ** – significance at $p<0.001$

Although the prevalence of PSSD, PSP, PSAP and PSEI varies depending on the instruments utilised and time of post-stroke assessment, the rates obtained in this study are comparable to the global prevalence reported in literature. In a recent systematic review and meta-analysis, the rate of PSSD, assessed using sleep quality questionnaire, has been established as between 19.8% to 69%[64], while in a review by Harrison and Field[26], these authors made an estimation of up to 11-66% for PSP. Moreover, the results of a meta-analysis involving 2,608 patients put the prevalence of PSAP at between 12 to 57%[65], while in reports, the prevalence of PSEI has been indicated as being up to 40%[16,17]. Furthermore, similar to reports by other researchers, sleep latency[31,66,67] and peripheral neuropathic pain[33,47] were mostly prevalent among the investigated community-dwelling stroke survivors in this study.

According to the findings of this study, several factors, including PSP, stroke duration, body mass, number of co-morbidities and social support, were associated with PSSD. These factors have also been mentioned in earlier studies[31,68-74]. Moreover, gender, marital status, income, employment status, education, stroke duration, stroke severity and functional independence were significantly associated with PSP. In previous studies, significant associations between PSP and gender, stroke duration, stroke severity and functional independence have been reported[26,33,45]. The relationship of PSP with some socio-demographic characteristics of stroke survivors, including marital status, income level, employment status and years of education, have not been properly elucidated in the literature as done in this study, which calls for more research. It is possible that stroke survivors with no companionship, low income level, unemployed or with low education level may experience or report different pain syndromes.

Concerning PSAP, PSP and PSSD as composite variables, none were associated with PSAP. However, while assessing the influence of different PSP and PSSD domains, PSAP was significantly associated with PSH, a domain of PSP. None of the PSSD domains were associated with PSAP in this study. While there is a lack of data to be compared with the findings of this study, PSH is a common pain subtype after stroke[26]. Although, the pathophysiology of PSH is not yet fully known, tension headache is common in stroke survivors and involves stimulation of trigemino-vascular system resulting from brain infarction[75-77]. Meanwhile, the bio-psychosocial model propounded by Gatchel et al. indicates a link between emotional distress and pain[78]. Evidence has earlier shown that emotional disorders, e.g. depression, are associated with pain in stroke[79]. The presence of pain often increases feelings of anger and irritability[80]. The findings of our study indicate that PSH, out of many domains of PSP, is implicated in anger and anger proneness among stroke survivors.

Stroke survivors exert more energy than during the pre-stroke stage while carrying out most basic activities, which often lead to physical exertion. Meanwhile, physical exertion is reportedly associated with PSH[80] due to blood vessel constriction in the face, neck and head, as well as increased blood pressure[81]. It is possible that these community-dwelling stroke survivors, while attempting to engage in social and physical activity, experience surges in PSH and thereby, becomes angry and irritable. Furthermore, stroke survivors may be prone to anger or become irritable if they are not able or rather willing to engage in social or physical activity when engaging in them results or precipitates headache. The findings of an earlier study had indicated that PSAP is a predictor of social isolation in patients with stroke[17]. In addition, the results of a qualitative study have indicated that stroke survivors with PSH may present with pain catastrophising as they opined that they may be experiencing repeat stroke or formation of a new clot with the attendant fear, heightened emotion and worry[76] which may precipitate anger. Some PSAP symptoms, including hitting or hurting others, kicking, biting, throwing objects, cursing and screaming, impulsiveness, hostility, intolerance and being less generous ,which are unfortunately often directed towards their caregivers, family, friends and colleagues[7,82]. These symptoms of anger further decrease quality of life for stroke survivors and impair good rehabilitation outcomes[9,10]. Therefore, appropriate attention may be needed in addressing the symptoms of PSH in this cohort for combating PSAP. This is more so as it has been indicated in reports that stroke survivors with PSP found PSH more disabling than any other domains of PSP and tend to report and use more analgesics for PSH than any other PSP domains[45]. Other factors in multivariate analysis found to be significantly associated with PSAP apart from PSH were education, employment and stroke severity. Several authors have similarly found that stroke severity and motor or neurological dysfunction is a consistent correlate of anger proneness in individuals with stroke[2,15,17,82,83]. However, there is dearth of data on the influence of socioeconomic factors, including education and employment, on PSAP as found in this study. It is plausible for stroke survivors with low socio-economic status to be more aggressive and irritable.

In respect to PSEI, PSP and not PSSD were associated with PSEI when treated as composite variables. In addition, PSH and sleep disturbances as PSP and PSSD domains were significantly associated with PSEI. This finding indicates that stroke survivors with pain may likely experience excessive laughing or crying. This is not farfetched as evidence has shown that emotional incontinence, such as moaning, groaning and crying are the main avenue for non-verbal vocalisations of pain by patients[84]. Furthermore, since PSP is a known factor associated with reduction in social participation and functional activities in stroke survivors[32,33,43,85], the loss or decrease in social or physical participation by stroke survivors occasioned by PSP can make them feeling sorry for themselves and may lead to emotional incontinence. A feeling of helplessness or inability to perform

basic pre-stroke activities by stroke survivors may encourage rumination, which may facilitate emotional incontinence. In a previous report, it has been noted that some psychological disorders, i.e. depression, anxiety and stress are common in patients with higher ruminating tendency[86].

Similar to the findings for PSAP, only PSH from PSP domains was significantly associated with PSEI. Coupled with the evidence which has shown that PSH is very disabling to stroke survivors[45], mood disorders are reportedly associated with headache[87,88]. Tension headache is the most common sub-type of headache in stroke which has been found to result from tension in the face and neck[80,81]. Meanwhile, emotional incontinence such as crying also activates and tenses up the jaw, neck and facial muscles, which may trigger headache[89]. Thus, there is apparent association between facial muscle tension resulting from emotional incontinence and headache after a stroke episode. Nonetheless, longitudinal studies may be warranted to ascertain the direction of association between the onset of PSH and emotional incontinence.

Furthermore, sleep disturbances as a domain of PSSD were significantly associated with PSEI. In this study, stroke survivors with emotional incontinence reported higher sleep disturbances than those without PSEI. One of the most common sleep disorders among stroke survivors is insomnia, which largely occurs from nighttime sleep disturbances[34,39,40]. According to PSQI, sleep disturbances include waking up in the middle of the night or early in the morning, having to get up to use the bathroom, experiencing pain, etc. Stroke survivors are prone to insomnia from sleep disturbances partly due to the medications often prescribed for stroke and its co-morbidities[90]. For instance, it has been reported that anti-hypertensive medications such as beta-blockers and diuretics induce insomnia, disrupt the rapid eye movement (REM) cycle, cause early morning wakeness, bad dreams and painful calf cramps while asleep[90]. Apart from strong evidence linking sleep disturbances to limited functional recovery and social participation, insomnia is strongly connected with poor life satisfaction among stroke patients [90], thus, it is possible for stroke survivors with sleep disturbances to be more prone to emotional disorders such as PSEI. It is common knowledge that sleep deprivation is an independent predictor of many psychological disorders, including depression, anxiety, fatigue and cognitive impairment[91], therefore, it is not surprising that sleep disturbances are associated with another related disorder, PSEI.

The brain tissue damage following stroke is related to sleep disturbances and emotional disorders experienced by stroke survivors[83,92]. Although, there is yet no consensus, it has been shown in reports that PSEI, PSAP and PSD share similar pathogenic serotonergic mechanisms and lesion distribution[7,83]. Thus, these factors suggest that indices alike sleep disturbances that are associated with depression, anxiety, cognition and fatigue may show similar traits as PSEI. This was also indicated in the findings of this study. However, as stated earlier, sleep disorders were not associated with PSAP in this study, which may warrant further inquiries on the mechanisms between sleep disorders and emotional post-stroke disorders. Nonetheless, these discrepancies may also be a result of different factors (genetic, social, environmental and neurologic) that are uniquely related to PSAP and PSEI[15,16]. In addition, despite the fact that PSD, PSAP, PSEI and other emotional disorders in stroke are associated with neurochemical derangement secondary to brain injury, there are subtle differences in their manifestations and correlates[15]. According to the literature on the subject, some of these differences include varying anatomic locations for each of these emotional disorders[4,17,93], as well as varying factors associated with these emotional disorders at different stages of stroke[15,94].

This study has a few potential limitations. First of all, this is a cross-sectional observational study, which precludes us from inferring causality. Secondly, although we employed validated tools in assessing sleep disorders, pain, emotional incontinence and anger proneness, their self-reporting nature may have introduced reporting bias. Thirdly, due to the methodology, we excluded patients with cognitive impairments, severe communication problems and those living alone, while including a relatively small sample from just two centres. Due to this, our findings may not be generalisable to all stroke survivors. Therefore, prospective studies with larger samples drawn from multiple centres may be needed to consolidate the findings of this study.

CONCLUSION

The rates of PSSD, PSP, PSAP and PSEI among Nigerian stroke survivors are 36.8%, 63.3%, 23.2% and 44.9%, respectively. Several factors including PSP, stroke duration, body mass, number of co-morbidities and social support were associated with PSSD. Furthermore, gender, marital status, income, employment status, education, stroke duration, stroke severity and functional independence were significantly correlated with PSP. The presence of post-stroke headache was associated with PSAP, while sleep disturbances and PSH were connected with PSEI. Specific, other than composite sleep disorders, and type, rather than presence or severity of pain after stroke, appear to be associated with PSAP and PSEI. Adequate screening and treatment of pain and sleep disorders among stroke survivors may lessen the prevalence and burden of PSAP and PSEI.

Ethics Committee

The Ethics Review Committee of the Osun State University Teaching Hospital, Osogbo, Nigeria gave approval for the study (LTH/EC/2021/01/496).

List of abbreviations

PSAP – Post-stroke anger proneness; PSEI- Post-stroke emotional incontinence; PSP – Post-stroke pain; PSSD – Post-stroke sleep disorders; CPSP – Central post-stroke pain; PPSP – Peripheral post-stroke pain; PSH – Post-stroke headache; PSD – Post-stroke depression; PSA – Post-stroke anxiety; PSF –

Post-stroke fatigue; STAS – Spielberger Trait Anger Scale; PSQI – Pittsburgh Sleep Quality Index; VAS – Visual Analogue Scale; DUFSS – Duke University of North Carolina Functional Social Support Questionnaire; MBI – Modified Barthel Index; NIHSS – National Institutes of Health Stroke Scale; aOR – Adjusted odds ratio; CI – Confidence interval

References

1. da Silva R.S., da Silva S.T., de Souza J.M., et al. Effects of inclined treadmill training on functional and cardiovascular parameters of stroke patients: study protocol for a randomized controlled trial. Trials 2019; 20 (1): 252.
2. Kim J.S. Post-stroke mood and emotional disturbances: Pharmacological therapy based on mechanisms. Strocke 2016; 18(3): 244-255.
3. Kim J.S. Pathologic laughter after unilateral stroke. J Neurol Sci 1997; 148: 121-125.
4. Kim J.S., Choi-Kwon S. Poststroke depression and emotional incontinence: correlation with lesion location. Neurology 2000; 54: 1805-1810.
5. Kim J.S. Post-stroke emotional incontinence after small lenticulocapular stroke: correlation with lesion location. J Neurol 2002; 249: 805-810.
6. Tang W.K., Chan S.S.M., Chiu H.F.K. et al. Emotional incontinence in Chinese stroke patients: diagnosis, frequency, and clinical and radiological correlates. J Neurol 2004; 251: 865-869.
7. Kim J.S., Choi-Kwon S., Kwon S.U., Seo Y.S. Inability to control anger or aggression after stroke. Neurology 2002; 58: 1106-1108.
8. Choi-Kwon S., Han S.W., Kwon S.U. et al. Fluoxetine treatment in poststroke depression, emotional incontinence, and anger proneness: a double-blind, placebo-controlled study. Stroke 2006; 37(1): 156-161.
9. Kauhanen M., Korpelainen J.T., Hiltunen P. et al. Poststroke depression correlates with cognitive impairment and neurological deficits. Stroke 1999; 30: 1875-1880.
10. King R.B. Quality of life after stroke. Stroke 1996; 27: 1467-1472.
11. Choi-Kwon S., Kim H.S., Kwon S.U., Kim J.S. Factors affecting the burden on caregivers of stroke survivors in Seoul, South Korea. Arch Phys Rehab Med 2005; 86: 1043-1048.
12. Kotila M., Numminen H., Waltimo O., Kaste M. Post-stroke depression and functional recovery in a population-based stroke register. The Finnstroke study. Eur J Neurol 1999; 6(3): 309-312.
13. Van de Weg F.B., Kuik D.J., Lankhorst G.J. Post-stroke depression and functional outcome: a cohort study investigating the influence of depression on functional recovery from stroke. Clin Rehabil 1999; 13: 268-272.
14. Noh S.M., Chung S.J., Kim K.K. et al. Emotional disturbance in CADASIL: Its impact on quality of life and caregiver burden. Cerebrovasc Dis 2014; 37: 188-194.
15. Choi-Kwon S., Han K., Choi S. et al. Poststroke depression and emotional incontinence: factors related to acute and subacute stages. Neurology 2012; 78: 1130-1137.
16. Choi M.H., Lim T.S., Yoon B.S. et al. Low Testosterone Level as a Predictor of Poststroke Emotional Disturbances: Anger Proneness and Emotional Incontinence. J Stroke Cerebrovasc Dis 2018; 27(12): 3549-3554.
17. Choi-Kwon S., Park D.I. Leadership in nursing research: Evidence based nursing in post-stroke emotional dysfunction. Nurs Sci Res 2020; 18(1): 18-24.
18. Xiao M., Huang G., Feng L. et al. Impact of sleep quality on post-stroke anxiety in stroke patients. Brain Behav 2020; 10: e01716.
19. Davis J.C., Falck R.S., Best J.R. et al. Examining the inter-relations of depression, physical function, and cognition with subjective sleep parameters among stroke survivors: a cross-sectional analysis. J Stroke Cerebrovasc Dis 2019; 28: 2115-2123.
20. Ferre A., Ribó M., Rodríguez-Luna D. et al. Strokes and their relationship with sleep and sleep disorders. Neurol 2013; 28: 103-118.
21. Kim J., Kim Y., Ik Yang K. et al. The relationship between sleep disturbance and functional status in mild stroke patients. Ann Rehabil Med 2015; 39: 545-552.
22. Breslau N., Roth T., Rosenthal L., Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry 1996; 39: 411-418.
23. Zhao L., Yang F., Sznajder K.K. et al. Resilience as the Mediating Factor in the Relationship Between Sleep Disturbance and Post-stroke Depression of Stroke Patients in China: A Structural Equation Modeling Analysis. Front Psychiatry 2021; 12: 625002.
24. Lundström E., Smits A., Terént A., Borg J. Risk factors for stroke-related pain 1 year after first ever stroke. Eur J Neurol 2009; 16: 188-193.
25. Jonsson A.C., Lindgren I., Hallstrom B. et al. Prevalence and intensity of pain after stroke: a population based study focusing on patients' perspectives. J Neurol Neurosurg Psychiatry 2006; 77: 590-595.
26. Harrison R.A., Field T.S. Post stroke pain: identification, assessment, and therapy. Cerebrovasc Dis 2015; 39(3-4): 190-201.
27. Robert G. Robinson, Ricardo E. Jorge, Post-Stroke Depression: A Review. Am J Psychiatry 2016; 173: 221-231.
28. Delpont B., Blanc C., Osseby G.V. et al. Pain after stroke. A review. Revue Neurol 2017; 174: 671-674.
29. Nadarajah M., Goh H.T. Post-stroke fatigue: A review on prevalence, correlates, measurement and management. Top. Stroke Rehabil 2015; 22: 208-220.
30. Scuteri D., Mantovani E., Tamburin S. et al. Opioids in Poststroke Pain: A Systematic Review and Meta-Analysis. Front. Pharmacol 2020; 11: 587050.
31. Khazaal W., Taliani M., Boutros C. et al. Psychological Complications at 3 Months Following Stroke: Prevalence and Correlates Among Stroke Survivors in Lebanon. Front. Psychol 2021; 12: 663267.
32. Liampas A., Velidakis N., Georgiou T. et al. Prevalence and Management Challenges in Central Post-Stroke Neuropathic Pain: A Systematic Review and Meta-analysis. Adv Ther 2020; 37: 3278-3291.
33. Westerlind E., Singh R., Persson H.C., Sunnerhagen K.S. Experienced pain after stroke: A cross-sectional 5-year follow-up study. BMC Neurol 2020; 20: 1-8.
34. Park D.I., Choi-Kwon S. Poststroke sleep disorders: an executive summary. J Neurosci Nurs 2018; 50(6): 314-317.
35. Suh M., Choi-Kwon S., Kim J.S. Sleep disturbances after cerebral infarction: role of depression and fatigue. J Stroke Cerebrovasc Dis 2014; 23(7): 1949-1955.
36. Dyken M.E., Bin Im K., Richerson B., Lin Dyken G.D.C. Sleep and stroke. Amerikaserikat: Oxford University Press; 2017.
37. Umbas D., Sulistyawati A., Bahar A. et al. Sleep quality in post stroke patient with and without type 2 diabetes mellitus: A comparative study. Med Clin Pract 2021; 4: 100202.
38. Baglioni C., Nissen C., Schweinoch A. et al. Polysomnographic Characteristics of Sleep in Stroke: A Systematic Review and Meta-Analysis. PLoS ONE 2016; 11(3): e0148496.
39. Pasic Z., Smajlovic D., Dostovic Z. et al. Incidence and types of sleep disorders in patients with stroke. Med Arch 2011; 65: 225-227.
40. Karaca B. Factors affecting poststroke sleep disorders. J Stroke Cerebrovasc Dis 2016; 25(3): 727-732.
41. Klit H., Finnerup N.B., Jensen T.S. Central post-stroke pain: clinical characteristics, pathophysiology, and management. Lancet Neurol 2009; 8(9): 857-868.
42. Klit H., Finnerup N.B., Overvad K. et al. Pain following stroke: a population-based follow -up study. PLoS One 2011; 6(11): e27607.
43. Delpont B., Blanc C., Osseby G.V. et al. Pain after stroke: a review. Rev Neurol (Paris) 2018; 174(10): 671-674.
44. Chan K-L., Campayo A., Moser D.J. et al. Aggressive behavior in patients with stroke: association with psychopathology and results of antidepressant treatment on aggression. Arch Phys Med Rehabil 2006; 87: 793-798.
45. Naess H., Lunde L., Brogger J., Waje-Andreassen U. Post-stroke pain o long-term follow -up: the Bergen stroke study. J Neurol 2010; 257(9): 1446-1452.
46. Seifert C.L., Mallar Chakravarty M., Srenger T. The complexities of pain after stroke: A review with a focus on central post-stroke pain. Panminerva Med 2013; 55: 1-10.
47. Treister A.K., Hatch M.H., Cramer S.C., Chang E.Y. Demystifying post-stroke pain: from etiology to treatment. PM R 2017; 9(1): 63-75.
48. Faulkner S., Sidey-Gibbons C. Use of the Pittsburgh Sleep Quality Index in People With Schizophrenia Spectrum Disorders: A Mixed Methods Study. Front. Psychiatry 2019; 10: 284.
49. Cai H., Wang X.P., Yang G.Y. Sleep Disorders in Stroke: An Update on Management. Aging Dis. 2021; 12(2): 570-585.
50. Kasiulevicius V., Sapoka V., Filipaviciute R. Sample size calculation in epidemiological studies. Gerontologija 2006; 7(4): 225-231.
51. Spilberger C.D., Jacobs G., Russell S., Crane R.S. Assessment of anger: the State-Trait Anger-Scale. In: Butcher JN, Spielberger CD, eds. Advances in Personality Assessment, vol. 2. Hillsdale, NJ: Lawrence Erlbaum Associates 1983: 161-189.
52. House A., Dennis M., Molyneux A. et al. Emotionalism after stroke. BMJ 1989; 298(6679): 991-994.
53. Buysse D.J., Reynolds C.F., III, Monk T.H. et al. The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. Psychiatry Res 1989; 28: 193e213.
54. Hashmi A.M., Khawaja IS., Butt Z. et al. The Pittsburgh Sleep Quality Index: validation of the Urdu translation. J Coll Physicians Surg Pak 2014; 24(2): 123-126.
55. Spallone V., Morganti R., D'amato C. et al. Validation of DN4 as a screening tool for neuropathic pain in painful diabetic polyneuropathy. Diabetic Med 2012; 29: 578-585.
56. Bashir A.H., Abdullahi A., Abba M.A., Mukhtar N.B. Central poststroke pain: its profile among stroke survivors in Kano, Nigeria. Behav. Neurol. 2017; 9318597.
57. Streiner D.L., Norman G.R., Cairney J. Health measurement scales: A practical guide to their development and use. Oxford University Press, New York. 1989.
58. Broadhead W.E., Gehlbach S.H., De Gruy F.V., Kaplan B.H. The Duke-UNC functional social support questionnaire: measurement of social support in family medicine patients. Med Care 1988; 26: 709-723.
59. de la Revilla Ahumada L., Bailón E., de Dios Luna J. et al. Validación de una escala de apoyo social funcional para su uso en la consulta del médico de familia [Validation of a Functional Social Support Scale for Use in the Family Doctor's Office]. Aten Primaria. 1991 Oct; 8(9): 688-892.
60. Shan S., Vanclay F., Cooper B. Improving the sensitivity of the Barthel Index for stroke rehabilitation. J Clin Epidemio1989; 42(8): 703-709.
61. Lee S.Y., Kim D.Y., Sohn M.K. et al. Determining the cut-off score for the Modified Barthel Index and the Modified Ranking Scale for assessment of functional independence and residual disability after stroke. PLoS One 2020; 15(1): e0226324.
62. Hage V. "The NIH stroke scale: a window into neurological status". Nurs Spectr 2011; 24(15): 44-49.
63. Bernhardt J., Hayward K.S., Kwakkel G. et al. Agreed definitions and a shared vision

for new standards in stroke recovery research: the stroke recovery and rehabilitation roundtable taskforce. Int J Stroke 2017; 12: 444-450.
64. Baylan S., Griffiths S., Grant N, et al. Incidence and prevalence of post-stroke insomnia: a systematic review and meta analysis. Sleep Med Rev 2020; 49.
65. Ramos-Perdigues S., Mane-Santacana A., Pintor-Perez L. Prevalence and associated factors of anger post stroke: a systematic review. Rev Neurol 2015; 60: 481-489.
66. Mansour A.H., Ayad M., El-Khayat N. et al. Post-stroke sleep disorders in Egyptian patients by using simply administered questionnaires: a study from Ain Shams University. Egypt J Neurol Psychiatry Neurosurg 2020; 56: 13.
67. Ilkhan G.D., Celikhisar H. Assessment of Sleep Quality and Related Factors in Stroke Patients. American J Multi Res Dev 2020; 2(6): 27-34.
68. Bakken L.N., Lee K.A., Kim H.S. et al. Sleep -Wake Patterns during the Acute Phase after First-Ever Stroke. Stroke Res Treat 2011; 2011: 936298.
69. Bakken L.N., Kim H.S., Finset A., Lerdal A. Subjective sleep quality in relation to objective sleep estimates: Comparison, gender differences and changes between the acute phase and the six-month follow-up after stroke. J Adv Nurs 2014; 70(3): 639-650.
70. Sterr A., Herron K., Dijk D.J., Ellis J. Time to wake-up: sleep problems and daytime sleepiness in long-term stroke survivors. Brain Inj 2008; 22(7–8): 575-579.
71. Da Rocha P.C.., Barroso MT., Dantas A.A. et al. Predictive factors of subjective sleep quality and insomnia complaint in patients with stroke: implications for clinical practice. An Acad Bras Cienc 2013; 85(3): 1197-1206.
72. Purani H., Friedrichsen S., Allen A.M. Sleep quality in cigarette smokers: Associations with smoking-related outcomes and exercise. Addict Behav 2019; 90: 71-76.
73. Liu J.T., Lee I.H., Wang C.H. et al. Cigarette smoking might impair memory and sleep quality. J Formos Med Assoc 2013; 112(5): 287-290.
74. Galligan N., Hevey D., Coen R., Harbison J. Clarifying the associations between anxiety, depression and fatigue following stroke. J. Health Psychol 2016; 2112: 2863-2871.
75. Hansen A.P., Marcussen N.S., Klit H. et al. Pain following stroke: a prospective study. Eur J Pain 2012; 16: 1128-1136.
76. Widar M., Ek A., Ahlstrom G. Coping with Long-Term Pain after a Stroke. J Pain Symptom Manag 2004; 27: 3215-3225.
77. Sommerfeld D.K., Welmer A.K. Pain following stroke, initially and at 3 and 18 months after stroke, and its association with other disabilities. Eur J Neurol 2012; 19: 1325-1330.
78. Gatchel R.J., Peng Y.B., Peters M.L. et al. The bio-psychosocial approach to chronic pain: Scientific advances and future directions. Psychol Bull 1998; 133: 581-624.
79. Lindgren I., Gard G., Brogårdh C. Shoulder pain after stroke—Experiences, consequences in daily life and effects of interventions: A qualitative study. Disabil Rehabil 2018; 40: 1176-1182.
80. Widar M., Ahlström G., Ek A.C. Health-related quality of life in persons with long-term pain after a stroke. J Clin Nurs 2004; 13: 497-505.
81. McCory P. Headaches and exercise. Sports Med 2012; 30: 221-229.
82. Kim J.S., Lee E.J., Chang D.I. et al. Efficacy of early administration of escitalopram on depressive and emotional symptoms and neurological dysfunction after stroke: a multicentre, double-blind, randomised, placebo-controlled study. Lancet Psychiatry 2017; 4(1): 33-41.
83. Choi-Kwon S., Han K., Cho K.H. et al. Factors associated with post-stroke anger proneness in ischaemic stroke patients. Eur J Neurol 2013; 20: 1305-1310.
84. Helmer L.M.L., Weijenberg R.A.F., de Vries R. et al. Crying out in pain-A systematic review into the validity of vocalization as an indicator for pain. Eur J Pain 2020; 24: 1703-1715.
85. Naess H., Beiske A.G., Myhr K.M. Quality of life among young patients with ischaemic stroke compared with patients with multiple sclerosis. Acta Neurol Scand 2008; 117: 181-185.
86. Michl L.C., McLaughlin K.A., Shepherd K., Nolen-Hoeksema S. Rumination as a Mechanism Linking Stressful Life Events to Symptoms of Depression and Anxiety: Longitudinal Evidence in Early Adolescents and Adults. J Abnorm Psychol 2013; 122(2): 339-352.
87. Wacogne C., Lacoste J.P., GuillibertE. et al. Stress, anxiety, depression and migraine. Cephalalgia 2003; 23(6): 451-455.
88. Peres MFP, Mercante JPP, Tobo PR, Kamei H, Bigal ME. Anxiety and depression symptoms and migraine: a symptom-based approach research. J Headache Pain 2017; 18(1): 37.
89. Arendt-Nielsen L. Headache: muscle tension, trigger points and referred pain. Int J Clin Pract Suppl 2015; 182: 8-12.
90. Cai H., Wang X.P., Yang G.Y. Sleep Disorders in Stroke: An Update on Management. Aging Dis. 2021; 12(2): 570-585.
91. Chennaoui M., Leger D., Gomez-Merino D. Sleep and the GH/IGF-1 axis: Consequences and countermeasures of sleep loss/disorders. Sleep Med Rev 2020; 49: 101223.
92. Khazaei S., Ayubi E., Khazaei M. et al. Sleep Quality and Related Determinants among Stroke Patients: A Cross-Sectional Study. Iran J Psych 2022; 17: 1: 84-90.
93. Grajny K., Pyata H., Spiege K. Depression symptoms in chronic left hemisphere stroke are related to dorsolateral prefrontal cortex damage. J Neuropsychiatry Clin Neurosci 2016; 28(4): 292-298.
94. Ko M., Choi-Kwon S., Jun S.E. et al. Poststroke emotional disturbances and a tryptophan hydroxylase 2 gene polymorphism. Brain Behav. 2018 Jan 11;8(2):e00892.

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