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Prevalence and risk factors of post-stroke pain in a Malaysian stroke centre: A cross sectional study

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Abstract

Background & Objectives: Post-stroke pain (PSP) is a common complication that is often overlooked. It leads to depression, impaired quality of life (QoL) and increased economic burden. In this study, we aimed to determine the prevalence and risk factors of PSP in a Malaysian stroke centre. *Methods*: This is a single-centered, cross-sectional study of 175 post-stroke patients attending the neurology clinic. Their demographic data and clinical variables were collected. They were interviewed using the Brief Pain Inventory (BPI), Geriatric Depression Scale (GDS), Barthel Score (BI) and EuroQol-5D (EQ-5D) questionnaires. *Results*: The prevalence of PSP was 26.3%, with most of them aged 51-70 years (52.2%). The types of pain included headache (30.4%), pain secondary to spasticity (32.6%), central post-stroke pain (26.1%) and shoulder joint pain (19.6%), in which they first experienced the pain between a week to three months post-index stroke. Our study showed that a high NIHSS, prolonged hospital stay for index stroke, poor modified Rankin Scale (mRS) and no post-discharge rehabilitation increased the probability of developing PSP. This resulted in depression, regression of functional status and poor QoL. There was no correlation between older age, gender, ethnicity, and pre-existing medical conditions with the development of PSP.
Conclusion: PSP should be diligently screened and treated in every stroke survivor to improve quality of life.

Keywords: Post-stroke pain, stroke, risk factors, quality of life, depression

INTRODUCTION

For decades, stroke has been a major global concern. In 2019, stroke was the third leading cause of disability and death worldwide with a total of 12.2 million incident cases of stroke and 6.5 million stroke related deaths.[1] From 2009 to 2016, The National Stroke Registry of Malaysia reported a total number of 11,284 cases of stroke.[2] Stroke was also known to be the top cause of death in Malaysia after ischaemic heart disease and pneumonia.[3]

The advancement in treatment and rehabilitation has led to a growing number of stroke survivors. Only one third will suffer from a certain degree of disability.[4] These patients experience numerous complications, namely, functional and cognitive decline, depression, suicidal ideation and pain. To date, PSP has been one complication that is often left unnoticed in stroke care and research.

Pain, defined as "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage", is perceived by our brain via two pathways.[5] The medial pain pathway that enables a patient to express the unpleasant feeling caused by the pain, and the lateral pain pathway that is responsible to locate the pain.[6,7]

PSP may begin during the acute onset of stroke or several months later. The incidence of PSP varies widely from 10% to 70%.[8] The vast difference can be explained by different study designs, cohort samples and follow up period.[9] A cross sectional study by Westerlind *et al*., proved that PSP can persist up to five years post-stroke.[10] Risk factors for PSP include old age, female gender, alcohol consumption, diabetes, depression and presence of any pain prior to index stroke.[8,11] In addition, large vessel ischaemic stroke, infarct

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at the thalamic, parietal and brainstem areas, post-stroke mRS of 3 to 5 and presence of spasticity increases the risk for PSP.[12]

PSP can generally be divided into neuropathic pain due to central post-stroke pain (CPSP) or a range of other pain types such as headache, shoulder joint pain and pain secondary to spasticity. It is also associated with muscle spasm and, to a smaller degree, complex regional pain syndrome.[13] A patient can have more than one type of pain at any time.[8]

PSP has a subjective nature and lack standard diagnostic criteria. To our knowledge, there is no Malaysian data on the prevalence of PSP and its impact. Hence, the primary aim of this study was to determine the prevalence of PSP in our stroke centre. The secondary end points were describing the types of pain, predictors of developing pain and to assess the impact of PSP on our stroke patients.

METHODS

This was a single centre, cross sectional study, conducted at the Neurology clinic of Universiti Kebangsaan Malaysia Medical Centre (UKMMC), with the approval of the Research Ethics Committee (Project Code: JPE-2021-494).

The sample size for this study was calculated based on a meta-analysis done by Lundstrom *et al*.[18] Taking a prevalence of 21.0%, with a confidence level of 95%, the sample size was 255 patients. Patients with first onset of stroke (cerebral infarction or intracerebral haemorrhage) within the last five years, aged 18 and above, who were able to give informed consent were recruited into the study.

Their background history and clinical variables were obtained from clinic notes prior to the interview. The assessment tools used were Brief Pain Inventory (BPI) to assess the pain severity; Geriatric Depression Scale (GDS) to look for depression; Barthel Score (BI) to assess the degree of dependency and EuroQol-5D (EQ-5D) score for QoL.

The statistical analysis was performed using IBM Statistical Product and Service Solutions (SPSS) version 26.0. Descriptive statistics was used to present the patients' background, underlying medical condition, stroke characteristics and PSP characteristics. Normally distributed data were expressed using mean ± standard deviation; whereas data that was not normally distributed were reported using median (inter-quartile range).

In addition, Pearson Chi-square test, Fisher's exact test, Independent t-test, and Mann-Whitney test were used to compare the significance between variables and mean values with the presence of PSP. A p-value of <0.05 was considered statistically significant. Multivariate analysis using binary logistic regression model was done to assess the independent risk factors for post-stroke patients.

RESULTS

Due to Covid-19, the number of patients visiting the clinic during this study was significantly reduced and we managed to recruit 175 post-stroke patients. The mean age (± standard deviation [SD]) was 63.7 years (±12.0 years), with predominantly males (N=108, 61.7%). More than half (N=88, 50.3%) were Malay. Hypertension (N=151, 86.3%) was the most common comorbidity. Patients with ischaemic stroke were the main contributors to this study with a total of 170 patients (97.1%) with thalamic (N=97, 55.4%) and cortical (N=82, 48.0%) areas being mainly involved. One-hundred-and-forty-six patients (83.4%) had lacunar syndrome (LACS). (Table 1)

The prevalence of PSP in our cohort of patients was 26.3% (N=46). The majority was between 51-70 years (N=24, 52.2%) and there was an equal gender distribution. Hypertension (N=40, 87.0%), hyperlipidaemia (N=18, 39.1%) and diabetes (N=14, 30.4%) were the top three comorbidities recorded. Only 9 (19.6%) complained of having any kind of pain prior to index stroke. Most of the patients in our cohort had their index stroke within one year (N=14, 30.4%) from the study period.

During the index stroke, patients with PSP presented to our hospital with a moderate NIHSS score (N=19, 50.0%). Lacunar syndrome (LACS) was the most common type of ischaemic stroke. The thalamic (N=22, 50.0%) and cortical (N=24, 54.5%) regions were equally affected among these patients. Patients with PSP were admitted for a median of 4 days, and they predominantly had a poor mRS score of 3-6 upon discharge (N=29, 63.0%). Twenty-eight (70.0%) were referred for rehabilitation post-discharge. More than half (N=25, 54.3%) were depressed, 34 (73.1%) had slight to moderate dependency level and a mean EQ5D score of 7.6 (±2.7).

Using the univariate analysis, we determined that age, gender, ethnicity, smoking, alcohol consumption, medical conditions, presence of pain and spasticity prior to index stroke, duration of

Table 1: Patients' demographic and stroke characteristics (N=175)

Characteristics	n (%)
Patient Demographic	
Ethnicity	
Malay	88 (50.3)
Chinese	71 (40.6)
Indian	13 (7.4)
Others	3 (1.7)
Age (years)	
31-50	28 (16.0)
51-70	98 (56.0)
71-90	49 (28.0)
Gender	
Male	108 (61.7)
Female	67 (38.3)
Employment	
Yes	72 (41.1)
No	103 (58.9)
Activity of daily living (ADL)	
Independent	174 (99.4)
Dependent	1 (0.6)
Smoker	
Yes	62 (35.4)
No	113 (64.6)
Alcohol Consumption	
Yes	17 (9.7)
No	158 (90.3)
Medical Condition	
Heart disease	35 (20.0)
Hypertension	151 (86.3)
Diabetes mellitus	53 (30.3)
HbA1C [Mean (±*SD*)]	8.1% (±2.3%)
Hyperlipidaemia	63 (36.0)
Dementia	1 (0.6)
Depression	6 (3.4)
Cancer	3 (1.7)
Joint disease	12 (6.9)
Pain prior to index stroke	21 (12.0)
Spasticity prior to index stroke	1 (0.6)
Stroke Characteristics	
Stroke	
Ischaemic	170 (97.1)
Haemorrhagic	5 (2.9)
Classification	
Haemorrhagic Stroke	5 (2.9)
Lacunar Syndrome (LACS)	146 (83.4)

Partial Anterior Circulation Syndrome (PACS)	9 (5.1)
Total Anterior Circulation Syndrome (TACS)	4 (2.3)
Posterior Circulation syndrome (POCS)	11 (6.3)
Thrombolysis	
Yes	17 (9.7)
No	153 (87.4)
Site	
Thalamic	97 (55.4)
Cortical	82 (48.0)
Brainstem	13 (7.6)
Glasgow Coma Scale (GCS)	
Mean (±*SD*)	14.8 (±0.5)
NIHSS	
Mild (0-4)	106 (70.2)
Moderate (5-15)	42 (27.8)
Severe (≥16)	3 (2.0)
Length of hospital stay (days)	
Median (IQR)	4 (2)
Range	1-20
Modified Rankin Scale (mRS)	
Good (0-2)	92 (58.6)
Poor (3-6)	65 (41.4)
Rehabilitation post discharge	
Yes	67 (42.7)
No	90 (57.3)

SD = Standard Deviation

stroke types of stroke, sensory disturbances, stroke classifications, stroke site and thrombolysis given were not statistically significant when correlated with the presence of PSP. (Table 2)

We further analyzed the other risk factors using Binary Logistic Regression model and noticed a significant difference within these two groups (pain and no pain group) with regards to the NIHSS, length of hospital stay, mRS on discharge, rehabilitation post-discharge, GDS score, BI score and EQ-5D score. We can conclude that high NIHSS, a longer hospital-stay, poor MRS on discharge and lack of rehabilitation post-discharge can contribute to developing PSP. These can lead to depression, dependence and poor QoL. (Table 3)

Among those with PSP, pain secondary to spasticity (N=15, 32.6%) and headache (N=14, 30.4%) were the most common types of pain encountered. They also complained of having CPSP and shoulder joint pain, with a total of 12 (26.1%) and 9 (19.6%) patients respectively. Most of them first experienced the pain within the first week to three months (N=33, 71.7%) from index stroke, affecting the same side as the weakness (N=32, 69.6%). Only 3 (6.5%) were affected by pain every day. 20 out of the 46 patients had mild pain during the interview session, with a majority of BPI severity score ranging 1-4 points. (Table 4)

DISCUSSION

The majority of our stroke survivors were aged 51-70 years, with hypertension as the main medical comorbidity. This is in keeping with the global statistics in 2019 which showed about two thirds of stroke occurred under the age of 70 years and hypertension was the largest single risk for stroke. In 2016, 9.5 million cases of ischemic stroke and 4.1 million hemorrhagic stroke were reported.[15] Although it is often mentioned that the incidence of stroke is almost equal between both genders, there appeared to be a male predominance in

Table 2: Univariate analysis of risk factors for post stroke pain.

Characteristics	Post-Stroke Patients (N=175)		Test value	*p*-value
	Pain (N=46) n (%)	No Pain (N=129) n (%)		
Patient Demographic				
Age (years)			0.70	0.70[a]
31-50	9 (19.6)	19 (67.9)		
51-70	26 (26.0)	74 (74.0)		
71-90	11 (23.4)	36 (76.6)		
Gender			3.62	0.06[a]
Male	23 (21.3)	85 (78.7)		
Female	23 (34.3)	44 (65.7)		
Ethnicity			2.80	0.09[a]
Malay	28 (31.8)	60 (68.2)		
Non Malay	18 (20.7)	69 (79.3)		
Smoker			1.40	0.24[a]
Yes	13 (21.0)	49 (79.0)		
No	33 (29.2)	80 (70.8)		
Alcohol Consumption				0.25[b]
Yes	2 (11.8)	15 (88.2)		
No	44 (27.8)	114 (72.2)		
Medical Condition				
Heart Disease	11 (31.4)	24 (68.6)	0.60	0.44[a]
Hypertension	40 (26.5)	111 (73.5)	0.02	0.88[a]
Diabetes Mellitus	14 (26.4)	39 (73.6)	0.00	0.98[a]
HbA1C [Mean (±*SD*)]	7.7% (±2.2%)	8.1% (±2.4%)		0.54[c]
Hyperlipidaemia	18 (28.6)	45 (71.4)	0.27	0.61[a]
Dementia	0 (0.0)	1 (100.0)		1.00[b]
Depression	1 (16.7)	5 (83.3)		1.00[b]
Cancer	0 (0.0)	3 (100.0)		0.57[b]
Joint Disease	4 (33.3)	8 (66.7)		0.52[b]
Pain prior to index stroke	9 (19.6)	12 (9.3)		0.07[a]
Spasticity prior to index stroke	0 (0.0)	1 (0.8)		1.00[a]
Stroke Characteristics				
Duration of stroke since onset (years)			4.42	0.35[a]
1	14 (21.2)	52 (78.8)		
2	11 (36.7)	19 (63.3)		
3	8 (36.4)	14 (63.6)		
4	6 (27.3)	16 (72.7)		
5	7 (20.0)	28 (80.0)		
Types of stroke				0.61[b]
Ischemic	44 (25.9)	126 (74.1)		
Haemorrhagic	2 (40.0)	3 (60.0)		
NIHSS on presentation*			18.04	**<0.005[a]**
Mild stroke (0-4)	16 (15.2)	89 (84.8)		
Moderate to severe stroke (≥5)	22 (47.8)	24 (52.2)		
Sensory disturbances				0.05[b]
Yes	5 (55.6)	4 (44.4)		
No	39 (24.2)	122 (75.8)		

Stroke classification			0.03	0.86[a]
Lacunar syndrome (LACS)	38 (26.0)	108 (74.0)		
Others	8 (27.6)	21 (72.4)		
Thrombolysis				0.15[b]
Yes	7 (41.2)	10 (58.8)		
No	37 (24.2)	116 (75.8)		
Stroke site				
Thalamic	22 (22.7)	75 (77.3)	1.21	0.27[a]
Cortex	24 (29.3)	58 (70.7)	1.03	0.31[a]
Brainstem	3 (23.1)	10 (76.9)		1.00[b]
Length of hospital stay (days)			-2.60	**0.009[d]**
Median (IQR)	5 (4)	4 (3)		
Range	2-20	1-19		
Modified Rankin Scale on discharge (mRS)			21.40	**<0.005[a]**
Good (0-2)	11 (12.0)	81 (88.0)		
Poor (3-6)	29 (44.6)	36 (55.4)		
Rehabilitation post-discharge			16.38	**<0.005[a]**
Yes	28 (41.8)	39 (58.2)		
No	12 (13.3)	78 (86.7)		
Post Stroke Complications				
Geriatric Depression Scale			41.27	**<0.005[a]**
No depression (0-4)	21 (15.2)	117 (84.8)		
Depressed (≥5)	25 (67.6)	12 (32.4)		
Barthel Index Score				**<0.005[b]**
Severe to total dependency (0-60)	12 (70.6)	5 (29.4)		
Slight to moderate dependency (61-100)	34 (21.5)	124 (78.5)		
EQ-5D score				**<0.005[c]**
Mean (±*SD*)	7.6±2.7	5.8±1.3		

SD = Standard deviation, [a] = Pearson Chi-Square test, [b] = Fisher's exact test, [c] = Independent t-test, [d] = Mann-Whitney test
*NIHSS on presentation for the pain sub-group, N=38 as there are 5 missing data.

Table 3: Multivariate analysis using Binary Logistic Regression model on independent risk factors for post-stroke pain

Characteristics	OR	95% CI	*p*-value
NIHSS	5.10	[2.32,11.19]	<0.005*
Length of hospital stay (days)	1.20	[1.05,1.36]	0.009*
Modified Rankin Scale on discharge (mRS)	5.93	[2.67,13.17]	<0.005*
Rehabilitation post-discharge	4.67	[2.14,10.16]	<0.005*
Geriatric Depression Scale	11.61	[5.06,26.63]	<0.005*
Barthel Index Score	8.75	[2.88,26.56]	<0.005*
EQ-5D score	1.56	[1.30,1.88]	<0.005*

OR = odd ratio, CI = confidence interval, * = statistically significant *p<0.05*

Table 4: Characteristics of pain in post-stroke patients

Characteristics	Post-stroke Pain (N=46) n (%)
Pain types	
Headache	14 (30.4)
Shoulder pain	9 (19.6)
Pain secondary to spasticity	15 (32.6)
Central post-stroke pain	12 (26.1)
Site	
Other sites	5 (10.9)
Same side as weakness	32 (69.6)
Onset	
<1 week	6 (13.0)
1 week to 3 months	33 (71.7)
3 to 6 months	3 (6.5)
>6 months	4 (8.7)
Frequency	
Everyday	3 (6.5)
Most days	17 (37.0)
Less than once a week	26 (56.5)
Other features	
Hypersensitivity to touch	0 (0.0)
Pain to touch	3 (6.5)
Brief Pain Inventory (BPI) Severity	
No pain (0)	0 (0.0)
Mild pain (1-4)	20 (43.5)
Moderate pain (5-6)	0 (0.0)
Severe pain (7-10)	0 (0.0)

our cohort.

The prevalence of new onset PSP among our stroke survivors was 26.3% (N=46), in parallel with other studies.[8] The 4 common types of PSP described by Hansen *et.al*. include headache, shoulder joint pain, pain secondary to spasticity and CPSP. This new onset of pain was seen at a younger age (mean age of 63.3 years).[8] Similarly, in our study, headache (N=14, 30.4%) and pain secondary to spasticity (N=15, 32.6%) were the main contributors to PSP, followed by CPSP (N=12, 26.1%) and shoulder joint pain (N=9, 19.6%). More than half of our patients (N=26, 56.5%, mean 63.7 years) were aged between 51 to 70 years.

In the recent Prevention Regimen for Effectively avoiding Second Stroke (PRoFESS) trial, the risk factors for PSP were accounted to increased stroke severity, female gender, presence of medical conditions (previous depression, hyperlipidaemia and diabetes mellitus), causing a greater dependence and cognitive decline.[16] There are also many studies which reported diabetes and depression as risk factors for PSP.[12,13,17]

Interestingly, from our study, we demonstrated that the PSP patients when compared to the no pain group, had higher NIHSS scores, longer admission for stroke and a poor mRS upon discharge. A higher NIHSS translates to a more disabling stroke. Some may even develop secondary complications such as orthostatic pneumonia, urinary tract infections or pressure sore, which requires longer hospital stay. Hence, poorer mRS upon discharge. These patients are prone to develop PSP. When they experience PSP of any form, if left untreated, depression sets in, causing a decline in their functional status and a poor quality of life.

Our study did not demonstrate a correlation between the ethnicity, gender and underlying medical conditions. The majority of our PSP cohort was of Malay (N=28, 60.9%) ethnicity.

This is representative of the national population as Malay is the largest ethnic group in Malaysia. Even though there was an over representation of male patients in our study, both genders were equally affected by PSP with no statistical significance. It can be argued that gender is not a reliable predictor of PSP although there may be cultural differences.[18] For example Zhang *et al*.[19] found that female stroke patients showed a higher mechanical pain sensitivity than male stroke patients whereas Andersen *et al*.[20] showed that gender was not a predictor of central pain after stroke. One Turkish study[21] showed a female preponderance while a Nigerian study showed the opposite.[22] Data from a Malaysian observational study supported our findings that there was no ethnic difference in pain tolerance although Indian females appeared to have a lower pain tolerance.[23]

Hypertension (N=40, 87.0%) was most commonly seen in our patients followed by hyperlipidaemia (N=18, 39.0%), diabetes (N=14, 30.4%), heart disease (N=11, 23.9%) and depression (N=1, 2.2%). The diabetic patients with PSP had suboptimal HbA1c. There were no statistical differences between these variables. Longstanding diabetes leads to peripheral neuropathy, often described as numbness experienced over the hands and feet. The numbness is perceived as a different sensation to pain among our PSP patients. We know that microvascular complications, including, diabetic neuropathy are strongly correlated with HbA1c. Although we did not perform nerve conductions studies in our diabetic patients, we felt it was unlikely that the pain they experienced was that of diabetic neuropathy due to its description and presence of moderate glycaemic control. Our cohort had one patient with depression whose condition was well-controlled, and she did not experience PSP.

There were no significant difference between the pain and no pain groups of post-stroke patients (p=0.27) with thalamic stroke. This can be explained by the fact that many cerebral regions process pain apart from the thalamus. Pain fibres are also seen in the cerebral cortex and brainstem. The more severe the stroke, the larger the affected area, the more pain fibres are involved and as a result, the patients experience a more severe pain.

Our data showed that PSP was associated with depression and poorer QoL. The relationship between PSP and depression may be two-way. Depression may be both a cause and a response to pain. PSP has long-lasting effects on mental health and QoL. Several studies have identified a significant negative association between depression and quality of life. Choi-Kwon *et al*. found that the presence of depression at 3 months post-stroke was significantly associated with a low QoL 3 years later.[24] Westerlind *et al*. found that patients who experience more frequent pain had a significant increase of depression at 5 years post-stroke.[9]

We found a significant association between PSP and dependence. We hypothesise that this relationship may also be bidirectional: Pain negatively affects physical function while greater functional dependence can also cause pain. However, evidence is conflicting. Jonsson *et al*. reported a significant association between a higher NIHSS score (greater disability) and pain intensity[10] while Westerlind *et al*. found that patients who were functionally dependent at discharge reported more frequent pain and had higher odds of experiencing more frequent pain at 5 years.[9] Other studies found no association between pain and physical function.[25,26] What is clear is that preserved physical function and independence were determined as important aspects of QoL.[27]

Our data also showed a significant association between PSP and declining QoL. Studies suggest that pain has a negative effect on QoL.[22,27] Pain, physical dependence, depression and QoL are all intertwined. It can be difficult to identify which precedes the other because the associations cross domains. The biopsychosocial model formulated by Gatchel *et al*. views illness as the complex interaction of biological, psychological and social factors.[28] There is a growing recognition that psychosocial factors, such as emotional stress, could influence the reporting of symptoms and response to treatment.

During our analysis, we noticed a significant negative association between rehabilitation and PSP. We postulate that in post-stroke patients with poor mRS who receive adequate rehabilitation post-discharge, the likelihood of developing PSP is low. Physical therapy is a known strategy to manage PSP. A simplified theory is that the imbalance of the excitatory and inhibitory systems within the sensory pathways are disrupted after a stroke resulting in pain. One possible explanation why rehabilitation may help in pain management is that it helps modify the maladaptive neuroplasticity in pain processing pathways.[29] Rehabilitation involves more than just physical therapy. Other strategies such as electrical stimulation and mirror therapy have also

been shown to help in CPSP.[30] Botulinum toxin injection and dynamic splints may help relieve pain caused by joint spasticity.

It has been reported that almost three quarters of stroke survivors experience PSP within the first two years of index stroke.[8] This can cause a substantial burden towards both the caregivers and the socio-economy of the country. These stroke survivors develop a degree of pain which reduces their ability to perform daily activities and contribute financially to the family. They depend on caregivers, who are often their spouses and children. The caregivers are expected to help the stroke patients with daily medical care, feeding and dressing on top of attending to their own personal needs. Hence, caregivers often get mentally and physically affected. Unfortunately, there are limited opportunities for self -relief and social support.[31] Financially, there will also be an increase in expenditure particularly when on pain treatment. In spite of this, PSP has not been extensively looked into in Malaysia. Physicians and care takers should be aware that undiagnosed PSP is present in a significant number of stroke survivors. Stroke management should not only include pharmacological treatment for risk factor control, but also a comprehensive stroke rehabilitation in every stroke survivor regardless of their condition post-stroke.

Our study had several limitations. It was conducted during the peak of the Covid-19 pandemic. The sample size was smaller than planned as the number of patients visiting the clinic throughout the duration of our study was reduced to comply to the standard operating procedures. Furthermore, this study was conducted in a single centre. Most of our patients were diagnosed with lacunar stroke with minor or no residual weakness. Therefore, our population cohort was not truly representative of the entire stroke spectrum. Most stroke survivors with severe disability or who were bedbound were either lost to tertiary hospital follow up or chose to isolate at home during the pandemic. We also excluded patients with disabilities that might affect the reliability of self-reported pain such as those with aphasia, neglect and cognitive impairment. Inclusion of these patients would likely demonstrate a more significant degree of PSP.

In conclusion, post-stroke pain is present in 26.3% of the stroke patients under follow up in UKMMC. The most common types of pain were headache, pain secondary to spasticity, central post-stroke pain and shoulder joint pain. Stroke survivors with higher NIHSS scores, longer admission for stroke and a poor mRS upon discharge were at higher risk of PSP. PSP was associated with physical dependence, depression and poor QoL. Rehabilitation helped reduce the development of PSP. We recommend that screening for PSP be part of the follow up routine for all stroke patients. Further comprehensive studies are needed for better evaluation and treatment of PSP in order to improve the quality of life of stroke survivors.

DISCLOSURE

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