



# BMJ Open Incidence, characteristics and outcome of post-stroke recrudescence in the Chinese population: a single-centre observational study

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## ABSTRACT

**Objectives** The aim of our observational study was to investigate the incidence, clinical characteristics and outcome of post-stroke recrudescence (PSR) in the Chinese population.

**Design and setting** Single-centre prospective observational study in China.

**Participants** A total of 1114 patients who had a suspected stroke were prospectively screened from October 2020 to February 2022.

**Outcome measures** The primary outcome was the proportion of patients with functional independence defined as a score of 0–2 on the modified Rankin Scale (mRS) at 3 months. Secondary outcomes were: early neurological improvement (ENI), defined as a National Institutes of Health Stroke Scale (NIHSS) score of 0 or an improvement of  $\geq 2$  points from admission at 24 hours; mortality within 3 months; stroke recurrence within 3 months and length of stay in hospital.

**Results** A total of 959 patients with cerebral infarction and 30 patients without an available magnetic resonance imaging (MRI) scan were excluded. Among the 125 included patients, 27 cases of PSR (2.4%), 50 cases of transient ischaemic attack (TIA) (4.5%) and 48 cases of stroke mimics (SMs) (4.3%) were identified. A higher frequency of infection at admission (22.2% vs 2%,  $p=0.007$ ) was observed in patients with PSR compared with patients with TIA, and a lower proportion of functional independence at 3 months (80% vs 98%,  $p=0.015$ ) was seen. Patients with TIA had a higher frequency of ENI compared with patients with PSR and SMs (98% vs 59.3%,  $p<0.001$ ; 98% vs 52.1%,  $p<0.001$ ). Patients with PSR exhibited a higher frequency of grade 2 Fazekas deep white matter hyperintensity compared with those with SMs (33.3% vs 8.3%,  $p=0.010$ ).

**Conclusions** PSR is not uncommon in patients presenting with stroke symptoms and can be distinguished from TIA and SMs based on a combination of clinical features and trigger in the Chinese population. The neurological deficits of patients with PSR often resolve within several days following the resolution of the trigger.

## INTRODUCTION

Focal neurological deficits commonly occur as a sudden onset in patients suspected of

## STRENGTHS AND LIMITATIONS OF THIS STUDY

- ⇒ This was a prospective observational study of the clinical features and outcomes of PSR in the Chinese population.
- ⇒ This study was conducted at a single centre, with a limited sample size.
- ⇒ Only suspected patients presenting within 72 hours of symptom onset were included, potentially introducing selection bias.
- ⇒ Patients who did not undergo an MRI scan were excluded, which may lead to an underestimation of the prevalence of PSR.

having acute ischaemic stroke. The hyper-acute clinical differentiation of stroke and non-stroke conditions remains a frequent challenge.<sup>1</sup> A stroke mimic (SM)s is typically characterised as a condition not as a result of cerebral ischaemia, yet it exhibits neurological symptoms that are indistinguishable from those of a stroke, with an estimated incidence of approximately 2.8–16% in the emergency room.<sup>2–4</sup> Differentiating ‘new focal neurological symptoms’ that occur in the presence of a previous stroke, known as post-stroke recrudescence (PSR)—a specific type of SMs—presents a unique challenge, with an estimated incidence of approximately 9–10%.<sup>5,6</sup>

Early identification of certain cases of SMs through relevant examinations, including clinical symptoms and ancillary tests, can be achieved; however, obtaining an early definitive diagnosis of PSR presents particular challenges. The presence or absence of ischaemic lesions can be differentiated by MRI; focal acute neurological deficits such as PSR, SMs, and transient ischaemic attack (TIA) cannot be distinguished, even after undergoing MRI examination. However, recent study has demonstrated a higher prevalence of chronic brain infarctions and white

matter hyperintensities (WMHs) in patients with TIA and ischaemic stroke when compared with those with SMs, suggesting a predictive role of these markers for TIA and ischaemic stroke.<sup>7</sup> We speculated that imaging markers might be valuable in differentiating PSR from SMs.

Although previous studies have focused on PSR, there is a scarcity of relevant research on imaging markers and prospective studies conducted within the Chinese population. The aim of this study was to compare the prevalence, clinical characteristics, imaging features and functional outcomes among patients with PSR, TIA and SMs in the Chinese population.

## METHODS

### Design, setting and population

We conducted a single-centre prospective observational study to investigate the incidence, clinical characteristics and outcome of PSR in the Chinese population. Patients at the First Affiliated Hospital of Chongqing Medical University with suspected acute ischaemic stroke within 72 hours of symptom onset at our institution between October 2020 and February 2022 were included.

Patients were eligible if they have met the following criteria: (1) age  $\geq 18$  years and (2) suspected acute ischaemic stroke within 72 hours of symptom onset after admission. Patients were excluded from the study if: (1) they had no MRI after admission and (2) they had ischaemic infraction. PSR, characterised by the emergence of 'new focal neurological symptoms' in patients with a history of stroke, was diagnosed according to the criteria established by Topcuoglu *et al.*<sup>5</sup> TIA relied on the presence of clinical symptoms characterised by temporary neurological dysfunction, absence of lesions on diffusion-weighted imaging of MRI and duration of less than 24 hours.<sup>8,9</sup> SMs was defined as a condition characterised by neurological symptoms similar to those of stroke but not caused by cerebral ischaemia,<sup>9</sup> and PSR was considered a separate entity from SMs.

### Data collection

A Research Electronic Data Capture (REDCap) system was used for data collection,<sup>10</sup> and the collected information was analysed anonymously. The following data from our study were extracted: demographics, clinical characteristics, medical history, medication use prior to admission, relevant laboratory results (white cell count, neutrophil blood cell count, neutrophil to lymphocyte ratio, high-sensitivity C reactive protein (hsCRP)), the level of serum sodium (hyponatraemia: serum sodium  $<135$  mmol/L; hypernatraemia: serum sodium  $>145$  mmol/L), the level of serum potassium (hypokalaemia: serum potassium  $<3.5$  mmol/L; hyperkalaemia: serum potassium  $>5.5$  mmol/L), National Institutes of Health Stroke Scale (NIHSS) score at admission and premorbid modified Rankin Scale (mRS) score, SMs categories, treatment details, complications, comorbidities, prognosis and length of stay.

## Imaging

The imaging was evaluated by two experienced neurologists, who were blinded to the clinical data. The WMH was evaluated using fluid-attenuated inversion recovery (FLAIR) sequences of MRI scans, while periventricular hyperintensity (PVH) and deep white matter pathology (DWMP) were rated on Fazekas scale.<sup>11</sup> Basal ganglia subscores were assigned as follows: a score of 0 indicates no lesions; a score of 1 indicates punctate foci; a score of 2 indicates beginning confluence of foci and a score of 3 indicates large confluent areas. For PVH subscores, a score of 0 indicates no lesions; a score of 1 indicates 'caps' or pencil lining; a score of 2 indicates smooth 'halo' and a score of 3 indicates irregular PVH extending into the deep white matter.<sup>11</sup> The visually presented quantity scale of age-related white matter changes (ARWMCs) was used to evaluate WMH. The degree of hyperintense on FLAIR was graded on a scale of 0–3 in the following brain territories: bilateral frontal, occipital-parietal, basal ganglia, temporal and infratentorial brain. White matter lesions were graded on a scale of 0–3 as follows: 0=no lesions, 1=focal lesions, 2=beginning confluence of lesions, 3=diffuse involvement of the entire region with or without involvement of U fibres. Basal ganglia were rated on a scale of 0–3 as follows: 0=no lesions, 1=a single observed focal lesion ( $\geq 5$  mm), 2=two or more focal lesions, 3=confluent lesions.<sup>12</sup>

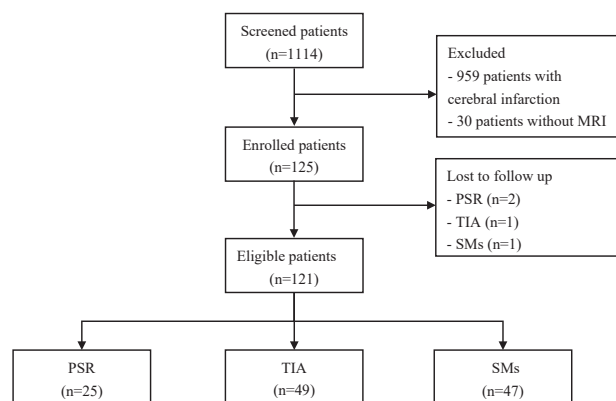
## Outcome measures

The primary outcome was the proportion of patients achieving functional independence at 3 months, defined as an mRS score of 0–2.<sup>13</sup> Secondary outcomes were: (1) early neurological improvement (ENI) defined as NIHSS score of 0 or improvement  $\geq 2$  points within 24 hours from admission; (2) mortality within 3 months; (3) stroke recurrence within 3 months and (4) length of stay in hospital. Patients were followed up by telephone interview at 3 months after onset of symptoms by trained investigators based on a standardised interview protocol. Follow-up information collected at each follow-up included mRS, all causes of death and stroke recurrence.

## Statistical analysis

Descriptive statistics were presented to describe the means with SD for variables with a normal distribution, and to describe the medians along with the IQR consisting of the 25th and 75th percentiles for variables with a non-normal distribution. The clinical characteristics of quantitative data among the three groups were analysed with the Kruskal-Wallis test, while the characteristics of qualitative data were compared with  $\chi^2$  test or Fisher exact. Comparisons between two groups were performed with the Bonferroni correction. When comparing differences within groups for nominal and quantitative data, statistical significance was deemed significant when the p value was less than 0.017 (0.05/3), in order to account for the conduct of multiple statistical tests within the same variable. Since the prospective cohort study aimed to explore

Figure 1 Flow chart of patient selection



**Figure 1** Flow chart of patient selection. PSR, post-stroke recrudescence; SMs, stroke mimics; TIA, transient ischaemic attack.

the prevalence, clinical characteristics, imaging features and functional outcomes among patients with PSR, TIA and SMs in the Chinese population, the sample size was not predetermined. A significance level of  $p < 0.05$  (two tailed) was considered statistically significant. Data analysis was conducted using SPSS V.26.0 software.

### Patient and public involvement

None.

## RESULTS

### Study participants and characteristics

A total of 1114 patients were screened between October 2020 and February 2022. Among them, 959 patients with cerebral infarction and 30 patients without an available MRI scan were excluded. The final analysis included 125 patients, of whom, 27 individuals were diagnosed with PSR, 50 with TIA and 48 with SMs. The flow chart was illustrated in figure 1. The median age was 70 years (IQR 64–75) and 55.2% were male. The median NIHSS score on admission was 1 (IQR 0–3).

The median age of patients with PSR was 69 years (IQR 59–75), and 55.6% of them were male (table 1). Sensory disorders and dysarthria were more frequently observed in patients with PSR compared with those patients with TIA (33.3% vs 10%,  $p=0.027$ ; 33.3% vs 10%,  $p=0.027$ ), respectively (online supplemental table 1). The most common presenting symptoms of PSR were sensory disorders (33.3%), followed by dysarthria, hemiparesis, altered consciousness and facial paralysis. The median NIHSS score on admission was 1 (IQR 0.5–4), while the median NIHSS score at 24 hours was 1 (IQR 0–2). Additionally, 88.9% of patients with PSR had a premorbid mRS score of  $\leq 2$ . Among the 27 patients with PSR, 23 had a history of infarcts, 3 had intracerebral haemorrhage, and 1 had both infarcts and intracerebral haemorrhage. The mean interval between the index stroke and recrudescence was 6.3 years (SD 7.6).

### Risk factors, triggers and imaging features

As shown in table 1 and the online supplemental table 1, the groups exhibited significant differences in the following aspects: ischaemic stroke ( $p < 0.001$ ), intracerebral haemorrhage ( $p=0.023$ ), premorbid mRS  $\leq 2$  ( $p=0.026$ ), infection ( $p=0.017$ ) and D-dimer ( $p=0.002$ ). Patients with PSR had a higher frequency of previous ischaemic stroke compared with those with TIA and SM (88.9% vs 8%,  $p < 0.001$ ; 88.9% vs 2.1%,  $p < 0.001$ ). Infection was more prevalent in patients with PSR compared with those with TIA (22.2% vs 2%,  $p=0.007$ ). Moreover, although pneumonia and hsCRP were more frequent in patients with PSR, the differences did not reach statistical significance. The use of benzodiazepines and unknown time of onset of hyponatraemia did not significantly differ among the three groups. The distribution of Fazekas PVH scale and ARWMC score showed no significant differences between the groups (table 2). However, grade 2 of the Fazekas DWMP scale score was more commonly observed in patients with PSR compared with those with SM (33.3% vs 8.3%;  $p=0.010$ ) in the subtype distribution of the Fazekas DWMP scale. The most common categories of SMs (online supplemental table 2) were peripheral vertigo (16.7%), peripheral neuropathy (8.3%) and cognitive decline (8.3%).

### Outcomes

As shown in table 3 and figure 2, the proportion of patients achieving functional independence at 3 months was lower in patients with PSR compared with patients with TIA (80% vs 98%;  $p=0.015$ ). ENI was more frequently observed in patients with TIA compared with patients with PSR and SM (98% vs 59.3%,  $p < 0.001$ ; 98% vs 52.1%,  $p < 0.001$ ). There was also a higher tendency of ENI in patients with PSR compared with SMs, although the difference did not reach statistical significance. The mean length of stay was longer in patients with SMs compared with those with TIA ( $10.5 \pm 5.3$  vs  $8.2 \pm 3.6$ ;  $p=0.016$ ). Moreover, there were no significant differences among the groups in term of mortality, stroke recurrence, and the composite outcomes of mortality and stroke recurrence within 3 months.

## DISCUSSION

The findings of this study indicate a significantly higher incidence of infection triggers in patients with PSR compared with those with TIA. PSR can develop following ischaemic stroke. Patients with PSR were more likely to achieve functional independence at 3 months after the resolution of the triggering event, compared with their premorbid state. ENI was more frequently observed in patients with TIA compared with those with PSR and SMs. Patients with PSR exhibited a higher incidence and extent of DWMP in Fazekas score 2 compared with those with SMs. Patients with TIA generally had shorter hospital stays compared with those with SMs.

**Table 1** Clinical characteristics in patients with PSR, TIA and SMs

Variables	Overall (n=125)	PSR (n=27)	TIA (n=50)	SMs (n=48)	P value		
					Between groups	PSR vs TIA	PSR vs SMs TIA vs SMs
Age, median (IQR), years	70.0 (64.0–75.0)	69.0 (59.0–75.0)	70.0 (64.2–74.8)	69.5 (61.5–75.2)	0.961	–	–
Sex, male, n (%)	69 (55.2)	15 (55.6)	31 (62.0)	23 (47.9)	0.374	–	–
Medical history							
Ischaemic stroke, n (%)	29 (23.2)	24 (88.9)	4 (8)	1 (2.1)	<0.001	<0.001*	0.362*
Intracerebral haemorrhage, n (%)	6 (4.8)	4 (14.8)	1 (2.0)	1 (2.1)	0.023	0.048*†	0.053*†
Hypertension, n (%)	79 (63.2)	19 (70.4)	26 (52.0)	34 (70.8)	0.106	–	–
Diabetes, n (%)	41 (32.8)	10 (37.0)	16 (32.0)	15 (31.2)	0.866	–	–
Coronary heart disease, n (%)	15 (12.0)	4 (14.8)	6 (12.0)	5 (10.4)	0.854	–	–
Atrial fibrillation, n (%)	6 (4.8)	3 (11.1)	2 (4.0)	1 (2.1)	0.202	–	–
Current smoking, n (%)	36 (28.8)	10 (37.0)	13 (26.0)	13 (27.1)	0.562	–	–
Current drinking, n (%)	37 (29.6)	10 (37.0)	14 (28.0)	13 (27.1)	0.63	–	–
Medications use							
Antiplatelet, n (%)	15 (12.0)	7 (25.9)	6 (12.0)	2 (4.2)	0.021	0.200*†	0.009*†
Lipid lowering, n (%)	7 (5.6)	0	4 (8.0)	3 (6.2)	0.335	–	–
Antihypertensive, n (%)	70 (56.0)	16 (59.3)	24 (48.0)	30 (62.5)	0.327	–	–
Antidiabetic, n (%)	38 (30.4)	9 (33.3)	14 (28.0)	15 (31.2)	0.877	–	–
Benzodiazepines, n (%)	8 (6.4)	1 (3.7)	3 (6.0)	4 (8.3)	0.726	–	–
Glucose on admission, median (IQR), mmol/L	7.4 (6.0–9.4)	7.6 (6.1–8.6)	7.3 (6.0–9.1)	7.4 (6.0–9.7)	0.942	–	–
Premorbid mRS ≤2, n (%)	121 (96.8)	24 (88.9)	50 (100.0)	47 (97.9)	0.026	0.040*†	0.130*†
NIHSS on admission, median (IQR)	1.0 (0.0–3.0)	1.0 (0.5–4.0)	0.0 (0.0–1.0)	1.5 (1.0–3.2)	<0.001	0.001	>0.999
NIHSS at 24 hours, median (IQR)	0.0 (0.0–2.0)	1.0 (0.0–2.0)	0.0 (0.0–1.0)	1.0 (0.0–2.0)	0.001	<0.001	>0.999
Infection, n (%)	14 (11.2)	6 (22.2)	1 (2.0)	7 (14.6)	0.017	0.007*†	0.527*†
Pneumonia, n (%)	11 (8.8)	4 (14.8)	1 (2.0)	6 (12.5)	0.086	–	–
Urinary infection, n (%)	1 (0.8)	1 (3.7)	0	0	0.161	–	–
Skin infection, n (%)	1 (0.8)	1 (3.7)	0	0	0.161	–	–
Other infection, n (%)	1 (0.8)	0	0	1 (2.3)	0.396	–	–
Fever on admission, n (%)	3 (2.4)	2 (7.4)	0	1 (2.1)	0.126	–	–
White cell count, median (IQR)	6.7 (5.4–8.3)	6.2 (5.4–7.9)	6.8 (5.2–8.3)	6.8 (6.0–8.8)	0.434	–	–
Neutrophil, median (IQR)	4.5 (3.2–6.0)	3.9 (3.1–6.0)	4.3 (3.2–5.8)	4.8 (3.6–6.5)	0.366	–	–
Neutrophil to lymphocyte ratio, median (IQR), %	2.9 (2.0–5.1)	3.3 (2.2–5.3)	2.6 (2.0–3.7)	3.3 (2.0–5.2)	0.481	–	–

Continued



**Table 1** Continued

Variables	Overall (n=125)	PSR (n=27)	TIA (n=50)	SMs (n=48)	P value		
					Between groups	PSR vs TIA	PSR vs SMs TIA vs SMs
hsCRP, mean±SD, mg/L	4.2±6.0	6.1±7.6	3.0±4.6	4.5±6.1	0.083	-	-
Hyponatraemia, n (%)	1 (0.8)	1 (0.8)	1 (0.8)	1 (0.8)	0.452	-	-
Hypertnatraemia, n (%)	8 (6.6)	2 (7.4)	2 (4.3)	4 (8.5)	0.696	-	-
Hyperglycaemia, n (%)	16 (13.4)	4 (15.4)	8 (16.3)	4 (9.1)	0.563	-	-

\*A significance threshold of  $p<0.017$  was applied to assess the statistical significance of intergroup comparisons.

†Fisher exact test is used for analysis.

hsCRP, high-sensitivity C reactive protein; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; PSR, post-stroke recrudescence; SMs, stroke mimics; TIA, transient ischaemic attack.

To the best of our knowledge, this is the first prospective comparative study of PSR and SMs in the Chinese population. The prevalence rate of PSR in our study was 2.4%, which falls within the lower range of 9–10% reported in previous studies.<sup>5 6</sup> Possible reasons for these variations include stricter MRI-based investigations in our study compared with earlier studies that relied solely on CT. Additionally, a subset of patients with PSR triggered by infections, especially those accompanied by fever, initially sought care at nearby medical facilities during COVID-19, and racial differences. Consistent with previous findings,<sup>5</sup> our study revealed a higher likelihood of PSR among patients with a history of ischaemic stroke or intracerebral haemorrhage. Additionally, we observed that infection was a significant trigger for PSR but not for TIA. Furthermore, patients with PSR tended to exhibit higher levels of hsCRP compared with patients with TIA, although this difference did not reach statistical significance. A previous study reported a strong correlation between post-stroke infection and an elevated risk of mortality and disability.<sup>14</sup> However, the precise underlying mechanism remains unclear. Existing evidence suggests that PSR can be induced by an immunological response triggered by infection,<sup>15 16</sup> neurochemical mechanisms mediated by GABA<sub>A</sub> following midazolam administration<sup>17 18</sup> and alterations in neuronal excitability and transmission due to sodium–water imbalances.<sup>5</sup> Additionally, potential triggers for PSR or SMs include the use of benzodiazepine, elevated D-dimer levels, hyponatraemia, hypotension and exposure to stressors.<sup>5 6 19–22</sup> This discrepancy is likely attributed to variations in enrolled patient settings, disparities in healthcare and the limited sample size of the study.

We observed recurrences of various neurological deficits, including changes in consciousness, gaze, facial paralysis, motor arm or leg, ataxia, sensory, language, dysarthria and neglect. Sensory impairments and dysarthria were the predominant neurological impairments in patients with PSR and SMs. This study is consistent with a previous study that has described functional SMs as the most common cause of SMs.<sup>4</sup> However, a high proportion of migraine and seizure was not observed in this study, which could be attributed to factors such as differences in study population, variations in inclusion criteria and exclusion of patients with PSR. Interestingly, patients with TIA were more prone to experience ENI compared with those with PSR and SMs. Additionally, we found that neurological deficits in patients with PSR resolved within several days, and none of the patients developed intracerebral haemorrhage following tissue plasminogen activator. These findings support the view that withholding intravenous thrombolysis from patients with suspected acute stroke deficits was unwarranted, consistent with the finding reported by Topcuoglu *et al.*<sup>5</sup>

A previous study showed that WMH is a reliable imaging marker associated with mortality and cardiovascular risk in ischaemic stroke.<sup>23</sup> However, the relationship between WMH markers and PSR remains unclear. Furthermore,

**Table 2** WMH and lacunes distribution in patients with PSR, TIA and SMs

Variables	Overall (n=125)	PSR (n=27)	TIA (n=50)	SMs (n=48)	P value			
					Between groups	PSR vs TIA	PSR vs SMs	TIA vs SMs
Fazekas PVH					0.342			
0–1, n (%)	55 (44.0)	10 (37.0)	22 (44.0)	23 (47.9)	0.660	–	–	–
2, n (%)	49 (39.2)	9 (33.3)	22 (44.0)	18 (37.5)	0.628	–	–	–
3, n (%)	21 (16.8)	8 (29.6)	6 (12.0)	7 (14.6)	0.124	–	–	–
Fazekas DWMP					0.015			
0–1, n (%)	95 (76.0)	17 (63.0)	39 (78.0)	39 (81.2)	0.187	–	–	–
2, n (%)	24 (19.2)	9 (33.3)	11 (22.0)	4 (8.3)	0.025	0.279*	0.010*†	0.06*
3, n (%)	6 (4.8)	1 (3.7)	0	5 (10.4)	0.052	–	–	–
ARWMC, median (IQR)	5.0 (2.0–8.0)	5.0 (2.5–9.0)	5.0 (2.2–8.0)	5.0 (2.0–7.0)	0.491	–	–	–
Lacunes, n (%)	36 (28.8)	10 (37.0)	15 (30.0)	11 (22.9)	0.419	–	–	–

\*A significance threshold of  $p < 0.017$  was applied to assess the statistical significance of intergroup comparisons.

†Fisher exact test is used for analysis.

ARWMC, age-related white matter change; DWMP, deep white matter pathology; PSR, post-stroke recrudescence; PVH, periventricular hyperintensity; SMs, stroke mimics; TIA, transient ischaemic attack; WMH, white matter hyperintensity.

a prior study demonstrated that the mean volume of WMH was greater in patients with TIA and minor stroke compared with SMs.<sup>24</sup> A recent study with a sample of 2112 patients revealed significant variations in the WMH rating scale among patients with stroke, TIA and SMs.<sup>7</sup> Our study found patients with PSR had a higher incidence of grade 2 of Fazekas DWMP according to the Fazekas scale compared with those with SMs. Therefore, we postulate that WMH could potentially serve as a marker for distinguishing patients with PSR from those with SMs. However, this hypothesis will be confirmed in prospective studies.

This study revealed that patients with PSR had a lower likelihood of achieving functional independence at 3 months compared with patients with TIA. This could be attributed to the presence of premorbid neurological

deficits in patients with PSR. Furthermore, this study demonstrated that neurological function in patients with PSR was comparable with their premorbid state, indicating that symptoms can be relieved after removing the trigger. In our study, 87.2% of patients with SMs achieved functional independence at 3 months, which aligns with the findings of a report.<sup>4</sup> Additionally, there were no significant differences observed among patients with PSR, TIA and SMs regarding stroke recurrence, mortality, or the composite outcome of mortality and stroke recurrence.

Several limitations should be taken into consideration when interpreting our results. First, we have only included suspected patients presenting within 72 hours of symptom onset which might lead to potential selection bias. Second, we excluded patients who did not

**Table 3** Comparison of outcomes in patients with PSR, TIA and SMs

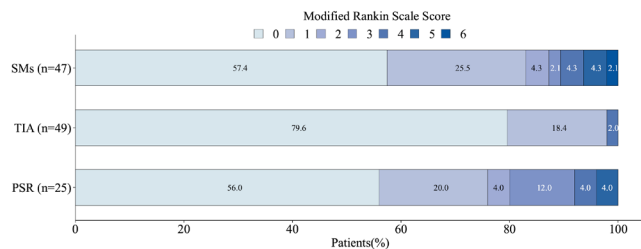
Variables	Overall (n=125)	PSR (n=27)	TIA (n=50)	SMs (n=48)	P value			
					Between groups	PSR vs TIA	PSR vs SMs	TIA vs SMs
Functional independence, n (%)*	109 (90.1)	20 (80.0)	48 (98.0)	41 (87.2)	0.036	0.015†‡	0.479†‡	0.057†‡
ENI, n (%)	90 (72)	16 (59.3)	49 (98.0)	25 (52.1)	<0.001	<0.001†	0.549†	<0.001†
Length of stay, mean±SD, days	9.6±4.9	10.6±5.8	8.2±3.6	10.5±5.3	0.033	0.054†	0.910†	0.016†
Mortality, n (%)*	1 (0.8)	0	0	1 (2.1)	0.446	–	–	–
Stroke recurrence, n (%)*	3 (2.4)	1 (3.7)	1 (2.0)	1 (2.1)	0.882	–	–	–
Composite outcomes of mortality and stroke recurrence, n (%)*	4 (3.2)	1 (3.7)	1 (2.0)	2 (4.2)	0.819	–	–	–

\*Of these individuals, two, one and one patients were lost to follow-up in patients with PSR, TIA and SM at 3 months, respectively.

†A significance threshold of  $p < 0.017$  was applied to assess the statistical significance of intergroup comparisons.

‡Fisher exact test is used for analysis.

ENI, early neurological improvement; PSR, post-stroke recrudescence; SMs, stroke mimics; TIA, transient ischaemic attack.



**Figure 2** Distribution of mRS scores at 3 months among patients with PSR, TIA and SMs. mRS, modified Rankin Scale; PSR, post-stroke recrudescence; SMs, stroke mimic; TIA, transient ischaemic attack.

underwent MRI scan which might underestimate the prevalence of PSR. Third, this is a single-centre study with a relatively small sample size; our findings needed to be validated in large-scale multicentre studies.

## CONCLUSIONS

PSR is not uncommon in patients presenting with stroke symptoms and can be distinguished from TIA and SMs based on a combination of clinical features and triggers in the Chinese population. The neurological deficits of patients with PSR often resolve within several days following the resolution of the trigger.

**Contributors** QL and M-JP—study conception and design. M-JP, J-LY, XH, LD, CC, X-NL, Z-QL and Z-JW—data collection. M-JP—writing (original draft). M-JP and J-LY—statistical analysis. All authors made the analysis and interpretation of the results. QL and PX—study supervision. QL—funding and writing (review and editing). QL accepts full responsibility for the work as guarantor and/or the conduct of the study, had access to the data and controlled the decision to publish.

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**Supplemental table 1.** Baseline characteristics in patients with PSR, TIA and SMs

Variables		Overall (n=125)	PSR (n=27)	TIA (n=50)	SMs (n=48)	P value			
						Between groups	PSR vs TIA	PSR vs SMs	TIA vs SMs
Systolic blood pressure on admission, median (IQR), mmHg		145.0 (130.0,161.0)	136.0 (125.5,164.5)	144.5 (134.5,156.5)	146.5 (130.8,162.5)	0.678	-	-	-
BMI, mean ± SD, Kg/m²		23.8±3.5	22.7±4.1	24.1±3.3	24.1±3.3	0.207	-	-	-
Abdominal circumference, mean±SD, cm		87.0±10.0	85.1±8.8	89.1±9.8	86.0±10.7	0.169	-	-	-
Serum urea nitrogen level, median (IQR), mmol/L		5.8 (4.2,7.4)	6.3 (3.9,8.1)	5.7 (4.6,7.0)	5.8 (4.1,7.3)	0.867	-	-	-
Creatinine level, median (IQR), mmol/L		70.0 (58.0,84.0)	71.0 (57.5,98.5)	71.5 (62.0,82.8)	63.5 (54.5,82.0)	0.276	-	-	-
Potassium level, mean ± SD, mmol/L		3.9±0.4	3.9±0.3	4.0±0.3	3.9±0.4	0.907	-	-	-
Serum sodium level ,mean ± SD, mmol/L		139.1±4.1	139.6±2.9	139.2±4.0	138.6±4.9	0.573	-	-	-
Serum uric acid level, mean ± SD, mmol/L		325.3±98.7	331.5±90.8	335.2±94.2	311.5±107.5	0.466	-	-	-
D-dimer, mean ± SD, mg/L		0.8±0.9	0.8±0.8	0.5±0.4	1.1±1.1	0.002	0.077*	0.235*	0.001*
Clinical symptoms on admission									
Consciousness, n (%)		20 (16.0)	6 (22.2)	4 (8.0)	10 (20.8)	0.136	-	-	-
Gaze, n (%)		3 (2.4)	1 (3.7)	1 (2.0)	1 (2.1)	0.882	-	-	-
Facial paralysis, n (%)		21 (16.8)	4 (14.8)	4 (8.0)	13 (27.1)	0.039	0.440*	0.223*	0.013*
Motor arm or leg, n (%)		24 (19.2)	7 (25.9)	5 (10.0)	12 (25.0)	0.102	-	-	-
Ataxia, n (%)		7 (5.6)	3 (11.1)	-	4 (8.3)	0.074	-	-	-

Sensory, n (%)	30 (24.0)	9 (33.3)	5 (10.0)	16 (33.3)	0.011	0.027*	>0.999*	0.005*
Language, n (%)	10 (8.0)	3 (11.1)	3 (6.0)	4 (8.3)	0.728	-	-	-
Dysarthria, n (%)	31 (24.8)	9 (33.3)	5 (10.0)	17 (35.4)	0.007	0.027*	0.856*	0.003*
Extinction-inattention, n (%)	3 (2.4)	0	0	3 (6.2)	0.085	-	-	-
Intravenous thrombolysis, n (%)	12 (9.6)	3 (11.1)	1 (2.0)	8 (16.7)	0.046	0.121*	0.736*	0.015*
Interval from index stroke to recrudescence, mean±SD, years	6.0±7.0	6.3±7.6	6.2±4.1	0.2 (-)	0.712	-	-	-
Unknown time of onset, n (%)	28 (22.4)	6 (22.2)	9 (18.0)	13 (27.1)	0.559	-	-	-

\*A significance threshold of  $P < 0.017$  was applied to assess the statistical significance of intergroup comparisons.

**Supplemental table 2.** Frequency of SMs categories

SMs categories	Number of patients	Percentage
Peripheral vertigo	8	16.7%
Peripheral neuropathy	4	8.3%
Cognitive decline	4	8.3%
Brain tumors	3	6.3%
Aneurysm	3	6.3%
Cervical spondylosis	3	6.3%
Sepsis-associated encephalopathy	3	6.3%
Cranial neuropathies	3	6.3%
Anxiety	2	4.2%
Seizure	2	4.2%
Migraine	2	4.2%
Hypokalemia	2	4.2%
Drugs	1	2.1%
Conversion disorders	1	2.1%
Subdural hemorrhage	1	2.1%
Hyperglycemia	1	2.1%
Parkinsonism	1	2.1%
Hypoglycemia	1	2.1%
Vitamin deficiency	1	2.1%
Lower extremity peripheral artery disease	1	2.1%
Cerebral amyloid angiopathy	1	2.1%
Total	48	