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ORIGINAL RESEARCH

Risk Factors for Post-Stroke Depression Following the Lifting of COVID-19 Restrictions

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Purpose: Research on post-stroke depression (PSD) following the lifting of coronavirus disease 2019 (COVID-19) restrictions remains sparse. This study aimed to investigate the factors associated with PSD after the easing of COVID-19 restriction measures.

Patients and Methods: This cross-sectional study was conducted with 947 stroke patients (cerebral hemorrhage and cerebral infarction) meeting the inclusion criteria. Participants completed a demographic questionnaire and the Patient Health Questionnaire-9 (PHQ-9). Additionally, data were collected on C-reactive protein (CRP), homocysteine (Hcy), modified Rankin Scale (mRS), stroke site, National Institutes of Health Stroke Scale (NIHSS), thyroid-stimulating hormone (TSH), and the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification. This study assessed correlations between these indices and PSD.

Results: Stroke patients with a PHQ-9 score ≥5 were identified as having PSD, with a prevalence rate of 14.15%. No significant correlation was found between previous COVID-19 infection and PSD. However, multiple regression analysis revealed associations between PSD and the following factors: TSH (OR: 0.87, 95% CI: 0.76–1), CRP levels (OR: 1.01, 95% CI: 1–1.02), family history of stroke (OR: 4.25, 95% CI: 1.66–10.88), migraine history (OR: 8.63, 95% CI: 2.49–29.85), and shorter sleep duration (OR: 0.6, 95% CI: 0.51–0.71) (all $P < 0.05$).

Conclusion: CRP, family history of stroke, migraine, sleep duration, and TSH are identified as independent risk factors for PSD following the lifting of COVID-19 restrictions.

Keywords: COVID-19, stroke, depression, thyroid-stimulating hormone

Introduction

Coronavirus disease 2019 (COVID-19), a novel coronavirus pneumonia, is a widespread infectious disease posing a significant global health threat. By March 2022, over 445 million infections and six million fatalities had been reported worldwide due to COVID-19.[1] Following the initial outbreak in December 2019, China implemented a dynamic zero-COVID policy with stringent defense measures. Considering the gradual decline in the pathogenicity of the novel coronavirus, the comprehensive team of the Joint prevention and control Mechanism of The State Council issued the "New Ten" on December 7, lifting China's strict COVID-19 prevention and control measures.[2] This led to a decline in positive cases after peaking at 6.94 million on December 22, 2022.[3]

Stroke remains a leading cause of disability and the second-highest contributor to global mortality.[4] According to the China Stroke High-risk Population Screening and Intervention Program, approximately 17.8 million adults in China are projected to experience a stroke in 2020 (95% confidence interval CI: 17.6–18 million), with 2.3 million fatalities (95% CI: 2.2–2.4 million). Additionally, about 12.5% of stroke survivors (95% CI: 12.4–12.5%) are expected to be disabled, resulting in 2.2 million stroke-related disabilities by 2025.[5] In 2020, the estimated stroke prevalence, incidence, and

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mortality rates in China were 2.6%, 505.2 per 100,000 person-years, and 343.4 per 100,000 person-years, respectively.[6] Stroke has emerged as a leading cause of death and disability in China, significantly impacting public health.

Post-stroke depression (PSD) is the most common and severe neuropsychiatric complication following a -stroke,[7] leading to increased mortality, more severe cognitive deficits, and a poorer quality of life compared to stroke survivors without PSD.[8,9] Risk factors for PSD encompass sleep-wake disorders,[10] C-reactive protein (CRP),[11] thyroid-stimulating hormone (TSH),[12] recurrent stroke[13,] diabetes mellitus, and cognitive function[14.] Sleep disruptions were prevalent during the COVID-19 pandemic,[15] with COVID-19 infections causing increased CRP[16] and decreased TSH levels.[17] Additionally, the prevalence and burden of depression surged during the pandemic.[18] Post-COVID-19 infection, patients often face sleep disturbances, cognitive impairments,[19] and heightened risks of diabetes and stroke.[20] Multiple studies have reported elevated depression rates among healthcare workers and the general population following the lifting of pandemic restrictions.[21,22] However, research on depression in stroke patients post-COVID-19 restrictions remains limited. Thus, further investigation is needed to explore the correlation between COVID-19 and PSD, as well as the associated risk factors for PSD after the lifting of COVID-19 measures.

Materials and Methods

Experimental Strategy and Patients

A cross-sectional study was employed to identify determinants linked to PSD following the lifting of control measures, aiming to establish a foundation for early detection and intervention. This research included all cerebral stroke patients (cerebral hemorrhage and infarction) admitted to the Neurology Department at the First People's Hospital of Changde City from March to September 2023 (Figure 1).

Patient inclusion criteria were as follows:

(1) Diagnosed with cerebral infarction or hemorrhage per the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10).

(2) Hemorrhage or infarction location identified via computed tomography (CT) or magnetic resonance imaging (MRI).

Exclusion criteria included:

(1) History of psychiatric disorders.

(2) Inability to complete the questionnaire due to consciousness disturbance or language impairment.

(3) Refusal to participate in the study.

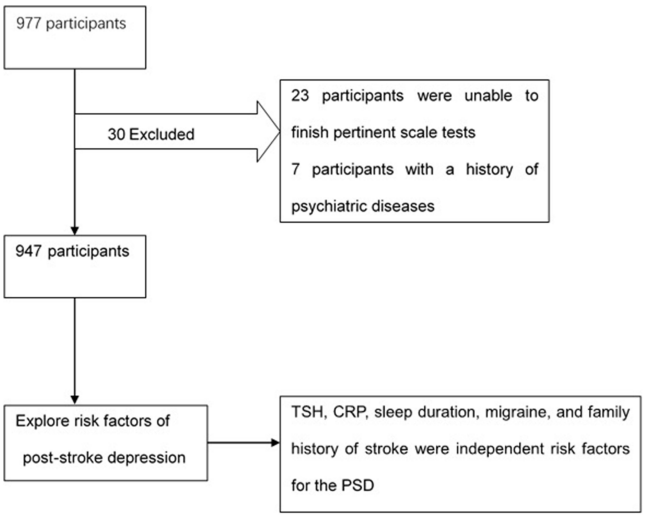

Figure 1 Flow chart of study.

This study received approval from the relevant ethics committee. Written informed consent was obtained from all participants or their families, with family members signing on behalf of stroke patients who were unable to do so. The first page of the questionnaire detailed the research's purpose and significance. Participants were also informed of their right to withdraw from the survey at any time.

Measures

Each patient was subjected to the following evaluations:

(1) Laboratory analysis: Upon admission, standard blood tests were performed to measure serum levels of TSH, homocysteine (Hcy), and CRP.

(2) Stroke-specific assessments: These included evaluating modified Rankin Scale (mRS) and National Institutes of Health Stroke Scale (NIHSS) scores, determining bleeding volume and stroke location, and categorizing the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) subtypes based on comprehensive imaging and clinical data.

(3) Questionnaire Survey: Questionnaires were developed using the Questionnaire Star platform, with professionally trained doctors administering face-to-face interviews during patient hospitalization. Responses were directly provided by the patients. The survey collected data on variables including sex, age, height, weight, employment status, marital status, educational attainment, presence of post-stroke pain, smoking and alcohol habits, COVID-19 infections, past medical history, and family history of psychiatric disorders and stroke. Additionally, assessments were conducted using the Patient Health Questionnaire-9 (PHQ-9).

Standard Blood Tests

TSH estimation: Fasting blood samples were collected at 6 a.m. the day after admission. TSH levels were measured using a standardized radioimmunoassay kit, with a reference range of 0.35–4.94 μIU/mL.

Hcy estimation: Fasting blood samples were collected at 6 a.m. the day after admission. Serum Hcy levels were evaluated using an enzyme cycling method, with a reference range of 0–15 μmol/L.

CRP estimation: Fasting blood samples were collected at 6 a.m. the day after admission. Serum CRP levels were quantified through immunoturbidimetry, with a detection range of 0–10 mg/L.

Stroke-Related Assessments

NIHSS: The NIHSS score, ranging from 0 to 42, was utilized to evaluate stroke severity, with 0 indicating normal function and higher scores denoting greater impairment.[23] Assessments were conducted by neurologists with extensive training and systematic expertise.

mRS: The mRS, a 5-point scale, measured residual disability in stroke patients, where 0 denoted no symptoms and 5 indicated severe disability. The scale's validity and both interobserver and intraobserver reliability in assessing the required assistance for varying degrees of autonomy in stroke patients were well-established.[23] Scoring was performed by neurologists with comprehensive training and systematic experience.

The TOAST classification system: TOAST classification system categorized cerebral infarction by etiology, comprising the following categories:

(1) Large-artery atherosclerosis,

(2) Cardioembolism,

(3) Small-artery occlusion (lacune),

(4) Stroke of other determined etiology,

(5) Stroke of undetermined etiology (Table 1).

A neurologist, with systematic training and extensive experience, assigned the classification diagnosis based on the patient's relevant examination results.[24]

Questionnaire Survey

PHQ-9: Depression status was assessed using the Chinese version of the PHQ-9, a 9-item scale measuring depressive symptom severity from 0 (complete absence of symptoms) to 3 (symptoms nearly every day). The total score ranged

Table 1 Sociodemographic Characteristics of Stroke Patients

Variables	Category	Mean (SD)
Sex	Male	608 (64.2%)
	Female	339 (35.8%)
Age		64.5 (15)
Marital Status	Married	941 (99.40)
	Dissociation	3 (0.30)
	Singlehood	2 (0.20)
	Widowhood	1 (0.10)
BMI		24.43 ± 5.11
Height		163 (11)
Weight		65 (16)
Employment status	Employment	140 (14.8%)
	Retirement	300 (31.7%)
	Unemployed	507 (53.5%)
Educational status	Junior high school and below	763 (80.7%)
	High school or technical	125 (13.2%)
	College or university	58 (6.1%)

Abbreviation: BMI, Body Mass Index.

from 0 to 27, with higher scores indicating greater depression severity. A threshold score of 5, established by previous studies,[25–27] identified depression (score ≥5). The PHQ-9 scores were categorized as follows:

no depression (0–4),
mild depression (5–9),
moderate depression (10–14), and
severe depression (≥15).

In this study, Cronbach's alpha was 0.722, and the KMO measure of sampling adequacy was 0.790.

Statistical Analyses

The IBM SPSS software package (version 20.0) conducted all data analyses. Continuous numerical variables with a normal distribution were presented as mean ± standard deviation, while non-normally distributed quantitative and rank data were shown as median (interquartile range). Normally distributed data were compared using an independent sample *t*-test, and the rank sum test compared non-normally distributed quantitative data. Count data were expressed as quantity (%). Differences were assessed using the chi-square test. Additionally, binary logistic regression identified independent risk factors for depression. Statistical significance was defined as $P < 0.05$.

Results

Clinical and Socio-Demographic Features

During the 6-month study period, 947 patients of both genders were enrolled. Clinical and demographic data, along with PHQ-9 scores, were presented in Tables 1–4. The average PHQ-9 score was 2.06 ± 2.64. Additionally, 134 patients

Table 2 Stroke-Related Information of Stroke Patients

Variables	Category	Mean (SD)
Type of stroke	Ischemic stroke	860 (90.8%)
	Hemorrhagic stroke	87 (9.2%)
Lesion site	brainstem	158 (16.7%)
	Bilateral cerebral hemispheres	105 (11.1%)
	Parencephalon	40 (4.2%)
	Right cerebral hemisphere	320 (33.8%)
	Left cerebral hemisphere	323 (34.1%)
TOAST Classification	Large vessel atherosclerosis	590 (68.8%)
	Small vessel occlusion	203 (23.7%)
	Cardio-embolic source	45 (5.2%)
	Undetermined etiologies	20 (2.3%)
Time since stroke	>6 months	14 (1.5%)
	>7 days ≤ 6 months	535 (56.5%)
	≤ 7 days	398 (42%)
Feel pain after a stroke	Yes	79 (8.3%)
	No	868 (91.7%)
Pre-stroke sleep duration	<5 h	177 (18.7%)
	5–6 h	135 (14.3%)
Amount of bleeding		4 (5.75)
NIHSS score		2 (8.75)
MRS score		4 (2)
PHQ-9		2.06 ± 2.64

Abbreviations: NIHSS, National Institute of Health Stroke Scale; mRS, modified Rankin scale; pre-stroke sleep duration, sleep duration 1 month before stroke; PHQ-9, Patient Health Questionnaire-9.

Table 3 Past Medical History of Stroke Patients

Variables	Category	Mean (SD)
History of Smoking	No	596 (62.9%)
	Current smoker	307 (32.4%)
	Previous smoker	44 (4.6%)
History of alcohol drinking	No	805 (85%)
	Currently drinking	115 (12.1%)
	Previously drinking	27 (2.9%)

(*Continued*)

Table 3 (Continued).

Variables	Category	Mean (SD)
Infected with COVID-19	Previously infecting	654 (69.5%)
	Currently infecting	0 (0%)
	No	287 (30.5%)
History of hypertension		686 (72.4%)
History of hyperlipidemia		113 (11.9%)
History of diabetes		283 (29.9%)
History of heart disease		191 (20.2%)
History of stroke		219 (23.1%)
History of chronic nephrosis		134 (14.1%)
History of hemicrania		16 (1.7%)
Family history of mental disorders		2 (0.2%)
Family history of stroke		29 (3.1%)

Table 4 Blood Test Results of Stroke Patients

Variables	Mean (SD)
Homocysteine	14.405 (6.71)
TSH	2.0925 (2.22)
CRP	3.25 (7.33)

Abbreviations: TSH, Thyroid-stimulating hormone; CRP, C-reactive protein.

(14.15%) were diagnosed with PSD. Specifically, mild depression was observed in 116 patients (12.25%), moderate depression in 14 patients (1.48%), and severe depression in 4 patients (0.42%).

Factors Associated with PSD

Patients were categorized based on their PHQ-9 scores to examine post-stroke depression-related factors. Those with PHQ-9 scores below 5 were placed in the non-depressed group, while individuals with scores of 5 or higher were classified as depressed. Tables 5–8 detailed the specific parameters of patients in both groups.

As shown in Tables 5–8, several significant differences emerged between the two groups. The depressed group had a lower TSH level (median quartile: 1.64 [1.71], P = 0.01) and elevated CRP concentration (3.91 [10.08], P = 0.04) compared to the non-depressed group. Post-stroke pain was more prevalent in the depressed group (P = 0.01). Regarding sleep duration, a higher proportion of the depressed group reported sleeping less than 5 hours (P = 0.01), and fewer individuals slept more than 7 hours (P = 0.01) compared to the non-depressed group. Additionally, a family history of stroke (P = 0.01), heart disease (P = 0.04), hyperlipidemia (P = 0.02), and migraine (P = 0.01) was more common in the depressed group.

Table 5 Sociodemographic Comparison Between Depressed and Non-Depressed Patients

Category	No Depression	Depression	T/Z/χ2	P
Sex			2.43	0.12
Male	530 (65.2)	78 (58.2)		
Female	283 (34.8)	56 (41.8)		
Age (years)	65.91 ± 10.91	65.1 ± 11.26	0.79	0.42
BMI	24.48 ± 5.02	24.43 ± 5.61	0.07	0.94
Weight	65 (14)	60 (18.75)	−1.86	0.06
Height	162.79 ± 7.27	162.55 ± 7.97	0.25	0.80
Employment status			2.79	0.25
Employed	114 (14)	26 (19.4)		
Retirement	258 (31.7)	42 (31.3)		
Unemployed	441 (54.2)	66 (49.3)		
Educational Status			1.08	0.58
Junior high school and below	660 (81.2)	103 (77.4)		
College or university	49 (6)	9 (6.8)		
High school or technical secondary school	104 (12.8)	21 (15.8)		

Notes: T, Statistics of independent sample *t* test; Z, Statistics of the rank sum test; χ2, Chi-square test statistics.
Abbreviation: BMI, Body Mass Index.

Table 6 Stroke-Related Information Comparison Between Depressed and Non-Depressed Patients

Category	No Depression	Depression	χ2	P
Type of stroke			0.75	0.38
Ischemic stroke	741 (91.1)	119 (88.8)		
Hemorrhagic stroke	72 (8.9)	15 (11.2)		
Lesion site			2.12	0.71
Bilateral cerebral hemispheres	131 (16.1)	27 (20.1)		
Parencephalon	88 (10.8)	17 (12.7)		
Right cerebral hemisphere	35 (4.3)	5 (3.7)		
Left cerebral hemisphere	279 (34.4)	41 (30.6)		
Bilateral cerebral hemispheres	279 (34.4)	44 (32.8)		
Type of stroke			0.75	0.38
Ischemic stroke	741 (91.1)	119 (88.8)		
Hemorrhagic stroke	72 (8.9)	15 (11.2)		

(*Continued*)

Table 6 (Continued).

Category	No Depression	Depression	χ2	P
TOAST Classification			7.23	0.06
Large vessel atherosclerosis	498 (67.4)	92 (77.3)		
Small vessel occlusion	186 (25.2)	17 (14.3)		
Cardio-embolic source	39 (5.3)	6 (5)		
Undetermined etiologies	16 (2.2)	4 (3.4)		
Time since stroke			0.74	0.69
>6 months	11 (1.4)	3 (2.2)		
>7 days ≤ 6 months	458 (56.3)	77 (57.5)		
≤7 days	344 (42.3)	54 (40.3)		
Amount of bleeding (mL)	5 (8.25)	3.5 (6)	−0.53	0.59
NIHSS score	2 (3)	2 (4)	1.09	0.28*
mRS score	2 (2)	2 (1)	1.48	0.14
Pre-stroke sleep duration			40.41	0.01*
<5 h	128 (15.7)	49 (36.6)		
5–6 h	115 (14.1)	20 (14.9)		
6–7 h	58 (7.1)	14 (10.4)		
>7 h	512 (63)	51 (38.1)		
Feel pain after a stroke			6.95	0.01*
Yes	60 (7.4)	19 (14.2)		
No	753 (92.6)	115 (85.8)		

Notes: pre-stroke sleep duration, sleep duration 1 month before stroke; χ2, Chi-square test statistics; *$P < 0.05$.
Abbreviations: NIHSS, National Institute of Health Stroke Scale; mRS, modified Rankin scale.

Table 7 Past Medical History Comparison Between Depressed and Non-Depressed Patients

Category	No Depression	Depression	χ2	P
History of Smoking			0.32	0.85
No	510 (62.7)	86 (64.2)		
Current smoker	264 (32.5)	43 (32.1)		
Previous smoker	39 (4.8)	5 (3.7)		
History of alcohol drinking			2.23	0.33
No	696 (62.7)	109 (64.2)		
Currently drinking	96 (32.5)	19 (32.1)		
Previously drinking	21 (4.8)	6 (3.7)		

(*Continued*)

Table 7 (Continued).

Category	No Depression	Depression	χ2	P
Infected with COVID-19			2.71	0.09
Previously infected	569 (70.5)	85 (63.4)		
No	238 (29.5)	49 (36.6)		
History of hypertension			1.53	0.22
No	230 (28.3)	31 (23.1)		
Yes	583 (71.7)	103 (76.9)		
History of hyperlipidemia			5.31	0.02*
No	724 (89.1)	110 (82.1)		
Yes	89 (10.9)	24 (17.9)		
History of diabetes			0.65	0.42
No	574 (70.6)	90 (67.2)		
Yes	239 (29.4)	44 (32.8)		
History of heart disease			4.35	0.04*
No	658 (80.9)	98 (73.1)		
Yes	155 (19.1)	36 (26.9)		
History of stroke			1.21	0.27
No	620 (76.3)	108 (80.6)		
Yes	193 (23.7)	26 (19.4)		
History of chronic nephrosis			3.54	0.06
No	705 (86.7)	108 (80.6)		
Yes	108 (13.3)	26 (19.4)		
History of hemicrania			20.35	0.01*
No	806 (99.1)	125 (93.3)		
Yes	7 (0.9)	9 (6.7)		
Family history of mental disorders			-	0.26
No	812 (99.9)	133 (99.3)		
Yes	1 (0.1)	1 (0.7)		
Family history of stroke			11.98	0.01*
No	795 (97.8)	123 (91.8)		
Yes	18 (2.2)	11 (8.2)		

Notes: χ2, Chi-square test statistics; *$P < 0.05$.

Additional statistical analysis using univariate and multivariate logistic regression identified elevated TSH (OR: 0.87, 95% CI: 0.76–1) and CRP levels (OR: 1.01, 95% CI: 1–1.02), a family history of stroke (OR: 4.25, 95% CI: 1.66–10.88), migraine history (OR: 8.63, 95% CI: 2.49–29.85), and shorter sleep duration (OR: 0.6, 95% CI: 0.51–0.71) as independent risk factors for PSD (Table 9).

Table 8 Blood Test Results Comparison Between Depressed and Non-Depressed Patients

Category	No Depression	Depression	Z	P
Homocysteine	14.17 (6.8)	13.5 (6.88)	−1.19	0.24
TSH	2.04 (1.96)	1.64 (1.71)	−2.95	0.01*
CRP	2.9 (6.88)	3.91 (10.08)	2.07	0.04*

Notes: Z, Statistics of the rank sum test; *$P < 0.05$.
Abbreviations: TSH, Thyroid-stimulating hormone; CRP, C-reactive protein.

Table 9 Univariate and Multivariate Logistic Regression Results

Depression	Single-Factor Regression		Multiple-Factor Regression	
	OR (95 CI%)	*P*	*OR (95 CI%)*	*P*
TSH	0.9 (0.8, 1)	0.06	0.87 (0.76, 1)	0.04*
CRP	1.01 (1, 1.02)	0.01	1.01 (1, 1.02)	0.01*
Feel pain after a stroke	0.48 (0.28, 0.84)	0.01	0.75 (0.38, 1.46)	0.39
Pre-stroke sleep duration	0.66 (0.57, 0.76)	0.01	0.6 (0.51, 0.71)	0.01*
History of hyperlipidemia	1.78 (1.08, 2.91)	0.02	1.46 (0.79, 2.72)	0.23
History of heart disease	1.56 (1.02, 2.37)	0.04	1.39 (0.84, 2.3)	0.2
History of hemicrania	8.29 (3.03, 22.66)	0.01	8.63 (2.49, 29.85)	0.01*
Family history of stroke	3.95 (1.82, 8.56)	0.01	4.25 (1.66, 10.88)	0.01*

Notes: pre-stroke sleep duration, sleep duration 1 month before stroke; *$P < 0.05$.

Discussion

This cross-sectional study registered 947 stroke patients to investigate factors influencing PSD following the removal of COVID-19 control measures.

The PSD prevalence rate was 14.15%. No significant correlation was found between previous COVID-19 infection and PSD ($P = 0.09$). Independent risk factors for PSD included a family history of stroke, CRP, TSH, migraine, and sleep duration.

This research identified a connection between lower TSH levels and PSD, consistent with previous findings.[28] Recent studies have also shown a positive correlation between low TSH levels and depression.[26,29,30] The association between TSH and PSD might be due to lower serum TSH levels, which increase the basal metabolic rate and the production of reactive oxygen species and free radicals, ultimately causing neurocytotoxicity.[31] Additionally, higher energy and oxygen demands could impair ischemic tolerance.[32] Thyroid hormone regulation has been suggested to play a role in nerve repair.[33] Conversely, a prior study reported significantly higher serum TSH levels in patients with lacunar stroke and PSD compared to those without PSD.[34] Other research has found no association between TSH levels and depression.[35] These discrepancies may stem from variations in study populations, stroke onset times, and TSH grouping. Consequently, our study included a large sample size with patients experiencing different durations and severities of cerebral infarction and hemorrhage. The TSH level was analyzed quantitatively rather than categorically. Furthermore, the association between TSH and PSD persisted even after adjusting for confounding variables such as stroke severity and onset time, suggesting that TSH serves as a predictor of PSD. Further research into the predictive model of TSH and PSD could enhance early clinical diagnosis of PSD.

This study identified a correlation between higher CRP concentrations and PSD, aligning with a meta-analysis that consistently demonstrated a significant association between elevated CRP levels and PSD.[11] The relationship between

depression and inflammatory markers is complex, involving autoimmune responses to damaging molecules, cellular sensitivity to toxic peptides, reduced omega-3 and antioxidant levels, psychological stressors, and oxidative and nitrosative stress.[36–38] Thus, further research into the predictive value of CRP for PSD is necessary to enable early clinical intervention for PSD.

Prior research indicates that short sleep duration preceding a stroke may independently increase the risk of PSD,[39] consistent with the current study's findings. Additionally, total sleep time under 6 hours predicts PSD.[40] A randomized controlled trial demonstrated that improving sleep quality can alleviate depressive symptoms.[41] Sleep disorders may lead to neuroendocrine and circadian disruptions, heightening depression risk.[42] Stroke-induced depression might arise in high-risk individuals through mechanisms such as hypoperfusion, inflammation, and inhibited neurogenesis.[14] Attention to the sleep conditions of stroke patients is therefore essential, and early interventions are necessary to reduce PSD incidence.

The study identified a correlation between migraine history and PSD. Previous research on the relationship between migraine and PSD is limited, with only a few studies indicating a link between migraine and depression.[43,44] This correlation may stem from the profound impact of migraines on patients' lives, including impaired cognitive function at work and in social activities such as relationships. Migraine sufferers may struggle to participate in activities, leading to feelings of guilt, isolation, and despair.[44,45] Additionally, depression and migraine share numerous pathological mechanisms, involving central nervous system morphology and dysfunction, neurotransmitter and receptor systems, hormonal regulation, neuroinflammation, environmental factors, genetic predisposition, personality, and temperament.[46] Consequently, patients with a history of stroke and migraine should be closely monitored due to their increased risk of developing PSD.

Lastly, depression correlated with a family history of stroke, likely due to the high morbidity, mortality, and recurrence rates of stroke. The increased caregiver burden among family members of stroke patients significantly elevates anxiety and depression rates within this group.[47] Additionally, a family history of stroke can impose economic and personal pressures on patients, heightening their vulnerability to depression. Consequently, patients with a family history of stroke require extra care and support.

This research has several limitations. First, its cross-sectional design complicates establishing a causal relationship between relevant factors and PSD. Second, a longitudinal cohort study is required to assess the long-term effects of COVID-19 infection on PSD. Third, the study sample has a limited incidence of cerebral hemorrhage. Finally, using scale scores as the sole diagnostic criteria for PSD may introduce bias. Therefore, further research is needed to investigate the correlation between PSD and COVID-19, identify predictors for PSD, and explore related pathogenic factors.

Conclusion

This study demonstrates that TSH, CRP, sleep duration, migraine, and a family history of stroke are independent risk factors for PSD following the lifting of COVID-19 restrictions.

Abbreviations

COVID-19, Coronavirus disease 2019; PSD, Post-stroke depression; ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th Revision; CT, computed tomography; MRI, magnetic resonance imaging; PHQ-9, The Patient Health Questionnaire - 9; GAD-7, The Generalized Anxiety Disorder-7; mRS, Modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; TOAST, the Trial of ORG 10172 in Acute Stroke Treatment; TSH, Thyroid-stimulating hormone; CRP, C-reactive protein; Hcy, homocysteine.

Ethics Approval and Informed Consent

The study protocol adhered to the ethical guidelines of the Declaration of Helsinki. Approval for this study was obtained from the Ethics Committee of the First People's Hospital of Changde City (YX-2023-078-01).

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests in this work.

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