

Special Review



Factors Associated With Post-Stroke Cognitive Impairment: A Narrative Review

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HIGHLIGHTS

- Many risk factors for post-stroke cognitive impairment (PSCI) can be identified.
- Comprehensive assessments should be conducted in patients with risk factors for PSCI.
- Various cognitive rehabilitation programs will be need for patients with PSCI.

Special Review



Factors Associated With Post-Stroke Cognitive Impairment: A Narrative Review

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ABSTRACT

Post-stroke cognitive impairment (PSCI) is a common and significant disorder affecting a considerable proportion of stroke patients. PSCI is a known factor that increases the risk of mortality, dependency, and institutionalization in stroke patients. The early prediction of PSCI and the implementation of cognitive rehabilitation could enhance the quality of life of stroke patients and reduce the burden on their families. It is therefore imperative to identify risk factors for PSCIs in the early stages of stroke and to implement early cognitive rehabilitation with an appropriate prognosis. A number of risk factors for PSCI can be identified in patient characteristics, clinical findings, and imaging findings. It is unfortunate that the majority of factors associated with PSCI are non-modifiable. However, the only modifiable factor that can be controlled is the management of stroke risk factors for secondary prevention. Further research is needed to elucidate the potential benefits of various cognitive rehabilitation programs for the prevention and improvement of PSCI.

Keywords: Stroke; Cognitive Impairment; Rehabilitation; Prognosis

INTRODUCTION

Post-stroke cognitive impairment (PSCI) is a prevalent disorder in stroke patients [1]. PSCI is a cognitive impairment that encompasses diminished functionality in specific cognitive domains, such as aphasia or memory impairment that is linked to particular brain regions, and strategic dementia that occurs in the hippocampus, thalamus, and pivotal cortical regions. Moreover, PSCI presents with a wide range of symptoms, many of which are associated with specific brain regions and overlap with those caused by diffuse vascular brain injury. Previous studies have estimated the prevalence of PSCI in stroke survivors to be between 60 and 75% [2]. A significant cognitive impairment was observed in 31.1% of stroke patients in the Korean Stroke Cohort for Functioning and Rehabilitation (KOSCO) longitudinal study six months after the stroke [3]. It is established that PSCI not only causes damage in itself, but also has a negative impact on overall outcomes in stroke patients.

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Conflict of Interest

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A recent review article suggests that cognitive function, when considered as a whole, demonstrated a significant medium-level association with basic activities of daily living (ADL), instrumental ADLs, and participation outcomes [2]. Further analysis of the same review article revealed that these adverse effects of PSCI remained consistent regardless of the sample size or the follow-up period of previous studies. In another population-based study, a decline in cognitive function by 10% or more between seven days and three months was associated with an approximate two-fold increased risk of mortality, dependency, and institutionalization after one year, compared to stable cognitive function [4].

A number of risk factors have been identified as contributing to PSCI. However, many of these risk factors are correlated with each other, which presents a challenge in their clinical application. The early prediction of PSCI and the implementation of cognitive rehabilitation could enhance the quality of life of stroke patients and reduce the burden on their families. Therefore, it is crucial to identify risk factors for PSCIs in the early stroke phase and to apply early cognitive rehabilitation with an appropriate prognosis.

The present review offers a summary of the factors associated with PSCI. This review might provide a foundation for predicting the occurrence of PSCI in the early stroke phase and for applying appropriate cognitive rehabilitation.

ASSOCIATED FACTORS WITH PSCI

Demographic characteristics

It is established that advancing age is a risk factor for cognitive decline in general [5]. Similarly, in the context of stroke, older age is linked to accelerated cognitive decline following the event [6]. A study demonstrated that stroke survivors with lower levels of education exhibited a 1.8-fold increased likelihood of developing poststroke dementia. Individuals who have experienced a stroke and have attained a higher level of education are better able to compensate for vascular brain injury, thereby maintaining a higher level of functioning for a longer period of time [7]. Recent systematic review [8] provided evidence to support an inverse association between higher levels of education and rates of dementia. In meta-analysis, the full model demonstrated a statistically significant effect of covariates ($QM(5)=144.48$ ($p < 0.01$), indicating a notable explanatory loading of the cognitive reserve proxy on diagnosis outcome [8]. It is well established that lower educational attainment is significant risk factors for PSCI. As society progresses at a rapid pace, it is well-established that age and educational level are correlated in Korea. In 2022, approximately 53% of the Korean population aged between 25 and 64 years old had completed tertiary education, while approximately 38.4% of the adult population had completed upper secondary education [9]. Accordingly, the concept of cognitive reserve has been proposed in Alzheimer's disease as a combination of age and educational level [10]. The theory of cognitive reserve describes individual differences in susceptibility to both age-related brain changes and pathologic changes, such as those associated with stroke. The KOSCO study demonstrated that lower cognitive reserve, specifically lower premorbid education and occupation, was associated with severe cognitive dysfunction and an increased risk of cognitive impairment both immediately and over the following 30 months after the stroke [11].

Another potential demographic risk factor for PSCI is sex differences. In general, women exhibit a higher lifetime prevalence of dementia [12]. A study conducted in 2020 examined

sex differences in post-stroke cognitive outcomes [13]. The findings indicated that women exhibited poorer cognitive outcomes three months after a stroke. In another recent study, male post-stroke patients are at higher risk for verbal memory impairment, while females are at higher risk of impairment in language, attention, and executive function [14]. Interestingly, the study recommended that caution should be advised when cognitive screening tools are employed for the identification of PSCI in one sex, given that the Mini Mental Status Examination exhibits higher sensitivity but lower specificity for women. The KOSCO study also showed that post-stroke cognitive outcomes were inferior in women, even after adjusting for confounding variables since there were significant differences in mean age and education level between men and women [15]. Therefore, it is important to understand the sex differences in PSCI to refine standard guidelines of post-stroke care and better address the unique needs of certain populations at higher risk.

APOE- ϵ 4 has been firmly established as the most significant genetic risk factor for Alzheimer's disease. It is linked to elevated levels of amyloid deposits and an earlier age of onset [16,17]. Genetic variants in APOE are also linked to small vessel disease (SVD) markers and cerebral amyloid angiopathy [18]. However, the Oxford vascular study cohort observed that ϵ 4 heterozygosity was not associated with an elevated risk of PSCI [19], while other studies have proposed a substantial role in the development of PSCI [20,21], thus the correlation between genetic variation and PSCI remains inconclusive.

Stroke characteristics

It is established that severe strokes accelerate the onset of dementia by 25 years, mild strokes by 4 years, and transient ischemic attacks (TIAs) by 2 years. This indicates that cognitive functions that are widely distributed across cortical areas, such as memory and attention, are less susceptible to focal insults [1]. The location of the stroke is a significant factor in determining the cognitive function. A stroke occurring in a cognitive-related area, such as the frontal lobes, caudate, thalamus, fornix, or medial temporal lobe, has the potential to result in PSCI. With regard to the location of the stroke, there is a heightened risk associated with lesions in the left hemisphere (odds ratio [OR], 1.4; 95% confidence interval [CI], 1.1–1.7; $p = 0.02$; 95% CI, 1.1–1.7; $p = 0.02$) [7]. It is postulated that this is due to the fact that language is an integral component of numerous ADL, and that left hemisphere strokes resulting in aphasia may serve to lower the threshold for diagnosing dementia. In instances of multiple strokes, our findings indicate that cognitive decline occurs when the strokes occur in disparate locations, as opposed to when they occur repeatedly in the same location. Additionally, multiple infarctions frequently result in a progressive course of cognitive disorders, ultimately leading to vascular dementia. A long-term follow-up study conducted in Norway revealed that, in a multivariable logistic regression analysis, lower age and lower medial temporal lobe atrophy (MTLA) grade on magnetic resonance imaging (MRI) at 12 months were independently associated with a favorable outcome. The adjusted OR (95% CI) was 0.94 (0.86–0.92) for age and 0.55 (0.35–0.85) for MTLA grade, respectively [22]. In a separate cohort study of intracerebral hemorrhage, multiple logistic regression analysis revealed a significant positive correlation between the risk of early periventricular and subcortical white matter injury and dominant-hemisphere hemorrhage (OR, 8.845; 95% CI, 3.347–23.371; $p < 0.001$). Additionally, mean corpuscular volume was identified as a contributing factor (OR, 1.079; 95% CI, 1.002–1.162; $p = 0.043$), as was admission systolic blood pressure (OR, 1.021; 95% CI, 1.005–1.038; $p = 0.012$) [23].

The distinction between ischemic and hemorrhagic stroke in PSCI remains inconclusive. A recent Oxford Vascular study revealed that the risk of poststroke dementia was 2.5 times higher for cerebral infarction than for cerebral hemorrhage [24]. However, these risk differences were less pronounced after adjusting for stroke severity and were no longer statistically significant, except for TIA. The results indicate that the discrepancy in the prevalence of PSCI between ischemic and hemorrhagic strokes was not definitive. Furthermore, the occurrence of PSCI was found to be significantly associated with the severity of the stroke.

Comorbidity

The clinical determinants of PSCI include pre-existing and coexisting diseases that act as risk factors for cardiovascular and cerebrovascular disease [25]. The Cognitive Function After Stroke (CogFAST) study demonstrated that the presence of three or more cardiovascular risk factors was associated with a 3.6-fold increased risk of poststroke dementia in the elderly population [26]. It is well established across studies that diabetes and atrial fibrillation increase the risk of poststroke dementia, with an increased likelihood of 1.5 and 1.9 times, respectively [7]. Atrial fibrillation has been demonstrated to elevate the risk of both the stroke index and subclinical cerebral infarction or widespread SVD. Furthermore, diabetes may also serve to exacerbate the overall burden of SVD. The results of autopsy studies indicate that diabetes is associated with an increased risk of brain infarction, particularly lacunar infarction, but not Alzheimer's disease [27]. The burden of SVD contributes to cognitive impairment and dementia, which can affect multiple brain regions and multiple domains, including executive function, processing speed, and memory [28]. A meta-analysis revealed that the presence of leukoaraiosis and brain atrophy elevated the risk of poststroke dementia by 2.3 and 2.2 times, respectively [7]. The concomitant presence of neuropathology and cerebrovascular disease is likely to accelerate the development of dementia. Individuals with mixed neuropathologic findings are three times more likely to develop dementia than those with SVD alone [1]. MTLA/amyloid β deposition is a strong risk factor for poststroke dementia [7]. High blood pressure and smoking have not been consistently demonstrated to be risk factors for PSCI [7]. While further research is necessary, large blood pressure variability in the early poststroke phase is associated with cognitive impairment.

Among the initial characteristics of stroke care, several complications were identified as predictors of PSCI. These included hypoxic ischemic episodes (OR, 2.4; 95% CI, 1.4–4.2; $p = 0.002$), urinary incontinence (OR, 6.4; 95% CI, 4.5–9.2; $p < 0.0001$), acute confusion (OR, 2.8; 95% CI, 1.5–5.3; $p = 0.0001$), speech impairment (OR, 4.1), early seizures (OR, 5.4; 95% CI, 2.4–12.1; $p < 0.0001$), recurrent stroke (OR, 2.3; 95% CI, 1.5–3.5; $p < 0.0001$), and abnormal EEG findings (OR, 2.7; 95% CI, 1.4–4.9; $p = 0.002$) [7].

Factors in neuroimaging

As imaging technology advances, the prediction of PSCI through brain imaging is also being actively investigated. A systematic review and meta-analysis of PSCI and computer tomography imaging indicates that white matter lesions (OR, 2.46; 95% CI, 1.25–4.84), cerebral atrophy (OR, 2.80; 95% CI, 1.21–6.51), and pre-existing stroke lesions (OR, 2.38; 95% CI, 1.06–5.32) were associated with post-stroke dementia. Additionally, white matter lesions (OR, 3.46; 95% CI, 2.17–5.52) were associated with PSCI [29]. In the case of MRI, previous research has identified associations between cognitive outcomes and the presence of cerebral atrophy (OR, 2.48; 95% CI, 1.15–4.62), the presence of microbleeds (OR, 1.36; 95% CI, 1.08–1.70). Furthermore, increasing severity of white matter hyperintensities (OR, 1.26; 95% CI, 1.06–

1.49) [23] has been linked to an elevated risk of PSCI [30]. European Stroke Organization and European Academy of Neurology joint guidelines on PSCI summarized about CT and MRI brain imaging for predicting future diagnosis [31]. This guideline concluded that the presence of significant white matter hyperintensities presumed to be of vascular origin on acute MRI brain imaging may assist in predicting cognitive outcomes [31].

Molecular biomarkers

An increasing number of studies have sought to identify the relevant molecular biomarkers in PSCI. Among these biomarkers, inflammatory markers such as C-reactive protein (CRP), interleukin (IL) 6, and IL-10 in blood, urine, and other body fluids have been linked to cognitive decline following a stroke [32]. In a separate meta-analysis, 40 studies were reviewed, and it was found that levels of homocysteine, CRP, total cholesterol, and low-density lipoprotein cholesterol were significantly higher in patients with PSCI than in the non-PSCI group [33].

Following a stroke, brain-derived neurotrophic factor (BDNF) is the primary neurotrophic protein. Research indicates that serum BDNF levels are markedly reduced in individuals with dementia, and severe cognitive impairment is linked to diminished BDNF levels [32].

CONCLUSION

As previously stated, numerous risk factors contribute to PSCI, with the prevailing hypothesis being that these factors interact with one another to precipitate the condition. In order to synthesize the aforementioned information, several research groups have recently proposed analytical models for PSCI using machine learning [34]. PSCI has the potential to be disabling for many patients, and a number of risk factors can be identified in patient characteristics, clinical findings, and imaging findings. It is unfortunate that the majority of factors associated with PSCI are non-modifiable. The only modifiable factor that can be controlled is the management of stroke risk factors for secondary prevention. While the efficacy of diverse cognitive rehabilitation programs following stroke has been documented [35,36], there is currently no compelling evidence that cognitive rehabilitation is an effective intervention for PSCI after stroke. Future research is needed to elucidate the potential benefits of various cognitive rehabilitation programs for the prevention and improvement of PSCI. In patients with risk factors, comprehensive cognitive assessments should be conducted to monitor for PSCI. Modification of these risk factors and the implementation of appropriate cognitive rehabilitation strategies for at-risk populations may prove beneficial in the future.

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