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STRATEGIC INFARCT AND COGNITIVE IMPAIRMENT: A NARRATIVE REVIEW

Neurology

Dr Pushpendra N Renjen	Sr. Consultant Neurologist & Academic Coordinator, Institute of Neurosciences, Indraprastha Apollo Hospitals
Dr. Priyal	Neurology Resident, Indraprastha Apollo Hospital, New Delhi.
Dr Dinesh Mohan Chaudhari	Attending Consultant Neurology, Institute of Neurosciences. Indraprastha Apollo Hospitals – 110076
Dr Anjali Mishra	Attending Consultant, Critical Care Medicine, Holy Family Hospital, Okhla, New Delhi
Dr Neelam Sahu	DNB Neurology, Indraprastha Apollo Hospital, New Delhi.
Dr Arushee Bhatnagar	Neurology Resident, Indraprastha Apollo Hospital, New Delhi.

ABSTRACT

The location of single strategic infarcts and the cognitive impairment after a stroke reflect the neuroanatomy of the brain's cognitive functions. Post-stroke dementia or post-stroke cognitive impairment (PSD/PSCI) is a form of cognitive disturbance caused by cerebrovascular disease that can manifest at different times after a stroke, including during the acute phase of stroke, during the recovery phase (while motor stroke symptoms improve), or months and years later. The onset of cognitive impairment depends on the location of infarct. Infarct in certain locations have cognitive impairment as a sole presentation from the outset. Infarcts in the left frontotemporal lobes, right parietal lobe, and left thalamus are notably correlated with cognitive impairment following a stroke. In the former, patients present with complains of motor difficulties as the main complain. In addition to these locations, vascular lesions in the thalamo-perforating area of the PCA may cause dementia and other forms of severe memory loss. A baseline (pre-morbid) and early post-stroke (early post-morbid) cognitive status should be evaluated and a protocol for the same is needed in regular clinical and ICU care facilities. MoCA assessment is commonly used for cognitive assessment. Its role in strategic infarcts is undermined as it leaves out the evaluation of symptoms corresponding to common strategic infarct locations such as the medial temporal lobe and thalami. There is a need for dedicated neuropsychological assessment score based on education level, and native-language for PSCI.

KEYWORDS

REVIEW

Post-stroke dementia (PSD) or post-stroke cognitive impairment (PSCI) is cognitive impairment due to CVD occurring in different timeframes, i.e., in the acute stage, in the recovery stage (while other stroke symptoms improve), or it maybe show delayed presentation until months or years after the stroke [1]. The onset of cognitive impairment is influenced by the location of the infarct. Infarcts occurring in specific areas of the brain can result in cognitive impairment as the primary manifestation right from the beginning. The first signs of PSI/PSD are changes in routine activities and repeating the same mistakes. The location of single strategic infarcts and PSCI echo with the neuroanatomy of cognitive functions. It also foresees the associated emotional strain on patients' lives [1]. Apart from location, disseminated superficial siderosis, a high cortical atrophy score, large number of cerebral microbleeds, cerebral amyloid angiopathy and older age at stroke are other risk factors for new-onset PSCI/PSD [2]. Leukoaraiosis became another independent predictor of PSCI [3]. Small vessel disease (SVD) of the thalamo-perforating branch of the PCA may give rise to a bilateral butterfly-shaped lesion due to affection of the intralaminar nuclei of the thalamus, and patients present with dementia and severe memory loss (thalamic dementia) [3]. The bilateral paramedian thalamic infarction syndrome consists of a transient coma followed by an apathetic hypersomnolent state. In addition, there is Korsakoff's type of amnesia (anterograde) and vertical gaze palsy. Strategic infarct in the same territories can give rise to similar presentation [3]. Similarly, the frontotemporal infarcts (another strategic infarct implicated in causing PSD/PSCI) cause personality and behavioral changes, including disinhibition, apathy, as well as executive dysfunction. Pre-stroke cognitive assessment should be considered while evaluating patients for PSI/PSD. Pre-stroke cognition is affected by presence of epileptic activity, sepsis, and pre-morbid/pre-stroke leukoaraiosis on brain imaging. Factors, such as education level (reflected in job type and early-life intelligence), age at stroke onset, severity of stroke, lesion multiplicity, hypertension, diabetes, and cardiac disease influence PSCI [1,5]. Previous cerebrovascular events, covert cerebrovascular disease (such as SVD), coexisting neurodegenerative conditions (evaluated using ADL, IADL, and IQ code), mood and psychiatric disturbances, fatigue, or a combination of these factors contribute to cognitive impairment before the stroke [6]. PSCI/PSD are also strongly related to pre-stroke cognition predictors such as leisure activities, and relationship status [1]. Early PSCI is strongly linked to infarct features (mostly size and location), while late PSCI is linked to ongoing small vessel disease (SVD) present on MRI [1]. At most, cognitive assessment protocols are unsuitable for people with dysphasia. There is a need for uniform cognitive scoring tests in post-stroke follow-up. It is important to assess the cognitive status of individuals before the stroke (baseline/pre-morbid) and in the early stages following the stroke (early post-morbid) and establish a protocol for such evaluations in regular clinical and ICU care settings.

Research done on “infarct-symptom mapping” enlightens us about biases in assessment of cognition in post- stroke follow-ups. The MoCA assessment is commonly used for cognitive evaluation, but its usefulness is limited in strategic infarcts as it does not adequately address symptoms related to common strategic infarct locations such as the medial temporal lobe and thalami. When the authors performed a multidomain neuropsychological evaluation, the prevalence of cognitive impairment was higher compared to cohorts in which the diagnosis was based on a single screening test (such as MoCA). MOCA detects cognitive deficits less effectively than multidomain neuropsychological testing as it predominantly tests dorsolateral prefrontal cortex, hippocampus, bilateral parietal lobes, anterior temporal lobes and orbitofrontal cortex. PSCI could be diagnosed with a cutoff of 21, using MoCA, with a sensitivity of 91.4%, a specificity of 75.8%, a positive predictive value of 80%, and a negative predictive value of 89.3%. The more time passes between a stroke and a cognitive assessment, the more likely the patient is to have cognitive impairment. In another study on the diagnostic accuracy of MOCA in diagnosing PSD/PSCI, it was demonstrated that MoCA is a quick bedside test in the acute phase of a stroke to diagnose mid-term PSCI. Due to the significant differences in demographic background, and characteristics, test normative data and cutoffs should be based on the same country population and should represent older and low-education subjects while determining cognitive test cutoff scores.

The neuroimaging predictors of PSCI differ depending on the time point of onset of cognitive impairment symptom, and the most

common findings predicting PSCI are global and medial temporal lobe atrophy. Prevalence of PSCI is less common in study groups (and articles) that leave out severe stroke cases. Researchers created a comprehensive brain map with high brain-lesion coverage by sampling data from twelve acute ischemic stroke cohorts, identifying strategic infarct locations associated with PSCI. They analyzed three-stroke subgroups: small subcortical infarcts, cortical or large subcortical infarcts, and infratentorial infarcts. Because of how rarely they were involved, their analyses did not include the most distant parts of the territories of the anterior cerebral artery, parts of the midbrain, and the temporal poles. A prediction score for patient's cognitive prognosis and his PSCI risk was formulated based on patient's infarct location. The calculation of the location impact score revealed that infarcts in the left frontotemporal lobe, the left thalamus, and the right parietal lobe were strongly linked to cognitive impairment. Conversely, their was a lower risk of PSCI from infarcts in the right occipital lobe, brainstem, and cerebellum [7-9]. With these limitations, MoCA assessment leaves out the evaluation of symptoms corresponding to common strategic infarct locations implicated in cognitive dysfunction such as the medial temporal lobe and thalami. Conversely, the lesion-symptom mapping ignores temporal pole infarcts have a role in causing PSD/PSCI, a region evaluated in MoCA. As a result, we require a dedicated neuropsychological assessment score based on education level, and native- language that targets these strategically infarcted locations. A minimum of three domains, corresponding to the three most common strategic infarct locations, should be assessed to exclude PSCI/PSD, and impaired performance in a single cognitive domain should be sufficient to establish PSCI. These single strategic infarcts appear to impair communication and information processing between different brain regions. When evaluating and treating stroke patients, healthcare providers must be aware of this association and consider the potential cognitive consequences of strategic infarcts.

CONCLUSION

Post-stroke dementia (PSD) or post-stroke cognitive impairment (PSCI) is cognitive impairment due to CVD occurring in different timeframes post-stroke. One of the major risks of early PSCI is the location of infarct. The neuroimaging predictors of PSCI differ depending on the time point of onset of cognitive impairment symptom, and the most common findings predicting PSCI are global and medial temporal lobe atrophy. A comprehensive brain map with high brain-lesion coverage identified strategic infarct locations associated with PSCI. MoCA assessment is great for cognitive screening in acute stroke and includes assessment of temporal poles but leaves out the evaluation of symptoms corresponding to common strategic infarct locations responsible for early PSCI such as the medial temporal lobe and thalami. Healthcare providers must be aware of this association and consider the potential cognitive consequences of strategic infarcts when treating stroke patients.

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