

EEG MARKERS OF COGNITIVE IMPAIRMENT IN CEREBRAL SMALL VESSEL DISEASE AND NEUROFEEDBACK: A NARRATIVE REVIEW

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ABSTRACT: Cerebral small vessel disease (CSVD) is a major cause of vascular cognitive impairment (VCI) and vascular dementia (VaD), yet early detection and monitoring remain challenging. Electroencephalography (EEG) may offer a non-invasive and cost-effective approach to identify functional brain alterations associated with CSVD. This narrative review summarizes current evidence on EEG markers in CSVD patients with mild or major cognitive impairment. Across studies, CSVD was consistently associated with spectral slowing (increased delta and theta power, reduced alpha activity), disrupted functional connectivity (particularly in fronto-parietal networks) and altered event-related potentials. Several EEG measures correlated with cognitive performance and lesion burden, suggesting potential value for disease monitoring. However, heterogeneity in diagnostic criteria and methodological approaches limits comparability across studies. Overall, EEG biomarkers appear promising for early detection and monitoring of CSVD and may provide targets for future neurofeedback interventions aimed at mitigating cognitive decline, which we also discuss.

INTRODUCTION

The term cerebral small vessel disease (CSVD) encompasses a heterogeneous group of pathological processes affecting the small vessels of the brain, including small arteries, capillaries, arterioles, and venules [1]. These pathologies can lead to both ischemic and hemorrhagic consequences, resulting in various parenchymal lesions. The progression of CSVD is highly variable, with many patients remaining asymptomatic for years (covert CSVD). However, the cumulative burden of lesions can eventually lead to cognitive, neuropsychiatric, and mobility disorders [2]. The prevalence of the condition increases with aging and is very common in older individuals [3], making CSVD a growing public health challenge.

Vascular cognitive impairment (VCI) refers to all forms of cognitive impairment due to cerebrovascular disease. According to the Vascular Impairment of Cognition Classification Consensus Study (VICCCS) [4], the classification of VCI severity ranges from mild VCI to vascular dementia (VaD). CSVD is the primary cause of VCI and subcortical ischemic vascular dementia

(SiVaD) [5], one of the four subtypes of VaD with post-stroke dementia, multi-infarct dementia, and mixed dementias. SiVaD is characterized by executive dysfunction, impairing information processing and problem resolution, caused by the disruption of prefrontal-subcortical connections [6].

The lesions related to CSVD are typically detected via magnetic resonance imaging (MRI) or computed tomography (CT) [7]. However, CSVD lesions can induce remote effects which, associated with patient-specific factors (e.g., age, genetic background, cognitive reserve), contribute to the heterogeneous clinical presentation [8]. Electroencephalography (EEG) is a promising alternative as it is non-invasive, relatively inexpensive, portable, and easily repeatable. Therefore, EEG could help identify neuromarkers of early CSVD-related cognitive impairment to improve the early identification and monitoring of the disease. Moreover, EEG-based brain-computer interfaces (BCIs) could support symptom management through neurofeedback techniques [9] that modulate these neuromarkers and potentially reduce associated cognitive deficit.

Few studies have investigated EEG neuromarkers of CSVD-related cognitive impairment, and heterogeneity in VCI definitions complicates interpretation, as many studies do not distinguish VCI subtypes. Babiloni et al. [10] reported reduced resting-state posterior alpha power, widespread increase in delta and theta activity, and prolonged N200 and P300 latencies in VCI patients. Dey et al. [11] found similar results in their systematic review focusing on CSVD patients. They additionally reported reduced coherence of higher-frequency rhythms across fronto-parietal networks, interpreted as markers of disrupted long-range connectivity linking white matter damage to attentional and executive dysfunction. In their systematic review of EEG and MEG biomarkers of VCI, Torres-Simón et al. [12] attempted to categorize the studies they included according to the VCI subtypes defined by the VICCCS classification and found EEG alterations consistent with previous reviews. While these reviews have substantially advanced understanding of EEG alterations in VCI, important gaps remain. First, only Dey et al. focuses exclusively on CSVD as the underlying pathology: Babiloni et al. evaluated major VCI as a broad category, and Torres-Simón et al. could not draw SiVaD-specific conclusions due to diagnostic

heterogeneity. Second, none of the existing reviews has examined the implications of these EEG findings for neurofeedback-based therapeutic interventions, despite growing interest in non-pharmacological brain stimulation approaches for cognitive impairment.

The aim of this narrative review is to summarize and update the findings from the existing reviews with a focus on CSVD patients with mild or major cognitive impairment. We also discuss the methodological heterogeneity across studies and the challenges associated with translating these findings into tools for the diagnosis or monitoring of covert CSVD. Finally, we outlined the open questions that remain to be addressed for the development of effective neurofeedback interventions.

MATERIALS AND METHODS

A search of the literature was conducted using PubMed, Scopus and PsycInfo databases with the following query in title and abstract: ("small vessel disease" OR "small-vessel disease" OR "subcortical vascular dementia" OR "subcortical ischemic vascular dementia" OR "subcortical vascular cognitive impairment" OR "subcortical vascular lesion" OR "subcortical cerebrovascular lesion" OR "subcortical vascular damage" OR "subcortical cerebrovascular damage") AND (EEG OR EEGs OR electroencephalogra* OR electro-encephalogra* OR "neural oscillation" OR "brain oscillation" OR ERP OR ERPs OR "event related potential" OR "event-related potential" OR P300 OR N200 OR N400 OR "spectral power" OR "power spectrum" OR "frequency band" OR "functional connectivity") AND (cognit* OR "cognitive dysfunction" OR "cognitive changes" OR "cognitive impairment" OR "cognitive disorder" OR "cognitive dysfunction" OR "cognitive decline" OR "mild cognitive impairment" OR attention OR "executive function" OR "memory").

The database search identified 160 references. Many articles identified using the term "functional connectivity" were excluded because they focused exclusively on MRI analyses rather than EEG data. After screening titles and abstracts according to the inclusion criteria, 18 articles were retained for further evaluation. Because several relevant EEG studies did not explicitly use terms related to CSVD in their titles, abstracts, or keywords, additional studies were identified through manual screening of the reference lists of selected articles, particularly from review papers by Dey et al. [11] and Torres-Simón et al. [12]. In total, 27 articles were included in the present review.

Studies were included in this review if they reported analyses of EEG data in patients with cognitive impairment associated with CSVD, or subcortical vascular lesions such as WMH and lacunes confirmed by brain imaging. Only peer-reviewed papers published in English were considered, with no restriction on the date of publication. Book chapters and case studies were excluded as well as studies including patients with

comorbid neurological or psychiatric disorders (e.g., Parkinson's disease or depression).

RESULTS

The main findings indicate that patients with VCI associated with subcortical lesions show a disruption in spectral activity, functional connectivity and event-related potentials (ERP) compared to healthy controls (HC). Some studies found a correlation between these results and neuropsychological scores or lesion burden. Multiple studies also compared EEG markers between VCI patients and Alzheimer's disease (AD) patients. The results of this review are detailed below and summarized in Table 1.

Spectral analysis: Multiple studies show that CSVD patients present a slowing of brain activity compared to HC with some differences compared to AD patients. Main results included a mean frequency in the CSVD group lower than in the HC group but higher than in the AD group [13], [14], slowing of theta/alpha transition frequency [15], [16], reduced alpha peak frequency [16], [17], increased power in delta and theta bands, decreased alpha power and decreased alpha/slow wave power ratio [13], [14], [15], [16], [18], [19], [20], [21], [22], [23]. Specifically, low-resolution brain electromagnetic tomography (LORETA) studies show that SiVaD patients exhibit largely abnormal distributed theta sources compared to AD patients [18] and present a significant decrease in parieto-occipital alpha 1 (8 - 10.5 Hz) and beta 1 (13 - 22 Hz) relative power current density and a decrease in frontal delta relative power current density compared with patients with mild non-vascular cognitive impairment [20].

Connectivity analysis: CSVD damages long-range white matter tracts, especially frontosubcortical circuits. Using EEG synchronization likelihood, Babiloni et al. reported reduced fronto-parietal and interhemispheric coupling in SiVaD and AD patients compared to HC, with a stronger fronto-parietal alpha 1 reduction in VaD patients [24]. Moretti et al. also found reduced fronto-parietal and fronto-temporal coherence in patients with mild cognitive impairment due to subcortical cerebrovascular disease, with increased low-frequency and decreased high-frequency coherence linked to subcortical damage [25], [26]. Studies using phase lag index (PLI) also showed reduced alpha-band connectivity in CSVD patients with VCI compared to CSVD patients without VCI, and altered directional connectivity patterns compared to HC [27], [28]. Additionally, studies showed disrupted long-range networks in the alpha band for patients with WMH, particularly in long-range connections to the frontal lobe [22] and abnormal changes in EEG microstates compared to HC [29].

Event-related potential analysis: Studies using auditory tasks using an oddball paradigm in SiVaD patients report a smaller amplitude in N100 [30], a smaller amplitude and longer latency in P300 [30], [31], a longer latency in N200 [32] and a smaller amplitude

and shorter latency in frontocentral mismatch negativity [33]. P300 prolonged latency during auditory tasks also seems to appear at an earlier stage in CSVD compared to AD [31]. Regarding studies using a visual oddball paradigm, SiVaD patients exhibit a smaller N75 to P100 amplitude [34] and a smaller amplitude and longer latency in P300 [35]. In a visual memory workload task, patients also had lower P450 amplitude than AD patients and longer latencies over anterior regions and shorter latencies over posterior regions compared to HC [36].

Correlation with neuropsychological scores or lesion burden: Some studies report correlations between these EEG abnormalities and the severity of dementia measured by neuropsychological scores such as the Mini-Mental State Examination (MMSE) [37], or with lesion burden scales such as the Fazekas score [38]. Zhao et al. found that abnormal alpha band oscillations were correlated with Fazekas score and neuropsychological tests [17]. Gawel et al. showed that

the alpha/slow-wave power ratio differentiates mild from moderate dementia in CSVD patients [13], [14] while Moretti et al. proposed the theta/alpha1 ratio as an indicator of subcortical damage [15]. Guo et al. also reported that Montreal Cognitive Assessment (MoCA) scores [39] were negatively correlated with delta power and positively with alpha power and with Fazekas score. Moreover, reduced global average power spectral density value and low education were risk factors for cognitive impairment independently of Fazekas score [21]. Sheorajpanday et al. also identified (delta+theta)/(alpha+beta) ratio as an independent predictor of VCI and found that pairwise derived brain symmetry index correlated with neuropsychological tests [23]. Connectivity measures also relate to disease progression, such as alpha-band PLI positively correlated with MMSE and coherence changes linked to WMH severity and poorer cognitive performance [26], [27].

Table 1: Summary of studies assessing EEG markers of VCI in patients with vascular subcortical lesions.

EEG feature	Main findings for VCI patients	Related references
Spectral	VCI vs HC: Decreased alpha and beta power Widespread increase in delta and theta power Slowing of theta/alpha transition frequency Lower mean frequency Reduced alpha peak frequency	[13]*, [14]*, [15] [°] , [16], [17]* [°] , [19]*, [20], [21]* [°] , [22] [°] , [23]*
	VCI vs AD: Abnormally distributed theta sources Higher alpha/slow wave power ratios and mean frequency Higher delta and theta power	[14]*, [16], [18]
Connectivity	VCI vs HC: Reduction of fronto-parietal and interhemispheric synchronization likelihood Increase of coherence in low frequency and decrease of coherence in high frequency in right fronto-temporal and temporo-parietal neural networks. Abnormal changes in microstates Lower phase-lag index in alpha band and abnormal directional pattern	[22] [°] , [24], [25], [26] [°] , [27]*, [28], [29]*
	VCI vs AD: Lower interhemispheric alpha 1 synchronization likelihood More homogeneous decrease of synchronization likelihood over the whole scalp	[24]
Event-related potentials	VCI vs HC: Longer latency and/or smaller amplitude	[30]*, [31], [32]*, [33], [35] [°] , [36]
	VCI vs AD: Lower P450 amplitude, lower N75-P100 amplitude Prolonged P300 latency earlier in the evolution of the disease	[31], [34], [36],

VCI : vascular cognitive impairment, with or without dementia depending on studies ; AD : Alzheimer's disease ; HC : healthy controls. References in bold indicate that the authors found a correlation between EEG markers and neuropsychological score (indicated by the "*" symbol) and/or the severity of cerebrovascular lesions (indicated by the "°" symbol).

DISCUSSION

Heterogeneity in diagnostic criteria and terminology: This review included studies on CSVD patients as well as those with subcortical vascular cognitive impairment or dementia, since CSVD is the main pathology in these conditions. However, inconsistent terminology across studies represents a limitation to this review: some describe CSVD lesions without naming them, while others report VCI without specifying subcortical origin. Consequently, relevant studies may have been missed despite the search strategy. Most articles diagnosed SiVaD using NINDS-

AIREN criteria [40], though some Chinese studies used national guidelines without official English translations. Some studies also assessed the Hachinski Ischemic Score [41] as additional criteria, particularly when differentiating with AD patients. Diagnoses typically required cognitive impairment or dementia supplemented by MRI or CT examination to ensure the subcortical vascular involvement and exclude patients with cortical or non-vascular lesions. However, methodological details varied: some studies mentioned STRIVE guideline [7] or rated WMH severity with Fazekas or ARWMC scales [42], but most only indicated the presence of lacunes or leukoaraiosis

without quantifying the severity of lesions. Variability in lesion burden, non-linear VCI progression, and limitations of MMSE for diagnosis of mild cognitive impairment [43] complicate comparisons across studies. These limitations are consistent with those reported by Torres-Simón et al. [12], who similarly noted that diagnostic heterogeneity prevented SiVaD-specific conclusions.

Use of EEG for diagnosis of asymptomatic CSVD: A recent article proposed an algorithm to diagnose CSVD patients with EEG and report a 96% accuracy in differentiating patients with or without VCI from HC [44]. However, the article does not specify whether the cross-validation procedure ensured strict subject separation between training and test sets, creating a substantial risk of intra-subject information leakage which can artificially inflate classification performance [45]. Other studies highlight potential EEG markers in asymptomatic CSVD: lower amplitude and longer latency of P300 [35], [31], increased theta power and disrupted alpha network [22], altered theta/alpha transition frequency [15], and abnormal EEG activity in regions involved in the default mode network [20] could be markers for cerebrovascular damage at an early stage of the disease, prior to clinical presentation. However, more longitudinal studies are necessary to assess if EEG markers could help predict the development of VCI in CSVD patients. Another limitation is the fact that the studies included in this review collected EEG data only during resting state conditions. The analysis of brain oscillation during cognitive tasks could be promising to better detect CSVD patients from healthy individuals and to monitor the progression of the disease.

Development of EEG-based neurofeedback therapies: To the best of our knowledge, only one study investigated neurofeedback for cognitive impairment in CSVD [46]. The authors report that an infralow-wave neurofeedback training increased alpha rhythms and improved patients' executive function up to 6–8 weeks post-training. However, this article did not provide detailed information about the neurofeedback protocol used during the experiment, complicating the replication of its results and the assessment of their validity [47]. More generally, there is a limited number of studies on EEG neurofeedback in patients with mild cognitive impairment or dementia [48]. Potential interventions for CSVD could target the alpha frequency band by increasing alpha power spectral density or alpha peak frequency as previous studies have reported that alpha-band training can improve cognitive performance [49]. Theta neurofeedback protocols also seem promising in improving executive function in older adults [50] and may warrant further investigation in patients with CSVD. A second neurofeedback approach could focus on coherence between brain regions in order to improve functional connectivity, but this type of protocol remains relatively underexplored. Similarly, ERP-based neurofeedback has primarily been applied to attentional training in individual with attention deficit hyperactivity disorder (ADHD) [51] but its use for broader cognitive

training is limited [52]. Therefore, given the limited evidence, more studies are necessary to assess whether neurofeedback can improve cognitive performance, particularly in CSVD patients. In addition to selecting appropriate EEG markers, several practical factors should be considered when designing neurofeedback protocols [53]. As these interventions primarily target older adults, the number and duration of sessions should be optimized to maintain engagement and minimize dropout, a common challenge in this population. Moreover, the type of feedback provided during training should be adapted to older adults, as intuitive and easily interpretable feedback may enhance both engagement and performance.

CONCLUSION

Subcortical lesions associated with CSVD often eventually lead to VCI or dementia and cause several EEG abnormalities. The most consistent finding is spectral slowing, characterized by increased delta and theta power and reduced alpha power. Other important results include disrupted functional connectivity, especially in connections involving frontal regions, and altered event-related potentials with reduced amplitude and prolonged latencies. Importantly, these changes can appear early, before cognitive impairment is evident. Some studies also report significant correlations between EEG markers and cognitive scores or lesion severity, suggesting that they may reflect disease progression. However, differences in diagnostic criteria and in the assessment of lesion burden and cognitive impairment limit the generalization of these findings. Moreover, most of the studies used resting-state EEG, leaving a gap in the literature regarding task-related activity. These findings also suggest potential for neurofeedback interventions. Future work will aim to identify EEG markers in patients with CSVD during cognitive tasks to guide the development of neurofeedback protocols and test whether modulating these signals improves cognition.

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