

CEREBROVASCULAR INJURY, POST-COVID NEUROLOGICAL COMPLICATIONS AND SEIZURE SUSCEPTIBILITY: AN INTEGRATIVE NARRATIVE REVIEW

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ABSTRACT

Background: Acute and chronic vascular brain injury may produce transient seizures, delayed epilepsy, and other paroxysmal neurological events. Post-infectious and neuroinflammatory factors complicate etiological assessment.

Objective: To synthesize published work on cerebrovascular and post-COVID neurological disorders and identify a clinically useful framework for recognizing seizure susceptibility without conflating associated syndromes.

Methods: A narrative thematic analysis of 33 user-supplied bibliographic records (2018–2026), supplemented by established neurological classification concepts, was undertaken. Original clinical data and meta-analysis were not available.

Results: The literature maps to three intersecting areas: stroke and rehabilitation, Parkinsonian syndromes and differential diagnosis, and neuroinflammatory biomarker studies. Structural lesions plausibly increase seizure risk; the selected studies do not permit pooled incidence estimates.

Conclusion: Temporal relationship to the vascular event, seizure semiology, etiological work-up, and targeted EEG/imaging are central to evaluation. Seizure-specific prospective data are still needed.

Keywords: cerebrovascular disease; post-stroke seizures; COVID-19; paroxysmal events; neuroinflammation; EEG; rehabilitation.

1. INTRODUCTION

Ischemic and hemorrhagic stroke can cause neurological symptoms ranging from persistent motor deficit to transient paroxysmal events. Epileptic seizures may arise during acute cortical injury or subsequently as a manifestation of altered neuronal networks. Yet not every involuntary movement, episode of collapse, or fluctuation of consciousness after stroke represents an epileptic seizure. The distinction influences investigation, treatment decisions, prognosis, and rehabilitation planning.

The bibliography supplied for this review includes regional clinical writing on post-stroke rehabilitation, COVID-19-associated stroke and neurological sequelae, neurodegenerative movement disorders, and molecular investigations of inflammation (Rakhimbaeva et al., 2023; Tuychiev et al., 2021; Akramova et al., 2025). These connected topics provide a useful setting to explore diagnostic reasoning, even though most articles were not designed to study seizure incidence directly.

Acute metabolic stress, glutamate-mediated excitotoxicity, damage to inhibitory circuits, blood–brain barrier dysfunction and glial activation may change neuronal excitability after vascular injury. Months or years later, network remodeling and gliosis may sustain a tendency toward unprovoked seizures. Thus the time of the event relative to injury is often as important as the motor appearance of the event.

The objective is to integrate clinical and mechanistic perspectives on seizure susceptibility in cerebrovascular and post-COVID neurological disorders, define boundaries of inference from the selected literature, and provide a structured diagnostic framework for future research.

2. MATERIALS AND METHODS

This narrative review used all 33 bibliographic entries furnished for the project as a prespecified source set. Eleven entries concerned Gazieva and collaborators, eleven Daminova and collaborators, and eleven Akramova and collaborators. Publication dates span 2018–2026. Some entries are conference supplements, and several require completion or source verification before journal submission.

Titles and available bibliographic descriptions were coded into four themes: (1) stroke and vascular injury, (2) post-COVID neurological outcomes and rehabilitation, (3) Parkinsonian and other movement disorders relevant to event differentiation, and (4) biomarkers of inflammation or neurodegeneration. Interpretive synthesis was qualitative, without effect-size pooling or claims of systematic literature capture.

The analytic unit was the published topic rather than an individual patient. Clinical concepts such as acute symptomatic seizures, delayed unprovoked seizures, focal-to-bilateral tonic-clonic progression and nonepileptic motor events were used to

structure interpretation. No new patient cohort, laboratory assay, neurophysiological dataset, ethics approval, statistical testing, or clinical risk estimate is represented in this manuscript.

3. RESULTS

3.1. Cerebrovascular injury and the timing of seizures

When cortical tissue is deprived of adequate perfusion, ATP depletion disrupts ion gradients and synaptic control. Excess excitatory neurotransmission, neuronal depolarization and local inflammation create conditions permissive to acute symptomatic seizures. In contrast, late unprovoked seizures may reflect an enduring network lesion with gliotic scarring, modified excitation–inhibition balance and aberrant connectivity.

Clinical evaluation should document the interval from stroke to the suspected episode, lesion distribution, neurological deficits and any intercurrent systemic trigger. A focal clonic movement, unilateral sensory aura, sudden behavioral arrest or focal-to-bilateral convulsive event can suggest seizure onset, while syncope, limb-shaking transient ischemic attacks and spasticity-associated movements require careful differentiation.

3.2. Post-COVID and postinfectious vascular contexts

The works of Rakhimbaeva, Gazieva, Ataniyazov and colleagues discuss the treatment, neurological consequences and rehabilitation of ischemic stroke in a COVID-19 context. Bobamuratova and collaborators additionally describe cavernous sinus thrombosis complications. Together these publications establish a regional research emphasis on vascular and systemic complications, rather than a uniform seizure-specific cohort.

Postinfectious events require more than a temporal association with SARS-CoV-2 for causal attribution. Hypoxia, fever, renal or hepatic failure, medication effects, systemic thromboinflammation, cerebral ischemia and pre-existing epilepsy may each contribute. Clinical documentation should therefore separate the underlying acute illness, structural insult, precipitating abnormalities and electroclinical seizure findings.

3.3. Overlap with movement and cognitive disorders

The Parkinson disease studies by Daminova, Akramova and their teams are not primary post-stroke seizure investigations; nonetheless, they stress an essential diagnostic point. Tremor, medication-related dyskinesia, dystonia, myoclonus and fluctuations in attention may resemble seizures in patients with complex neurological disease.

Vascular parkinsonism and cognitive symptoms can coexist with cerebrovascular pathology, complicating bedside interpretation. Episodes that are prolonged, posture-dependent, medication-related or linked to orthostatic change may suggest non-epileptic explanations. Conversely, highly stereotyped brief events with evolving motor semiology or postictal dysfunction warrant further epileptological assessment.

3.4. Role of neuroinflammatory and injury markers

The biomarker-oriented publications of Akramova and collaborators explore MCP-1, alpha-synuclein and indicators of neuroaxonal injury in Parkinson disease and Parkinson-plus syndromes. These molecular programs are relevant to a broader hypothesis that chronic inflammation and degenerative injury influence neural networks.

However, measurements reported in parkinsonian cohorts cannot be transferred to patients with post-stroke epilepsy as validated diagnostic or prognostic thresholds. Such translational hypotheses require control for age, systemic inflammation, lesion volume, vascular comorbidity and concurrent medications.

Table 1. Comparative clinical and methodological framework

Clinical setting	Key diagnostic consideration	Evidence boundary
Acute stroke	Structural lesion, metabolic triggers, focal semiology	Do not infer epilepsy from one acute event
Late post-stroke events	Recurrent unprovoked episodes, EEG and MRI	Estimate risk only with longitudinal data

Post-COVID neurological syndrome	Distinguish infection, vascular injury and systemic stress	Temporal association is not causation
Movement or cognitive disorder	Differentiate tremor, dyskinesia, syncope and seizure	Semiology alone is not always definitive

4. DISCUSSION

The reviewed sources favor an integrated rather than reductionist approach to post-stroke paroxysmal events. First, seizure diagnosis is a clinical-electrophysiological assessment, not a synonym for abnormal movement. Second, lesion-associated epileptogenesis represents only one explanation for transient symptoms after cerebrovascular disease. Third, vascular and postinfectious comorbidities may alter both clinical appearance and interpretation of laboratory findings.

An initial evaluation should establish episode chronology and obtain witness accounts or videos. A focused examination can identify cortical deficits, postictal paresis, tremor, rigidity, orthostatic abnormalities and features of systemic illness. Blood glucose, electrolytes and other laboratory tests should be selected according to presentation. Urgent neuroimaging is appropriate where new structural pathology is suspected. EEG supports diagnostic characterization but a normal brief routine recording does not exclude epilepsy.

Immediate treatment decisions differ between an acute symptomatic seizure associated with a reversible trigger and recurring unprovoked seizures associated with persistent brain pathology. Long-term antiseizure management should not be inferred solely from a history of stroke or a single ambiguous spell. Likewise, rehabilitation is indicated for functional recovery but the cited rehabilitation papers do not establish that a particular exercise regimen prevents epileptogenesis.

Methodological limitations are substantial. The set was selected for author coverage rather than exhaustive retrieval; source designs vary, including abstracts and narrative papers; seizure outcomes are absent in much of the bibliography; and two identically titled rehabilitation publications may represent overlapping material.

Therefore percentages, risk ratios, correlations, and therapy effectiveness estimates cannot responsibly be computed from these records.

A useful prospective study would consecutively enroll patients with imaging-confirmed stroke, classify early versus late events, capture standardized semiology and EEG, record lesion location and severity, and adjudicate outcomes independently. Post-COVID exposure and inflammatory biomarkers should be treated as prespecified covariates rather than explanations assumed in advance. Such research would enable clearer differentiation between association, prediction and causation.

5. CONCLUSION

Cerebrovascular injury can alter brain-network excitability and may lead to acute symptomatic seizures or later epilepsy, but a broad range of nonepileptic paroxysmal presentations also occurs in neurologically complex patients. The available regional literature provides contextual evidence concerning ischemic stroke, post-COVID neurological injury, rehabilitation and neurodegeneration, without supporting numerical seizure-risk estimates.

Clinical precision requires attention to event timing, semiology, EEG, imaging and reversible triggers. The combined literature supports a research agenda linking vascular lesion characterization to prospectively measured electroclinical outcomes and clearly defined biomarker hypotheses.

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