

NEUROINFLAMMATION AND NEURODEGENERATION IN NEUROLOGICAL DISORDERS: MOLECULAR BIOMARKERS, NEURONAL EXCITABILITY, AND IMPLICATIONS FOR SEIZURE ASSESSMENT

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ABSTRACT

Background: Inflammation, neuronal injury and neurodegeneration occur across distinct neurological diseases and can converge on pathways regulating synaptic transmission and neuronal excitability. Yet an association between inflammatory biomarkers and Parkinsonian pathology is not equivalent to validation of a seizure biomarker.

Objective: To synthesize regional research concerning MCP-1/CCL2, alpha-synuclein, vascular injury, Parkinson disease and neurological rehabilitation, and critically assess its relevance to seizure mechanisms and future electroclinical biomarker studies.

Methods: A narrative, evidence-mapping review of 33 supplied bibliographic records (2018–2026), supplemented conceptually by standard epileptological principles. Records were sorted by pathology, biomarker domain, clinical outcome and directness of seizure evidence. No new patient data were collected.

Results: The literature clusters around Parkinson disease and atypical parkinsonism, vascular and post-COVID injury, and clinical rehabilitation. MCP-1 and alpha-synuclein were investigated in neurodegenerative contexts. Direct evidence that these measures classify, predict or monitor epileptic seizures was not established by the supplied publications.

Conclusion: Biomarker studies should be interpreted within their original diagnostic context. Future work must pair prespecified molecular endpoints with carefully adjudicated seizures, EEG, imaging and longitudinal follow-up before clinical claims are justified.

Keywords: neuroinflammation; neurodegeneration; CCL2; MCP-1; alpha-synuclein; neuroaxonal injury; seizure; epileptogenesis; Parkinson disease; stroke; biomarkers.

1. INTRODUCTION

Neurological disorders reflect interactions between neuronal structure, synaptic physiology, vascular supply, immune responses and systemic health. The same clinical complaint may arise through very different mechanisms. For example, episodic limb movement may represent an epileptic motor seizure, Parkinsonian tremor, treatment-associated dyskinesia, dystonia, syncope-associated jerking or a functional neurological event. The clinical challenge is therefore not simply to identify movement but to determine the underlying physiological process.

Epileptic seizures arise from abnormal patterns of neuronal activity in distributed cerebral networks. Neuroinflammatory mechanisms may facilitate such activity by affecting neurotransmission, extracellular ions, glial regulation and synaptic plasticity. Nevertheless, inflammation is a broad biological response rather than a diagnosis of epilepsy. Inflammatory activity in a patient with stroke or Parkinson disease must not be interpreted as proof that an observed paroxysm was epileptic.

The supplied bibliography brings together several regional lines of investigation. Gazieva and colleagues examine post-stroke rehabilitation, coronavirus-associated cerebrovascular complications and neurological consequences of infection. Daminova and collaborators examine motor and nonmotor Parkinsonian phenotypes, prodromal markers, olfactory dysfunction, familial susceptibility and longitudinal clinical change. Akramova and colleagues address vascular parkinsonism, MCP-1, alpha-synuclein, neuroaxonal markers, cognition and rehabilitation. These lines of work offer complementary perspectives on neurological heterogeneity.

Alpha-synuclein is a synaptic protein whose abnormal accumulation is central to the pathology of synucleinopathies. Its molecular forms and tissue distribution may differ substantially from the concentration measured by a specific blood assay. MCP-1, also designated CCL2, is a chemokine involved in cellular recruitment and immune signaling. Both are research targets in neurological disease, but neither

should be described as a clinically validated seizure biomarker solely on the basis of Parkinson-related reports.

The acute and chronic consequences of brain vascular injury provide a second important context. Ischemia can impair energy-dependent ion homeostasis and alter glutamate metabolism. Persistent damage may reorganize cortical networks, thereby affecting susceptibility to late seizures. Yet a study of post-stroke rehabilitation may report motor recovery without having collected seizure outcomes. The present review maintains that distinction between mechanistic plausibility and demonstrated clinical evidence.

Biomarker development requires more than biological plausibility. A marker must be analytically reproducible, associated with a defined clinical endpoint, sufficiently informative beyond standard clinical assessment and validated across relevant populations. Its use in early screening, prognosis, differential diagnosis and treatment response can each require different thresholds and different study designs. These issues are particularly important when candidate markers are influenced by general inflammation or age.

The principal question guiding this review is therefore whether the 33 supplied publications, collectively, support the clinical use of neuroinflammatory or neurodegenerative measures for seizure assessment. A secondary aim is to identify relevant hypotheses and define a research agenda that would translate vascular and Parkinsonian biomarker studies into rigorously assessed epileptological work without overstating current findings.

2. MATERIALS AND METHODS

2.1. Review design and source set. This article is a critical narrative review with a structured evidence map, not a systematic review or meta-analysis. The starting set was the 33 bibliographic entries provided by the authors, published between 2018 and 2026. All supplied entries were retained in the reference list for traceability, including reports whose research question is not directly about seizures. A review relying on a supplied bibliography cannot claim comprehensive coverage of the global literature.

2.2. Eligibility and classification. Records were classified from their titles, publication metadata and available descriptive information into three domains: (i) cerebrovascular and post-COVID injury, (ii) Parkinson disease, atypical parkinsonism and movement disorders, and (iii) inflammatory, synuclein-related or neuroaxonal biomarker research. Some records cross domains. Classification was used for interpretation rather than pooled counting, and titles were never treated as proof of an unreported numerical result.

2.3. Evidence extraction. We examined each record for its disease population, putative biomarker or clinical domain, apparent study type, relationship to neuronal excitability, and relevance to seizure occurrence, EEG, clinical semiology or treatment response. The central distinction was between direct seizure evidence and indirect contextual evidence. Only direct studies measuring seizure outcomes could justify a claim about seizure risk, discrimination or prognosis.

2.4. Critical appraisal. Because article-level methods and data were not uniformly accessible for all entries, no formal risk-of-bias scores were assigned. Journal supplements and conference abstracts were treated as preliminary or abbreviated communications when full methods were unavailable. Two records with matching titles and author lists but different journal venues were retained as supplied; they were not assumed to represent independent patient cohorts.

2.5. Synthesis strategy. Findings were organized by mechanisms, phenotype and clinical applicability. No odds ratios, hazard ratios, sensitivities, specificities, group sizes or p values were imputed. Where evidence was mechanistic rather than clinically tested, the discussion uses conditional language. This approach seeks to provide a clinically useful synthesis while preserving the limits of the underlying source set.

2.6. Ethical considerations. The work involved no direct human recruitment, biological sampling or identifiable patient data. Accordingly, no newly acquired clinical consent or intervention ethics approval is claimed. Any subsequent prospective study would require institutional approval and a prespecified data-protection plan.

3. RESULTS

3.1. Landscape of the included evidence

The source set contains 33 bibliographic entries corresponding to the three author-centered groups provided: 11 associated with Gazieva, 11 associated with Daminova and 11 associated with Akramova. The grouping describes the provenance of the source list rather than independent data sets. Because author groups overlap, the number of records cannot be equated to the number of distinct patient samples.

Vascular and postinfectious publications address the neurological context of ischemic stroke, COVID-19 and rehabilitation. Parkinsonian publications address fluctuating motor symptoms, axial and olfactory manifestations, nonmotor function, familial susceptibility and neurodegenerative disease progression. The biomarker-oriented subset focuses on MCP-1, alpha-synuclein and markers of neuronal injury in Parkinson disease and Parkinson-plus conditions.

Crucially, this thematic spread does not form a body of direct epilepsy studies. The results of this review therefore describe evidence relevance and identifiable gaps rather than estimates of seizure prevalence. Bibliographic coverage of a neurological topic cannot substitute for direct measurement of seizures or epileptiform EEG phenomena.

Evidence cluster	Examples from supplied research	Seizure-relevant interpretation
Vascular/post-COVID	Gazieva; Rakhimbaeva; Ataniyazov; Tuychiev	Cerebral injury mechanisms may provide context; seizure endpoints not verified
Parkinsonian clinical phenotypes	Daminova; Akramova; Rakhimbaeva	Movement symptoms may mimic seizures; diagnostic distinction essential
Inflammatory / synuclein markers	Akramova and collaborators	Candidate biological pathways, not validated epilepsy tests

Neurorehabilitation	Gazieva; Rakhimbaeva; Akramova	Functional outcomes relevant, but prevention of seizures not demonstrated
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3.2. Cellular pathways connecting inflammation and excitability

Microglia and astrocytes are active regulators of neural homeostasis. Their signaling influences extracellular potassium handling, glutamate uptake, trophic responses and the structural stability of synaptic networks. Disturbance of those functions can modify neuronal firing thresholds. In an acutely injured brain, changes in extracellular neurotransmitter concentrations and ionic gradients may create a permissive environment for hypersynchronous activity.

Chemokine pathways contribute to immune-cell trafficking and glial communication. MCP-1/CCL2 is one such signal, but its circulating concentration is affected by systemic inflammation and many non-neurological processes. A higher or lower serum concentration in a Parkinsonian group cannot, without EEG-linked seizure assessment, establish a relationship with the timing or character of epileptic events.

Inflammatory effects may be time dependent. An early response can support debris clearance and repair, while persistent activation may adversely affect synaptic function or tissue integrity. Treating all neuroinflammation as uniformly harmful oversimplifies the biology and risks misleading interpretation of biomarker directionality.

The relevant level of biological measurement also matters. A plasma chemokine may not reflect the same process as a local inflammatory change in a specific cortical seizure network. Peripheral biomarker studies require careful attention to the blood–brain barrier, sampling time and potential systemic confounders before cortical mechanisms are inferred.

3.3. MCP-1 / CCL2 in the context of Parkinson-related research

Akramova and collaborators published reports concerning associations between serum MCP-1 and motor impairment severity, the marker's potential role in neuroinflammation, and the combined diagnostic interest of MCP-1 and alpha-

synuclein. These papers directly motivate attention to chemokine signaling in Parkinson disease and Parkinson-plus syndromes.

The interpretive endpoint in these reports is Parkinsonian disease characterization, not electroclinically confirmed seizures. Even if MCP-1 distinguishes certain neurodegenerative groups, that finding would not demonstrate an ability to differentiate an epileptic motor seizure from tremor or dystonia. Diagnostic targets, reference standards and patient spectra are different.

Translation into epilepsy would require defining whether MCP-1 is intended to predict first seizure, identify active epileptogenesis after a lesion, discriminate an event in the emergency department or track treatment response. A single nonspecific inflammatory measurement is unlikely to answer all four questions without independent validation.

3.4. Alpha-synuclein and synaptic pathology

The reviewed work on alpha-synuclein examines the molecular heterogeneity of Parkinson disease and related syndromes, including reported plasma differences between diagnostic groups. Alpha-synuclein is biologically relevant to synaptic organization and neurodegeneration, but differences in concentration need not correspond directly to neuronal network hyperexcitability.

Assays differ according to whether they quantify total, modified, aggregated or other molecular forms, and according to matrix and handling procedure. Preanalytical contamination and the time from sample collection to processing can further affect interpretation. Without harmonized methods, results from different platforms should not be pooled casually.

For an epilepsy study, alpha-synuclein would need comparison against established clinical and electrophysiological data, with appropriate control groups including Parkinsonian patients without seizures, patients with confirmed epilepsy from other etiologies, and individuals with nonepileptic motor events.

3.5. Neuroaxonal injury and disease severity

An additional Akramova publication addresses a neuroaxonal injury marker and its relationship to clinical characteristics of Parkinson disease and atypical parkinsonism. This approach reflects the growing interest in biochemical

measurements of neurological injury, but the marker should not be assigned a specific seizure threshold in the absence of seizure-focused evidence.

Neuroaxonal injury is not specific to epilepsy. Stroke, trauma, neurodegeneration, infection and systemic insults can all influence candidate measures of neuronal damage. Disease severity and biomarker levels can correlate while neither proves that a marker is causal nor that it improves diagnostic decisions for transient motor symptoms.

3.6. Vascular injury, infection and neuroinflammatory context

The vascular subgroup of the source set examines ischemic stroke, COVID-19-related cerebrovascular manifestations, postinfectious neurological outcomes and rehabilitation. These reports contextualize how acute structural disease may coexist with inflammation and neurological deficits. Cortical lesions and complications of severe systemic illness can create conditions in which acute symptomatic seizures must be considered.

The relationship is not automatic. An episode occurring after COVID-19 could be provoked by acute metabolic abnormalities, vascular injury, medication effects or another disorder; its explanation requires a specific clinical investigation. The diagnosis of epilepsy should not be assigned solely because the patient has a post-COVID history.

Long-term stroke outcomes also vary. Early seizures near the time of the vascular event and late unprovoked seizures do not have identical implications. The available rehabilitation titles do not provide direct evidence that any particular rehabilitation protocol independently reduces epilepsy incidence.

The broader clinical lesson is that the same biological signal may be produced by different pathologies. A biomarker influenced by vascular injury and Parkinsonian neurodegeneration would need adequate differential diagnostic specificity before it could help identify a seizure-related disease process.

3.7. Movement disorders as seizure mimics

Daminova and collaborators describe several Parkinson-related motor and nonmotor domains relevant to recognition of paroxysmal phenomena. Tremor is generally oscillatory; dystonia consists of sustained or intermittent patterned contractions;

dyskinesia often produces variable involuntary movements related to medication state. Myoclonus comprises brief jerks but is not necessarily epileptic.

Epileptic motor seizures may also produce stereotyped jerking, abnormal postures or a sudden behavioral arrest. Thus, a single descriptive label such as “shaking” is insufficient. The clinician must reconstruct sequence, onset, duration, awareness, triggers, medication timing and recovery.

Autonomic dysfunction and orthostatic hypotension in Parkinsonian patients may cause loss of consciousness or transient jerking associated with syncope. A reflex assumption that convulsive-looking movements equal epilepsy can lead to unnecessary antiseizure prescriptions while a cardiovascular mechanism remains untreated.

Observation	Potential mechanism	Key assessment
Brief, stereotyped focal jerks	Focal epileptic motor event or non-epileptic myoclonus	Detailed semiology; EEG when indicated
Rest-predominant oscillatory movement	Parkinsonian tremor	Movement examination and medication state
Twisting or sustained postures	Dystonia	Postural/task dependence; temporal pattern
Irregular choreiform movements	Dopaminergic dyskinesia	Relationship to levodopa dosing
Collapse with short jerks	Syncope or epileptic convulsion	Prodrome, ECG, eyewitness history
Variable events with atypical patterns	Functional or other paroxysmal episodes	Typical-event capture when clinically appropriate

3.8. Evidence gaps identified by the mapping exercise

First, the supplied bibliography does not contain a coherent series of seizure-defined cohorts paired with MCP-1 and alpha-synuclein testing. Second, available titles are not sufficient to establish whether biomarker sampling preceded, followed or

coincided with a neurological event. Third, EEG confirmation and standardized seizure classification are not documented across the source set.

Fourth, several records are supplement communications or have incomplete volume, issue or page metadata. Fifth, two supplied articles share the same title and listed authors but appear under different journal venues, raising the possibility of overlapping material. These issues limit quantitative aggregation and any assertion that the 33 records represent 33 independent clinical studies.

Finally, there is no basis in these records for constructing a biomarker-based clinical prediction score. The review can specify plausible relationships and research needs, but it cannot report diagnostic accuracy, risk ratios or treatment efficacy for seizure outcomes.

4. DISCUSSION

4.1. Biological plausibility versus clinical validity

The central finding of this review is conceptual: neuroinflammation and neurodegeneration are biologically relevant to neuronal network function, yet relevance does not equal clinical validation for seizures. A mechanism may operate across diseases while producing different phenotypes. The same chemokine can signal systemic immune activation in one setting and participate in local tissue responses in another.

Biomarker discovery proceeds through distinct stages: analytical performance, clinical association, incremental diagnostic value, external reproducibility and demonstration of usefulness in patient care. The supplied Parkinsonian literature contributes to the rationale for discovery. It does not, on its own, complete the later stages for epilepsy.

The site of neurological pathology matters. A focal cortical lesion can support seizure generation even when peripheral inflammatory measurements are unremarkable. Conversely, a systemic inflammatory signal can be elevated without clinical or electrographic evidence of an epileptic event. Clinical context must therefore remain the anchor of interpretation.

4.2. Designing clinically meaningful biomarker studies

A prospective study should first define its primary endpoint: for example, the occurrence of a first unprovoked seizure after stroke, electroclinically confirmed epilepsy in a neurodegenerative population, or differentiation of epileptic versus non-epileptic paroxysms in an outpatient clinic. Each endpoint has different sampling requirements and different sources of bias.

Participants should undergo standardized phenotyping, recording of vascular comorbidity, neurodegenerative diagnosis, medication exposure, inflammatory illness, renal function and relevant metabolic variables. Biomarker samples require a uniform protocol specifying specimen type, processing, storage, assay lot and quality controls.

EEG must be interpreted within clinical context. A normal routine EEG does not exclude epilepsy, and an isolated interictal abnormality does not automatically explain a witnessed event. In diagnostic-accuracy studies, the reference standard should ideally include expert adjudication of the typical event, supported by prolonged video-EEG when clinically appropriate.

Investigators should prespecify models, avoid data leakage between derivation and validation samples, and report calibration as well as discrimination. Incremental value above clinical history, EEG and imaging should be quantified rather than assumed. External validation in independent clinical settings is essential before biomarkers are advocated for routine care.

4.3. Clinical implications for neurological practice

For patients with a newly observed seizure-like episode, immediate priorities remain safety, identification of acute causes, recognition of prolonged convulsive activity and appropriate referral. Acute symptomatic seizures may reflect metabolic imbalance, infection, hemorrhage or ischemia. Biochemical research biomarkers should not delay emergency stabilization or established diagnostic procedures.

In Parkinson disease, clinicians should actively consider medication-associated dyskinesia, dystonia, orthostatic phenomena and cognitive fluctuations. The episodic quality of a movement is not enough to diagnose epilepsy. Conversely, a

person with Parkinson disease can also develop genuine epileptic seizures, particularly when other neurological pathology coexists.

In post-stroke and post-COVID contexts, clinicians must distinguish structural injury, acute physiological disturbances and late epileptogenic processes. Rehabilitation planning and seizure management are related aspects of care, but no rehabilitation method in the supplied bibliography can be assumed to prevent epilepsy without explicit seizure outcomes.

4.4. Relevance of the regional scientific literature

The regional reports offer a useful continuum of clinical problems encountered in neurologic practice in Uzbekistan: cerebrovascular injury, postinfectious deficits, Parkinsonian disability, cognition, motor manifestations and rehabilitation. Their inclusion promotes discussion of local research priorities and clinically relevant populations.

Nevertheless, responsible synthesis must distinguish the clinical question studied in each paper from the new question posed by the current review. Citations to Parkinsonian MCP-1 studies can support a statement that MCP-1 has been investigated in Parkinson disease. They cannot support a claim that MCP-1 predicts seizure frequency unless the published work actually assessed that endpoint.

This distinction is particularly important when manuscripts are submitted to peer-reviewed journals. Transparent evidence mapping, explicit limits and cautious language strengthen an article more than unsourced quantitative claims. The scientific value here lies in specifying a translational hypothesis and the tests required to assess it.

4.5. Limitations

This is a narrative review based on a predetermined set of 33 bibliographic entries rather than a reproducible search of all relevant databases. It may miss important epilepsy-specific evidence. Full texts were not uniformly inspected, and several entries have incomplete metadata. The resulting synthesis is therefore qualitative and exploratory.

The available information does not permit formal appraisal of risk of bias, patient overlap, assay methods or clinical endpoint definitions in every record. The source

list contains conference supplements and potentially overlapping publications. The review does not estimate seizure prevalence, establish associations statistically or recommend biomarkers for diagnostic use.

Future systematic reviews should define search strings, screening procedures and quality criteria, while prospective clinical studies should provide EEG-supported seizure ascertainment and standardized molecular protocols. Such designs are necessary to move from plausible mechanisms to clinically actionable evidence.

5. CONCLUSION

Neuroinflammation, neurodegeneration and vascular injury contribute to the biological complexity of neurological disease and can intersect with mechanisms governing neuronal excitability. MCP-1/CCL2, alpha-synuclein and neuroaxonal injury measures offer useful research perspectives in Parkinson disease and atypical parkinsonism.

However, the supplied 33 publications do not validate these molecules as seizure-specific biomarkers or demonstrate that biomarker-based classification improves clinical diagnosis. Epileptic seizures must still be differentiated from Parkinsonian tremor, dystonia, dyskinesia, syncope and other seizure mimics through rigorous clinical assessment and, when indicated, EEG and neuroimaging.

The most useful next step for research is a prospective, clearly defined electroclinical study integrating molecular samples, recognized seizure endpoints, imaging findings and external validation. Until such evidence becomes available, biomarker findings should be presented as exploratory and disease-context dependent.

PRACTICAL RESEARCH RECOMMENDATIONS

- Prespecify seizure type and whether the outcome is acute symptomatic seizure, late unprovoked seizure or documented epilepsy.
- Distinguish tremor, dyskinesia, dystonia, syncope and functional events using standardized clinical descriptions.
- Collect inflammatory and synuclein biomarkers with consistent specimen handling and analytical quality controls.

- Use EEG and neuroimaging as complementary evidence, not as interchangeable diagnostic substitutes.
- Report all negative as well as positive results, and validate predictive models in independent cohorts.

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