

PARKINSONIAN MOVEMENT DISORDERS, NEUROINFLAMMATORY BIOMARKERS AND SEIZURE MIMICS: A CRITICAL NARRATIVE REVIEW

Dilshoda Akramova¹, Dilxush Chori o'g'li Meyliyev²

¹ Tashkent State Medical University, Tashkent, Uzbekistan

² Neurologist, Interdistrict Center for Combined Injuries and Acute Vascular Diseases, Guzor District, Kashkadarya Region, Uzbekistan

ABSTRACT

Background: Parkinson disease and related neurodegenerative disorders generate diverse motor and nonmotor manifestations, some of which may resemble epileptic seizures. Concurrent research on inflammatory and neuronal injury biomarkers has expanded but is not equivalent to seizure biomarker validation.

Objective: To integrate the clinical literature on parkinsonism with neuroinflammatory and vascular studies, highlighting the differential diagnosis of seizure mimics and limits of biomarker interpretation.

Methods: A qualitative analysis of 33 supplied bibliographic records published between 2018 and 2026 was conducted. Thematically related sources were mapped to motor phenomenology, cognition, vascular comorbidity and molecular markers.

Results: Tremor, dystonia, dyskinesia, myoclonus, syncope and fluctuating attention present different diagnostic problems. MCP-1, alpha-synuclein and injury markers may inform neurodegeneration research but are not established seizure-specific tests in the reviewed publications.

Conclusion: Diagnosis must prioritize observed phenomenology and clinical context, reserving EEG and imaging for appropriate questions. Prospective seizure-focused validation of proposed molecular markers is necessary.

Keywords: Parkinson disease; atypical parkinsonism; tremor; dystonia; myoclonus; seizures; MCP-1; alpha-synuclein.

1. INTRODUCTION

Parkinson disease is commonly recognized through bradykinesia, rigidity and resting tremor, but its full clinical spectrum includes dystonia, motor fluctuations, dyskinesia, sleep disturbance, cognitive changes, autonomic dysfunction and

affective symptoms. These phenomena sometimes appear suddenly or episodically, resulting in concern about seizures. The presence of an involuntary movement, however, does not itself establish an epileptic mechanism.

Investigations by Daminova and colleagues address motor fluctuations, axial reflexes, olfactory impairment, prodromal biomarkers, nonmotor manifestations and genetic predisposition. Publications by Akramova and collaborators explore Parkinson-plus syndromes, temporal-rhythmic rehabilitation, MCP-1, alpha-synuclein and neuronal injury measures. Meanwhile the cerebrovascular studies led by Rakhimbaeva and Gazieva give important context for structural and vascular differential diagnoses.

The clinical consequences of confusing seizure mimics with epilepsy include unnecessary exposure to antiseizure medications, missed recognition of orthostatic hypotension or treatment-associated dyskinesia, and delayed correction of reversible disturbances. Conversely, confidently attributing every episodic phenomenon in an older person to Parkinson disease may conceal coexisting seizures from cortical injury.

This narrative review synthesizes the supplied literature to develop a differential diagnostic framework and critically examine whether molecular findings from degenerative syndromes can be applied to epileptic disease.

1.1. Rationale and scope of the review

A central diagnostic challenge is that an observed abnormal movement is a phenotype, not an etiological diagnosis. An identical-looking jerk may arise from cortical epileptic activity, non-epileptic cortical hyperexcitability, brainstem or spinal motor generators, medication-induced dyskinesia, or functional mechanisms. Conversely, focal seizures may present predominantly with autonomic, sensory or cognitive symptoms and little visible movement. In parkinsonism, autonomic dysfunction, sleep disorders, impaired attention and exposure to multiple medications broaden the differential diagnosis beyond conventional motor phenomenology.

The supplied regional bibliography offers an informative but indirect evidence base. Daminova and colleagues focused on familial characteristics, olfactory function,

motor fluctuations, axial reflexes, prodromal changes and nonmotor clinical outcomes in Parkinson disease. Rakhimbaeva and Akramova examined clinical, neuroimaging and cognitive questions in vascular parkinsonism. Akramova and collaborators subsequently examined circulating inflammatory and synuclein-associated markers in Parkinson and Parkinson-plus syndromes. These distinct lines of work motivate an integrated diagnostic discussion but do not constitute a cohort study of epilepsy among parkinsonian patients.

The review therefore separates three levels of inference: descriptions that can be attributed to the titles and scope of supplied papers; established clinical principles concerning seizures and movement disorders; and hypotheses requiring formal prospective testing. Maintaining this distinction prevents the common error of turning a biologically plausible association into an asserted clinical finding.

2. MATERIALS AND METHODS

The evidence base was the same prespecified set of 33 bibliographic entries from three author-related lists provided for this project. Sources include indexed journal reports, regional articles and conference supplements spanning 2018–2026. No database search completeness or systematic-review status is claimed.

Entries were grouped into parkinsonian motor manifestations, nonmotor and cognitive manifestations, neurodegenerative biomarkers, cerebrovascular and postinfectious disease, and rehabilitation. Each group was assessed for the relevance of its reported topic to episodic motor phenomenology and the distinction between direct evidence and plausible extrapolation.

The synthesis compared clinical patterns such as stereotypy, duration, relation to rest or movement, awareness, medication timing and post-event state. Biomarkers were assessed against the question of whether an association with neurodegenerative disease establishes specificity for epileptic seizures. No individual patient data were available and no quantitative estimates were calculated.

2.1. Evidence mapping and interpretation

The 33 supplied records were retained as a fixed source set to preserve the requested regional research context. Eleven entries concern publications associated with Gazieva and collaborators, eleven concern Daminova and collaborators, and eleven

concern Akramova and collaborators. This grouping reflects the supplied lists rather than mutually exclusive authorship: some studies include authors from more than one list. Duplicate-content risk is particularly relevant to the same-titled post-COVID rehabilitation items published in two different journals, and those entries were not assumed to represent independent datasets.

Sources were mapped to domains rather than ranked by citation count: (a) motor fluctuations and other parkinsonian movement phenomena; (b) prodromal, autonomic, cognitive and nonmotor presentations; (c) atypical and vascular parkinsonism; (d) alpha-synuclein, MCP-1 and injury-related biomarkers; and (e) stroke, postinfectious vascular injury and rehabilitation. The synthesis assessed what each domain contributes to an evaluation of potential seizure mimics, and what it cannot establish.

2.2. Definitions and safeguards against overinterpretation

In this review, a seizure denotes a transient occurrence of signs or symptoms due to abnormal excessive or synchronous neuronal activity. An epileptic motor seizure may be focal or generalized, whereas myoclonus, tremor, dystonia and dyskinesia are descriptive movement categories whose causes must be investigated separately. “Seizure mimic” designates an event that resembles an epileptic manifestation but may arise through a different mechanism. A normal interictal EEG cannot by itself exclude epilepsy, and incidental interictal abnormalities do not establish that every event is epileptic.

No numerical pooled outcomes were extracted from abstracts alone. No frequencies, effect sizes, diagnostic sensitivities, biomarker thresholds or treatment benefits are reported as if derived from the supplied set unless explicitly provided in an accessible bibliographic record. This is a methodological safeguard rather than a weakness to be concealed. The limitations of a narrative design include selection bias, variable publication types, incomplete bibliographic metadata and absence of independent full-text risk-of-bias assessment.

3. RESULTS

3.1. Phenomenology of parkinsonian motor events

Resting tremor is commonly oscillatory and may fluctuate with movement or posture. Off-period dystonia can involve painful sustained contraction, while levodopa-associated dyskinesia may result in irregular choreiform or dystonic activity. Motor fluctuations follow patterns related to medication exposure and disease progression. These features contrast with some epileptic motor seizures, which often display abrupt, stereotyped evolution, although no single sign is invariably diagnostic.

Daminova and coworkers focused on preventing or treating motor fluctuations and characterized nonmotor manifestations and disease progression. These papers support the existence of several clinically distinct motor and nonmotor syndromes, but do not provide comparative sensitivity or specificity estimates for seizure diagnosis.

3.2. Myoclonus, altered awareness and differential diagnosis

Myoclonus may reflect cortical, subcortical, spinal or peripheral generators and may be epileptic or nonepileptic. Establishing its origin requires the clinical context and, in selected cases, electrophysiological correlation. Brief jerks during movement, sleep transitions, metabolic illness or medication exposure should not automatically be labeled epileptic seizures.

Transient unresponsiveness can arise from autonomic failure, orthostatic hypotension, sleep attacks, medication effects and acute medical conditions. Cognitive impairment in vascular parkinsonism further complicates witness descriptions. A careful history should ask whether episodes are posture-dependent, linked to doses, accompanied by loss of tone or pallor, or followed by confusion.

3.3. Vascular and postinfectious comorbidity

Older patients with parkinsonism may also experience cerebrovascular lesions. Rakhimbaeva and Akramova reported on vascular parkinsonism and cognitive changes; Gazieva-associated papers address stroke, post-COVID neurological sequelae and rehabilitation. These sources explain why a single symptom may have competing etiologies and why structural assessment sometimes matters more than phenomenological labels alone.

In a patient with a new stereotyped focal event and a cortical stroke lesion, seizure should remain in the differential diagnosis even if established Parkinson disease is present. Conversely, repetitive limb movements that consistently track levodopa dosing may be better evaluated as treatment-associated dyskinesia.

3.4. Molecular biomarkers and interpretative limits

Akramova and collaborators investigated MCP-1 and alpha-synuclein in Parkinson disease and Parkinson-plus syndromes, including associations with motor impairment and diagnostic discrimination in these neurodegenerative populations. Other publications address neuronal injury measures and clinical-biochemical correlates in patients with comorbid type 2 diabetes.

MCP-1 is a chemokine participating in immune-cell trafficking; alpha-synuclein is relevant to synuclein-related neuropathology. Both are legitimate research candidates for understanding neural disease processes, but the supplied sources do not establish a seizure-specific threshold, sensitivity, specificity, or prospective seizure-prediction model. Inference from neurodegeneration to epileptogenesis must be clearly marked as hypothesis, not demonstrated result.

Table 1. Comparative clinical and methodological framework

Event type	Clue supporting diagnosis	Important alternative
Parkinsonian tremor	Oscillation at rest; context of bradykinesia	Focal rhythmic epileptic motor event
Dystonia/ dyskinesia	Patterned posture or dose-related movements	Tonic or hypermotor seizure
Myoclonus	Very brief jerks; variable generators	Epileptic myoclonic seizure
Transient unresponsiveness	Posture, blood pressure, medication context	Focal impaired-consciousness seizure
Focal convulsive episode	Stereotyped evolution, post-event findings	Limb-shaking ischemia or nonepileptic event

3.5. Medication-associated motor fluctuations and altered responsiveness

Motor symptoms in treated Parkinson disease can change substantially across the dosing cycle. Wearing-off may produce re-emergent tremor, painful foot dystonia or

immobility; peak-dose periods may bring choreiform or dystonic dyskinesia. Timing relative to levodopa or other dopaminergic medicines is therefore an essential element of the episode history. Medication diaries and caregiver videos help distinguish a recurrent medication-state phenomenon from a brief, stereotyped seizure. The Daminova motor-fluctuation publications are most relevant to this branch of the differential diagnosis, but they should not be cited as establishing epilepsy-specific treatment effects.

Transient loss of responsiveness poses a separate problem. Orthostatic hypotension is common in autonomic dysfunction and may cause syncope with brief convulsive movements resulting from reduced cerebral perfusion. A prodrome of dizziness, visual dimming or pallor, standing posture, and rapid recovery supports a possible syncopal mechanism; none is individually decisive. Sedative effects, sleep attacks, hypoglycemia, arrhythmia, infection and delirium may also produce fluctuating awareness. A cardiovascular evaluation, including orthostatic blood-pressure assessment and electrocardiography when appropriate, can be as important as EEG.

3.6. Focal neurological lesions and dual diagnoses

Vascular parkinsonism and idiopathic Parkinson disease may coexist with cerebrovascular lesions. Clinical and neuroimaging investigations by Rakhimbaeva and Akramova underscore the importance of separating an extrapyramidal syndrome from coexisting vascular disease. In a patient with a prior cortical infarct and a new stereotyped unilateral jerking episode, focal seizure becomes a meaningful diagnostic consideration even when the patient also has longstanding resting tremor. Conversely, a change in tremor amplitude after an ischemic event does not by itself indicate epileptic activity.

The Gazieva-related papers on ischemic stroke, post-COVID complications and rehabilitation provide context for structural injury, multimorbidity and recovery. They do not define seizure incidence in the present review. Acute ischemia, hemorrhage, metabolic disturbances and later cortical remodeling can contribute to seizure vulnerability in different time windows. Clinicians should document the relationship between events and an acute neurological insult, because distinguishing

acute symptomatic seizures from later unprovoked seizures may change prognosis and the rationale for long-term treatment.

3.7. A stepwise bedside differentiation strategy

First, obtain a detailed account of the first seconds of a typical event: resting or standing posture, medication phase, awareness, behavioral arrest, aura, autonomic symptoms and laterality. Second, characterize the movement itself: oscillatory tremor, sudden shock-like jerk, sustained abnormal posture, irregular flowing dyskinesia, or rhythmic clonic activity. Third, document duration, recurrence, sleep association, potential triggers and the immediate recovery period. Fourth, review medication changes and concomitant illness. Fifth, select targeted tests on the basis of the differential diagnosis rather than ordering every modality indiscriminately.

A routine EEG may support the diagnosis when epileptiform abnormalities are present in a compatible clinical context, but failure to capture an event is a major limitation. Extended ambulatory or inpatient video-EEG is valuable when an event is frequent, atypical, disabling or treatment-resistant and diagnosis remains uncertain. Surface electromyography and EEG–EMG back-averaging may help characterize difficult myoclonus in specialist settings. Brain MRI is particularly important when new focal signs or structural etiologies are suspected. Blood glucose, electrolytes and other investigations should be guided by clinical acuity.

3.8. Clinical examples illustrating diagnostic uncertainty

Illustrative scenario A: An older person with established Parkinson disease develops irregular involuntary trunk and limb movements approximately one hour after a dopaminergic dose, remains fully interactive, and experiences similar symptoms at comparable medication times. This pattern may suggest peak-dose dyskinesia rather than epilepsy. However, clinicians must review a typical event and consider alternative diagnoses if features are not consistent across episodes.

Illustrative scenario B: A patient with parkinsonism and recent cortical stroke experiences a sudden brief stereotyped sequence of unilateral hand jerking followed by several minutes of transient weakness. The event raises concern for a focal motor seizure with postictal weakness, although recurrent transient ischemia and other focal phenomena may require assessment. The example demonstrates why

background movement-disorder diagnoses must not obscure new cerebrovascular or epileptic pathology.

Illustrative scenario C: A patient with autonomic symptoms becomes pale after standing, collapses, has several brief limb jerks and rapidly returns to baseline while supine. Convulsive syncope is a key possibility; cardiac and orthostatic assessment should accompany neurological review. These vignettes are didactic examples, not patients from the referenced studies.

3.9. The role of nonmotor and prodromal research

Daminova and Saidvaliev examined olfactory function and prodromal biomarkers, while other Daminova publications address familial, genealogical and nonmotor aspects of disease evolution. Such work demonstrates that movement disorders extend beyond visible tremor and rigidity. It is clinically useful because transient cognitive or autonomic episodes may be misclassified when a movement-disorder diagnosis dominates the chart. However, olfactory dysfunction, prodromal Parkinson markers and family history do not confirm or exclude an epileptic seizure.

3.10. Molecular results as hypothesis-generating context

The Akramova-related publications report research programs centered on MCP-1, alpha-synuclein and markers of neuronal or axonal injury in Parkinson and Parkinson-plus syndromes. A chemokine concentration may reflect multiple systemic and central inflammatory influences, and peripheral blood measurements do not map directly onto a specific cortical epileptic network. Likewise, disease-associated alpha-synuclein biology is not equivalent to ictal electrophysiology. The existing reports can motivate integrated studies, but do not validate a circulating marker as a stand-alone classifier of seizure versus dyskinesia.

A useful next-stage design would combine prospective event characterization, blinded specialist adjudication, synchronized video-EEG when feasible, structural imaging, medication-state documentation, and standardized biomarker sampling. Prespecified comparisons could distinguish participants with electroclinically confirmed seizures, clinically defined movement abnormalities without seizures, and relevant vascular comorbidity. Statistical analyses would need adjustment for

age, systemic inflammation, renal function, dopaminergic therapy and severity of brain injury. Without this design, biomarker associations remain exploratory.

4. DISCUSSION

At the bedside, the first diagnostic task is to describe the phenomenon before assigning a disease label. Ask whether movements are rhythmic or irregular, continuous or abrupt, unilateral or bilateral, stimulus-sensitive or medication-dependent. Determine whether awareness is interrupted and whether there is clear post-event confusion or weakness. Obtain eyewitness video when feasible, and review concomitant drugs and systemic illness.

Some epileptic events have no large motor component, whereas several nonepileptic syndromes cause dramatic involuntary movements. EEG may support seizure diagnosis when interpreted with the clinical history, but a normal routine EEG cannot rule out epilepsy. Video-EEG can be especially helpful for capturing uncertain recurrent events. MRI is useful when focal structural disease is suspected, and cardiovascular evaluation matters for collapse or suspected syncope.

The molecular narrative should remain equally disciplined. Biomarkers that correlate with motor disability or neurodegenerative phenotypes are not automatically suitable for differentiating epileptic from nonepileptic paroxysms. Validation would require independent patient cohorts, standardized assay handling, blinded seizure adjudication, clear clinical comparators and calibration of predictive performance.

Although all 33 supplied bibliographic records are cited in the reference list, their levels of relevance differ. Publications addressing stroke and COVID-19 contribute contextual information on comorbidity; parkinsonism papers provide direct background on motor phenomenology; biomarker papers support molecular hypotheses. Some items are abstracts, some are incompletely specified, and two same-titled rehabilitation records may overlap.

A future prospective study could recruit patients with Parkinson disease and atypical parkinsonism who report new paroxysmal episodes. Standardized video documentation, orthostatic vital signs, medication diaries, EEG when appropriate and structural imaging would enable blinded event classification. Measuring MCP-

1, alpha-synuclein or neuronal injury biomarkers should be an exploratory adjunct with prespecified analytical controls, not the diagnostic reference standard.

4.1. Diagnostic consequences and clinical priorities

Mislabeling dyskinesia or tremor as epilepsy may expose patients to adverse effects and drug interactions without addressing the driving movement disorder. Conversely, assuming that every episodic movement in a person with Parkinson disease reflects parkinsonism can delay recognition of a first seizure, stroke, arrhythmia or metabolic emergency. The practical objective is not to force every episode into a single neurological diagnosis but to preserve multiple reasonable hypotheses until the temporal and electrophysiological evidence becomes adequate. The differential diagnosis is especially demanding when dementia, communication difficulties or poor event recall limit direct history. Caregiver descriptions and home recordings may be more informative than retrospective terminology such as “convulsion” or “attack.” Examination between episodes should assess bradykinesia and rigidity, orthostatic physiology, focal neurological deficits and medication-related movement patterns. Specialist referrals should be driven by the consequences of diagnostic uncertainty and the possibility of acute risk.

4.2. Implications for rehabilitation and multidisciplinary care

The rehabilitation publications in the supplied bibliography provide an important reminder that a correct diagnostic label is only one component of care. Neurological recovery may involve mobility, cognitive performance, swallowing, communication, fall prevention and the capacity to perform activities of daily living. In parkinsonian patients, structured exercise, therapy planning and medication timing need to account for episodes of involuntary movement or transient unresponsiveness. After stroke, rehabilitation decisions must also accommodate fatigue, vascular comorbidities and possible recurrent neurological events.

A multidisciplinary model can involve a movement-disorder neurologist, epilepsy specialist, rehabilitation physician, physiotherapist, nurse and—when indicated—cardiology and clinical neurophysiology services. This approach is particularly relevant to older adults with simultaneous Parkinson disease, cerebrovascular disease and polypharmacy. The supplied references are useful for contextual

discussion of rehabilitation and vascular burden; however, the current review does not establish that any single rehabilitation protocol reduces seizures.

4.3. Research gaps and evidence quality

The major gap is the absence of direct event-level comparative data connecting the supplied movement-disorder literature with electrophysiologically verified seizures. Many records are local clinical reports or conference-supplement abstracts; some provide incomplete journal metadata. Cross-sectional biomarker findings are especially prone to confounding and cannot resolve whether inflammatory signaling precedes neurological deterioration or reflects its consequences. Even detailed phenomenological observations may be biased if diagnostic certainty is not prespecified.

Future multicenter prospective work should define index events in advance, distinguish incident seizures from recurrent movement phenomena, record focal structural lesions and cerebrovascular risk, and follow outcomes after differential diagnosis is established. Primary endpoints might include the proportion of events adjudicated as epileptic, interobserver agreement on video phenomenology, and incremental diagnostic yield of EEG beyond structured clinical assessment. Secondary endpoints could include falls, emergency attendance, quality of life, inappropriate antiseizure exposure and changes in rehabilitation participation.

Reporting should follow an appropriate observational or systematic-review guideline for the actual study design, with transparent inclusion criteria, missing-data handling, and protocol registration where relevant. Until these studies are available, conclusions should remain clinically cautious: the 33 supplied publications show a broad regional body of research in vascular and degenerative neurology, not a pooled evidence base for seizure biomarkers or seizure prevalence in Parkinson disease.

5. CONCLUSION

Paroxysmal motor and nonmotor manifestations in Parkinson disease and atypical parkinsonism are heterogeneous and may be mistaken for epilepsy. Classification requires careful characterization of tremor, dystonia, dyskinesia, myoclonus and impaired responsiveness, particularly in patients with vascular comorbidity.

The reviewed MCP-1, alpha-synuclein and neuroaxonal marker publications inform broader mechanistic research but do not yet validate seizure diagnosis or prediction. Clinically grounded, prospective work linking phenotyped events to EEG and biomarker data is the necessary next step.

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