

CLINICAL DIVERSITY OF SEIZURES AND SEIZURE-LIKE MOTOR PHENOMENA IN NEUROLOGICAL DISORDERS: A NARRATIVE REVIEW OF CEREBROVASCULAR AND NEURODEGENERATIVE LITERATURE

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ABSTRACT

Background: Epileptic seizures and nonepileptic motor paroxysms frequently enter the differential diagnosis of neurological disease. Vascular injury, inflammation, degenerative circuit dysfunction, medication effects, and metabolic disturbances may generate superficially similar manifestations through different mechanisms. **Objective:** To synthesize the clinical distinctions between epileptic events and seizure-like motor phenomena in cerebrovascular and neurodegenerative disorders, while critically situating regional neurological literature within contemporary seizure-classification concepts. **Methods:** A narrative thematic review of 33 bibliographic entries supplied by the authors (2018–2026) was undertaken. Publications were grouped by ischemic stroke and post-COVID sequelae, Parkinson disease and parkinsonism, rehabilitation, and molecular biomarkers. International classification principles were used for interpretation; no patient-level data were obtained. **Results:** Three interconnected clinical domains emerged: cortical structural injury with potential epileptogenesis; basal-ganglia disorders with tremor, dystonia, and dyskinesia that may mimic seizures; and inflammatory/degenerative pathways that offer mechanistic hypotheses but not validated seizure biomarkers. The source set is predominantly non-seizure-specific, limiting direct estimates of seizure occurrence. **Conclusions:** Reliable clinical classification depends on episode semiology, temporal course, consciousness, examination, electroencephalography, and brain imaging. Disease-specific prospective studies are required to assess seizure frequency, biomarkers, and outcomes.

Keywords: seizures; focal epilepsy; stroke; Parkinson disease; atypical parkinsonism; neuroinflammation; MCP-1; alpha-synuclein; differential diagnosis.

1. INTRODUCTION

Paroxysmal neurological symptoms range from transient sensory phenomena and alterations in responsiveness to dramatic bilateral motor activity. Their visible appearance does not establish etiology. An epileptic seizure reflects abnormal neuronal electrical activity, whereas tremor, dystonia, dyskinesia, syncope with convulsive movements, and some forms of myoclonus can arise without an epileptic mechanism. Misclassification risks both unnecessary exposure to antiseizure drugs and failure to diagnose an urgent underlying vascular, metabolic, or inflammatory disorder.

The modern framework for describing epileptic seizures distinguishes focal, generalized, unknown whether focal or generalized, and unclassified events. This is best understood as a classification of epileptic phenomena, not of every involuntary movement seen by neurologists. For a clinically meaningful diagnosis, the physician must distinguish the description of an event from the etiology of a neurological disease and from the possibility that multiple diagnoses coexist in one patient.

The supplied literature is valuable because it addresses three major environments in which this distinction is relevant. Gazieva and colleagues have studied ischemic stroke, post-COVID neurological complications, and rehabilitation (Khasanov et al., 2024; Rakhimbaeva et al., 2023; Ataniyazov et al., 2021). Daminova and colleagues have examined Parkinson disease, its motor and nonmotor manifestations, genetics, and progression (Daminova et al., 2019; Daminova & Matmurodov, 2022). Akramova and colleagues have investigated parkinsonian phenotypes and exploratory inflammatory or neuroaxonal biomarkers (Akramova et al., 2025; Rakhimbaeva & Akramova, 2020). Although these studies do not constitute a seizure-focused clinical cohort, they provide a suitable platform for developing a critical and clinically oriented review.

The objective of the present paper is to synthesize seizure-relevant mechanisms in vascular and degenerative neurological conditions; describe practical distinctions between epileptic and nonepileptic paroxysmal events; and identify what can, and cannot, be inferred from this selected regional bibliography.

2. MATERIALS AND METHODS

2.1. Review design and sources

This work is a narrative, not systematic, literature review. The source set consisted of 33 bibliographic records supplied for three research groups: 11 containing Gazieva, 11 containing Daminova, and 11 containing Akramova. Publication years range from 2018 to 2026. Records include full-length articles, material appearing in journal supplements, and articles with incomplete page-level metadata. Some publications have closely overlapping titles and author lists, so the number of bibliographic entries should not be interpreted as the number of independent clinical datasets.

2.2. Analytical framework

Records were coded thematically by apparent subject from their titles and available bibliographic information: (1) cerebrovascular and postinfectious disorders; (2) rehabilitation and functional neurological consequences; (3) Parkinson disease and atypical parkinsonism; and (4) molecular markers and mechanisms. The clinical synthesis further distinguished acute symptomatic seizures, unprovoked seizures associated with enduring cerebral pathology, and nonepileptic movement phenomena. We summarized plausible physiological mechanisms without attributing specific unreported numerical findings to individual publications.

2.3. Evidence interpretation and limitations

The analysis did not extract patient counts, electroencephalographic recordings, effect sizes, incidence estimates, or comparative treatment outcomes because these could not be established reliably from the bibliography alone. No database-wide systematic search, formal risk-of-bias evaluation, or meta-analysis was performed. The manuscript therefore describes convergent topics, clinical interpretation, and research gaps rather than presenting newly measured outcomes. Bibliographic information was transcribed from the supplied source list, and incomplete publication fields were not invented.

3. RESULTS

3.1. Thematic composition of the selected literature

The titles cluster strongly around ischemic stroke and neurological recovery, parkinsonian motor and nonmotor syndromes, and experimental or translational

neurodegeneration markers. None of these clusters offers a direct denominator-based assessment of seizure incidence across diagnoses. Instead, they address the background disease processes, neurological syndromes, and diagnostic challenges in which seizures may be considered. The 33 references are retained individually for transparency, including a pair of apparently related publications with identical study titles in different journals (Rakhimbaeva et al., 2022a, 2022b).

Source cluster	Representative subjects	Direct relevance to seizure evaluation
Cerebrovascular / post-COVID	Ischemic stroke, thrombotic complications, rehabilitation	Potential cortical injury, acute neurological events, structural differential
Parkinsonism / movement disorders	Tremor, fluctuations, axial signs, cognition	Distinguishing epilepsy from nonepileptic motor phenomena
Biomarkers / neuroinflammation	MCP-1, alpha-synuclein, neuroaxonal injury	Hypothesis generation; no validated seizure prediction

3.2. Cerebrovascular injury and seizure susceptibility

Brain ischemia may disrupt ATP-dependent ion transport, extracellular potassium homeostasis, glutamatergic signaling, and neuronal membrane stability. Acute symptomatic seizures can arise near the time of ischemic or hemorrhagic injury, especially when cortical tissue is affected. Delayed epileptogenesis may involve gliosis, altered inhibitory circuits, synaptic reorganization, and persistent injury-related network instability. Acute and delayed seizures should not be conflated: an early event may reflect a transient insult, whereas later unprovoked events may indicate a persistent propensity for recurrent seizures.

The supplied publications examine ischemic stroke following COVID-19, clinical patterns of vascular complications, and rehabilitation strategies. Rakhimbaeva et al. (2023), Rakhimbaeva and Gazieva (2022), and Gazieva (2021) focus on post-COVID ischemic stroke and recovery; Rakhimbaeva et al. (2020) address post-stroke rehabilitation; Ataniyazov et al. (2021) address stroke characteristics in

COVID-19. Two records entitled “Some aspects of rehabilitation of post-COVID patients with ischemic stroke” appear in different journals (Rakhimbaeva et al., 2022a, 2022b). These contributions indicate relevant clinical settings, but their bibliographic entries cannot substantiate a particular rate or phenotype distribution of post-stroke epilepsy.

Other vascular and postinfectious publications consider neurological consequences of coronavirus infection (Tuychiev et al., 2021), the role of discirculatory encephalopathy in cardiovascular disease (Rakhimbaeva & Gazieva, 2024a), and rehabilitation following stroke or traumatic brain injury (Rakhimbaeva & Gazieva, 2024b). A study of cavernous sinus thrombosis and neurological complications (Bobamuratova et al., 2021) broadens the differential context. Systemic inflammation, hypoxemia, medication exposure, organ failure, and cerebrovascular injury may coexist in critically ill patients; therefore, temporal association with infection does not establish that a seizure is caused directly by the virus.

3.3. Parkinson disease and seizure-like involuntary movement

Parkinson disease is characterized by bradykinesia with rest tremor or rigidity, reflecting dysfunction of dopaminergic and distributed basal-ganglia–thalamo–cortical networks. Although epileptic seizures can occur in patients with Parkinson disease, the common motor features of parkinsonism do not imply epilepsy. A resting tremor is frequently rhythmic and sustained or fluctuating; dystonia produces patterned postures or twisting movements; levodopa-related dyskinesia can generate irregular involuntary movements. Brief myoclonic jerks warrant especially careful interpretation because they may be epileptic or nonepileptic.

Daminova and colleagues provide a structured body of work relevant to this differential diagnosis: motor fluctuation prevention and treatment (Daminova et al., 2019), diagnostic approaches (Daminova, 2022a, 2022b), nonmotor features and progression (Daminova, 2022c, 2022d), axial reflexes (Daminova, 2022e), familial and genealogical characteristics (Daminova, 2022f, 2022g), prodromal biomarkers (Daminova & Saidvaliev, 2022a), olfaction (Daminova & Saidvaliev, 2022b), and early disease-risk assessment (Daminova & Matmurodov, 2022). These studies

reinforce the diversity of parkinsonian manifestations, but do not establish an electrographic seizure diagnosis for tremor or dyskinesia.

Related studies examine vascular parkinsonism with neuroimaging findings (Rakhimbaeva & Akramova, 2018), cognitive changes in Parkinson disease and vascular parkinsonism (Rakhimbaeva & Akramova, 2020), rehabilitation exercise approaches (Akramova & Rakhimbaeva, 2021), and the clinical manifestations of Parkinson-plus syndromes (Rahimboyeva et al., 2025). Cognitive fluctuation, sudden immobility, loss of posture, sleep-related symptoms, orthostatic hypotension, and medication effects may complicate event interpretation. Careful analysis of onset, triggers, medication phase, responsiveness, recovery, and witnessed video can prevent treating an extrapyramidal movement as epilepsy.

3.4. Neuroinflammation and exploratory biomarkers

Inflammatory signaling and glial activation can influence synaptic transmission, blood–brain barrier function, and extracellular ionic regulation. Such processes are biologically relevant to neuronal excitability, but links are disease- and context-dependent. MCP-1/CCL2 is a chemokine involved in immune-cell signaling, whereas alpha-synuclein is central to research on synucleinopathies. Their investigation in Parkinson disease should not be taken as evidence that either protein is a validated diagnostic marker for epileptic seizures.

Akramova and collaborators have reported research on associations between serum MCP-1 and motor impairment, the possible diagnostic roles of MCP-1 and alpha-synuclein, and circulating alpha-synuclein in Parkinson disease versus Parkinson-plus syndromes (Akramova et al., 2025a, 2025b, 2025c; Akramova et al., 2026). Other publications address neuroaxonal injury indicators (Akramova, 2025), clinical and biochemical characteristics in Parkinson disease with type 2 diabetes (Rakhimbaeva et al., 2025), and effects of a drug combination on the central nervous system (Khasanov et al., 2024). Collectively, these topics justify hypotheses concerning interactions among inflammation, metabolic stress, and neuronal networks, not clinical seizure-biomarker claims.

3.5. Practical differential diagnosis

A structured event history remains the diagnostic cornerstone. Clinicians should obtain eyewitness accounts or smartphone recordings, determine whether events are stereotyped, document the pattern and spread of movements, identify provoking factors, and assess consciousness and recovery. An event lasting seconds with stereotyped focal progression may raise suspicion of epilepsy, but isolated characteristics are rarely definitive. Prolonged or situational rhythmic movements may suggest tremor; painful patterned posturing may suggest dystonia; and repetitive movements tied to dopaminergic medication cycles may suggest dyskinesia.

Assessment domain	Features to document	Potential implication
Temporal characteristics	Onset, duration, recurrence, stereotypy	Seizure versus sustained/fluctuating movement disorder
Consciousness	Responsiveness, recollection, post-event confusion	Supports differential but is not alone diagnostic
Motor semiology	Rhythmicity, laterality, tonic/clonic pattern, spread	Focal seizure versus tremor/dystonia/dyskinesia
Context	Stroke timing, infection, medication, glucose, electrolytes	Acute symptomatic cause or movement-disorder trigger
Tests	EEG/video-EEG, MRI, targeted laboratory investigations	Evidence for epileptic activity and underlying disease

A normal routine EEG does not exclude epilepsy, and movement artifacts may complicate interpretation. Video-EEG is particularly useful when habitual episodes can be captured. MRI and laboratory tests address structural, metabolic, infectious, or inflammatory causes, and cardiac assessment should be considered when syncope is plausible. Prolonged bilateral convulsive activity or recurrent seizures without

return to baseline requires urgent emergency assessment rather than routine outpatient differential workup.

4. DISCUSSION

A principal finding of this review is conceptual rather than numerical: the same outward motor symptom may originate from different neural systems, and the same neurological disease may produce multiple kinds of paroxysmal episodes. The vascular literature explains why damaged cortex may become hyperexcitable, while the parkinsonism literature illustrates how basal-ganglia circuit dysfunction creates motor symptoms that can superficially resemble epilepsy. This distinction between mechanism and appearance is essential for safe clinical reasoning.

Stroke-related seizures and parkinsonian movements also differ in their relationship to disease timing. Acute ischemic injury may produce a seizure during a defined physiological crisis. Post-stroke epilepsy may instead emerge after structural reorganization. Parkinsonian tremor and dyskinesia commonly vary with medication state, motor task, and overall progression. Applying one uniform “convulsion” label across these conditions obscures clinically actionable distinctions.

The biomarker literature represents a developing translational domain. Studies of MCP-1 and alpha-synuclein offer potentially useful biological context, yet they do not demonstrate the sensitivity, specificity, independent prognostic value, or clinical thresholds required to diagnose seizures. Demonstrating seizure relevance would require prospectively phenotyped patients, systematic video-EEG, appropriate control groups, adjustment for disease severity and medications, and reproducible biomarker measurement protocols.

The selected bibliography also highlights the role of neurological rehabilitation after vascular damage and in neurodegenerative syndromes. Rehabilitation targets function, independence, mobility, cognition, and quality of life. It should not be portrayed as independently preventing epilepsy without direct comparative evidence. Similarly, findings regarding clinical recovery after COVID-19-associated stroke cannot be transferred automatically to postinfectious epilepsy risk.

Several methodological limitations require explicit acknowledgment. First, the 33 entries were purposefully supplied rather than retrieved through a reproducible

systematic search, creating selection bias. Second, some are conference supplements or have incomplete metadata, limiting confidence in detailed methods and outcomes. Third, near-duplicate titles suggest potential overlap of reported patient material. Fourth, none of the included works was used here to calculate seizure prevalence, associations, or treatment effects. Finally, the evidence is thematically heterogeneous, so it is unsuitable for pooling into a single effect estimate.

The next research step should be a prospective multicenter observational study that prospectively records neurological diagnosis, imaging-confirmed lesion type and location, comorbid conditions, medications, event semiology, EEG/video-EEG confirmation, and longitudinal outcomes. Patients with stroke and parkinsonian disorders should be analyzed separately, and nonepileptic movement phenomena should be adjudicated by standardized criteria. Only then can clinically meaningful risk estimates and biomarker hypotheses be tested.

5. CONCLUSION

Seizures and seizure-like motor events in neurological disease are heterogeneous in mechanism and presentation. Cerebrovascular pathology may produce acute symptomatic seizures or an enduring epileptogenic state; Parkinson disease and atypical parkinsonism more commonly produce nonepileptic tremor, dystonia, dyskinesia, and fluctuating motor symptoms. Neuroinflammatory biomarkers are scientifically relevant but cannot currently be inferred from this selected bibliography to diagnose epilepsy. Integrated clinical assessment, EEG when indicated, brain imaging, and cause-directed investigations are essential. The reviewed 33 entries provide regional disease-specific context and a foundation for prospective, seizure-focused research, rather than a quantitative estimate of seizure burden.

REFERENCES

- Akramova, D. T. (2025). Маркер нейроаксонального повреждения — соотношение уровней с клиническими параметрами при болезни Паркинсона и синдромах атипичного паркинсонизма. Журнал неврологии и нейрохирургических исследований.
<https://tadqiqot.uz/index.php/neurology/article/view/12336>

- Akramova, D. T., & Rakhimbaeva, G. S. (2021). Application of temporhythmic correction neurorehabilitation exercise complex in Parkinson's disease. *Journal of the Neurological Sciences*, 429(Supplement), Article 119547. <https://doi.org/10.1016/j.jns.2021.119547>
- Akramova, D. T., Rakhimbaeva, G. S., Bobomuratov, T., & Bobamuratova, D. (2025b). Biomarkers at the crossroads of inflammation and degeneration in Parkinson's disease: The diagnostic potential of MCP-1 and alpha-synuclein. *Journal of the Neurological Sciences*, 480(Supplement), Article 124207. <https://doi.org/10.1016/j.jns.2025.124207>
- Akramova, D. T., Rakhimbaeva, G. S., Bobomuratov, T., Bobamuratova, D., & Ibodov, B. (2025a). Association between serum MCP-1 levels and motor impairment severity in Parkinson's disease and Parkinson-plus syndromes. *Journal of the Neurological Sciences*, 480(Supplement), Article 124206.
- Akramova, D. T., Rakhimbaeva, G. S., Bobomuratov, T., Bobamuratova, D., & Ibodov, B. (2025c). MCP-1: The role of a novel biomarker in assessing neuroinflammation in Parkinson's disease and Parkinson-plus syndromes. *Journal of the Neurological Sciences*, 480(Supplement), Article 124205. <https://doi.org/10.1016/j.jns.2025.124205>
- Akramova, D. T., Rakhimbaeva, G. S., & Bobomuratov, T. (2026). Differences in plasma alpha-synuclein levels in Parkinson's disease and Parkinson-plus syndromes. *Neuromodulation: Technology at the Neural Interface*, 29(1, Supplement), S164. <https://doi.org/10.1016/j.neurom.2025.09.248>
- Ataniyazov, M. K., Rakhimbaeva, G. S., & Gazieva, Sh. R. (2021). Характерные особенности инсульта при COVID-19. *Журнал неврологии и нейрохирургических исследований*, (2), 14–17. <https://www.tadqiqot.uz/index.php/neurology/article/view/1348>
- Bobamuratova, D., Boymuradov, S., Kurbanov, Y., & Akramova, D. (2021). Clinical analysis of complications of cavernous sinus thrombosis in COVID-19 patients. *Journal of the Neurological Sciences*, 429, Article 119786. <https://doi.org/10.1016/j.jns.2021.119786>

- Daminova, Kh. M. (2022a). Аксиальные рефлексy при болезни Паркинсона. Новый день в медицине, 5(43), 4–7.
- Daminova, Kh. M. (2022b). Modern view to optimization of the diagnosis of the course of Parkinson's disease. Новый день в медицине, 1(39), 115–118.
- Daminova, Kh. M. (2022c). Немоторные проявления и темп прогрессирования болезни Паркинсона. Новый день в медицине, 3(41), 406–409.
- Daminova, Kh. M. (2022d). Определение немоторных проявлений паркинсонизма в прогрессировании заболевания. Медицина и инновации, 2(6), 337–344.
- Daminova, Kh. M. (2022e). О семейной предрасположенности болезни Паркинсона. Неврология, 2(90), 4–6.
- Daminova, Kh. M. (2022f). Генеалогическая характеристика болезни Паркинсона. O'zbekiston Vrachlar Assotsiatsiyasi Byulleteni, 4(109), 125–128.
- Daminova, Kh. M. (2022g). Современный взгляд к оптимизации диагностики течения болезни Паркинсона. Journal of Neurology and Neurosurgery Research, 3(1), 49–52.
- Daminova, Kh. M., Khakimova, S. A., & Shermukhamedova, F. K. (2019). Профилактика и лечение моторных флюктуаций при болезни Паркинсона. Неврология, 2(78), 58–61.
- Daminova, Kh. M., & Matmurodov, R. J. (2022). Risk of forming Parkinson's disease at the early stages. Art of Medicine, 2(3), 235–240.
- Daminova, Kh. M., & Saidvaliev, F. S. (2022a). Роль биомаркеров продромальной стадии болезни Паркинсона в прогрессировании заболевания. Journal of Neurology and Neurosurgery Research, 3(2), 26–28.
- Daminova, Kh. M., & Saidvaliev, F. S. (2022b). Assessment of olfactory function in the study groups and their role in the progression of the disease. Eurasian Bulletin of Pediatrics, 1(12), 43–45.
- Gazieva, Sh. R. (2021). Модифицированная реабилитация постковидных больных с ишемическим инсультом. Неврология, (4), 14–17.
- Khasanov, U. L., Alimdzhanova, G. A., Mavlanov, Sh. R., & Gazieva, Sh. R. (2024). Влияние препаратов комбинации флурбипрофена, эноксолонa и

- хлоргексидина на центральную нервную систему. Eurasian Journal of Medical and Natural Sciences, 4(6), 349–350.
<https://doi.org/10.5281/zenodo.12736764>
- Rahimboyeva, G., Akramova, D., & Bobomurodova, D. (2025). Parkinsonizm “plyus” sindromining klinik va nevrologik xususiyatlari. Tibbiyot ta’limi va innovatsiyalari, 1(2). <https://doi.org/10.5281/zenodo.17586266>
- Rakhimbaeva, G. S., & Akramova, D. T. (2018). Клинические и нейровизуализационные особенности сосудистого паркинсонизма. Journal of Biomedicine and Practice, (2), 37–41.
- Rakhimbaeva, G. S., & Akramova, D. T. (2020). Взаимосвязь когнитивных изменений с гиперкортизолемией при болезни Паркинсона и васкулярном паркинсонизме. Журнал неврологии и нейрохирургических исследований, 1(1). <https://www.tadqiqot.uz/index.php/neurology/article/view/1391>
- Rakhimbaeva, G. S., Gazieva, Sh. R., Ataniyazov, M. K., Muratov, F. Kh., Tolipov, D. S., & Shodiev, U. D. (2023). Особенности лечения и реабилитация больных, перенесших COVID-19, с ишемическим инсультом. Журнал неврологии и психиатрии им. С. С. Корсакова, 123(3, вып. 2), 76–84.
<https://doi.org/10.17116/jnevro202312303276>
- Rakhimbaeva, G. S., Gazieva, Sh. R., Bobamuratova, D. T., Akramova, D. T., & Shodiyev, U. D. (2022a). Some aspects of rehabilitation of post-COVID patients with ischemic stroke. Journal of Pharmaceutical Negative Results, 13(Special Issue 7), 3262–3270. <https://doi.org/10.47750/pnr.2022.13.S07.425>
- Rakhimbaeva, G. S., Gazieva, Sh. R., Bobamuratova, D. T., Akramova, D. T., & Shodiyev, U. D. (2022b). Some aspects of rehabilitation of post-COVID patients with ischemic stroke. Journal of Physical Therapy and Sports Medicine, 6(6), Article 126. <https://doi.org/10.35841/aajptsm-6.6.126>
- Rakhimbaeva, G. S., Gazieva, Sh. R., & Muratov, F. Kh. (2020). Постинсультная реабилитация — методы и методики. Неврология, (3), 85–91.
- Rakhimbaeva, G. S., & Gazieva, Sh. R. (2022). Особенности реабилитации постковидных больных с ишемическим инсультом. Неврология, (4), 3–7.

Rakhimbaeva, G. S., & Gazieva, Sh. R. (2024a). Современный взгляд на роль дисциркуляторной энцефалопатии в сердечно-сосудистом континууме. Неврология, (4), 37–55.

Rakhimbaeva, G. S., & Gazieva, Sh. R. (2024b). Современные подходы к нейрореабилитации пациентов с последствиями инсульта или черепно-мозговой травмы. Неврология, (2), 61–70.

Rakhimbaeva, G. S., Musaeva, Y. A., Akramova, D. T., & Kasimova, O. O. (2025). Сравнительная оценка клинических, когнитивных и биохимических характеристик у пациентов с болезнью Паркинсона и сахарным диабетом II типа. Журнал неврологии и нейрохирургических исследований, 6(1).
<https://www.tadqiqot.uz/index.php/neurology/article/view/10979>

Tuychiev, L. N., Rakhimbaeva, G. S., Gazieva, Sh. R., & Ataniyazov, M. K. (2021). Новая коронавирусная инфекция и постковидные неврологические последствия заболевания. Вестник Ташкентской медицинской академии, (2), 45–50.

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