REVIEW ARTICLE

Stroke Mimics and Chameleons: A Practical Diagnostic Approach for the Clinician

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Abstract

Stroke remains a leading cause of death and long-term disability worldwide, with time-dependent reperfusion therapies offering substantial benefit when administered promptly. However, up to 30% of patients presenting with acute focal neurological deficits are ultimately diagnosed with stroke mimics-conditions that closely imitate stroke but have fundamentally different pathophysiology and management. Conversely, stroke chameleons are true cerebrovascular events that present with atypical, non-classical features, leading to dangerous diagnostic delay. This review provides a practical, evidence-based framework for distinguishing common stroke mimics from true ischemic events, recognizing stroke chameleons in atypical presentations, and making safe, informed decisions regarding thrombolysis when diagnostic uncertainty persists.

1. Introduction

Acute ischemic stroke demands rapid recognition and intervention; every minute of delayed reperfusion translates to millions of lost neurons. The advent of intravenous thrombolysis and mechanical thrombectomy has transformed outcomes, but these therapies carry risks and are contraindicated in non-vascular conditions. The diagnostic challenge is twofold: first, distinguishing stroke mimics from true stroke to avoid unnecessary, potentially harmful treatment; and second, recognizing stroke chameleons-true cerebrovascular events masquerading as benign conditions—to prevent catastrophic missed diagnoses. The concept of stroke mimics and chameleons was elegantly articulated by Moulin and Leys [1], who emphasized that the differential diagnosis of acute focal neurological deficits extends far beyond cerebrovascular disease.

Stroke mimics account for approximately 25-30% of all stroke presentations in emergency departments, with functional neurological disturbances representing the most frequent culprits [1]. Conversely, stroke chameleons-particularly posterior circulation events-are missed in up to 35% of initial assessments, with unrecognised cerebellar stroke associated with an eight-fold increased risk of death [1]. This review synthesizes current evidence to provide clinicians with practical diagnostic algorithms, bedside examination techniques, and management strategies for this challenging clinical scenario.

2. Stroke Mimics: The Great Imitators

2.1 Functional Neurological Disorder (FND)

Functional neurological disorder (FND) has emerged as the most common stroke mimic in contemporary practice, accounting for 28-30% of all mimic cases [2]. The condition is characterized by genuine neurological symptoms-often including hemiparesis, aphasia, or visual disturbance-without identifiable structural brain pathology. The pathophysiology involves aberrant neural network functioning rather than tissue destruction, making it a software problem rather than a hardware problem. A recent retrospective study from Trieste University Hospital analyzed 84 patients with FND presenting as stroke mimics between 2018 and 2022 [2].

The cohort was predominantly female (65.5%) with a mean age of 45 years, significantly younger than typical stroke populations. Hemiparesis or hemisensory loss was the presenting feature in 75% of cases, speech disorder in 10.7%, and vertigo or gait disorder in 4.8%. Notably, 32.1% had a history of psychiatric disorders, and 48.8% had prior neurological disease or stroke risk factors-highlighting that FND and vascular risk are not mutually exclusive [2]. Diagnostic Approach: The diagnosis of FND relies on positive clinical signs rather than the exclusion of other conditions.

Hoover's sign—where hip extension weakness paradoxically normalizes during contralateral hip flexion against resistance-demonstrates that motor pathways are intact but volitional control is impaired [3]. Other positive signs include give-way weakness, non-anatomical sensory loss, and entrainment (where tremor frequency changes to match rhythmic movements of the unaffected limb) [3]. Advanced neuroimaging, including CT perfusion and MRI, typically yields normal or non-specific results, while EEG shows normal background activity without epileptiform discharges [3]. Thrombolysis in FND: A critical management question arises when FND cannot be definitively excluded within the therapeutic window.

The Trieste study reported that thrombolysis was safely administered in 31% of FND patients without significant complications, and 44.7% experienced symptom resolution within 24 hours [2]. This aligns with pooled data suggesting that stroke mimics treated with intravenous thrombolysis have favorable outcomes and low intracranial hemorrhage rates [7]. The principle is clear: when diagnostic uncertainty exists, the potential benefit of thrombolysis in true stroke outweighs the risk in mimics [7]. Management: Once FND is recognized, management shifts from vascular protocols to a multidisciplinary approach.

Specialized physiotherapy emphasizing automatic movement retraining, cognitive behavioral therapy, and clear, empathic communication of the diagnosis are cornerstone interventions [2]. Stroke rehabilitation techniques— such as strengthening exercises or walking aids—are contraindicated, as they reinforce abnormal movement patterns [3].

2.2 Seizure and Postictal Phenomena

Seizures represent one of the most common neurological stroke mimics [6]. Todd's paralysis-transient postictal hemiparesis-can persist for hours, closely mimicking acute ischemic stroke. The phenomenon results from neuronal exhaustion and altered cortical excitability following seizure activity. Unlike stroke, Todd's paralysis gradually resolves, and the history may reveal witnessed seizure activity, though this is often unavailable in unwitnessed events [6].

Differentiating features include the presence of tongue biting (particularly lateral tongue injury), witnessed convulsive activity, and gradual rather than sudden onset of deficits [6]. However, these features are not always present, and the distinction can be challenging. EEG may demonstrate postictal slowing or, if performed during the event,epileptiform activity [6]. CT perfusion imaging in seizure-related stroke mimics can show focal hyperperfusion rather than the hypoperfusion characteristic of ischemia,though this finding requires expert interpretation [6]. 2.2.1 First-Onset Seizure as Stroke Mimic First-onset seizures in adults represent a particularly challenging stroke mimic, as they often present without a witnessed convulsive phase and may be mistaken for acute cerebrovascular events [25].

The postictal state can include focal deficits such as hemiparesis (Todd's paralysis), aphasia, or visual field defects, lasting from minutes to hours. Unlike recurrent seizures in patients with known epilepsy, first-onset seizures lack historical context, making the diagnosis entirely dependent on clinical acumen [25]. A critical clue is the gradual resolution of deficits over time, though this may not be apparent in the emergency setting. Neuroimaging is essential to exclude structural lesions-including acute stroke-that may have triggered the seizure.

EEG,if available, may show postictal slowing or epileptiform discharges, but a normal EEG does not exclude the diagnosis [25]. Management focuses on identifying the underlying cause, with antiepileptic therapy initiated if recurrence risk is high. Stroke protocols should be maintained until vascular etiology is definitively excluded,particularly in patients with vascular risk factors [25]. 2.2.2 Seizure as First Manifestation of Acute Stroke Conversely, seizures may represent the first manifestation of an acute stroke,particularly when the infarct involves the deep temporal lobe [30]. The temporal lobe,with its low seizure threshold and rich vascular supply from the middle and posterior cerebral arteries, is highly susceptible to epileptogenic transformmation following ischemic injury.

Patients presenting with a first-ever seizure in the context of acute neurological deficits may have an underlying stroke as the primary pathology, with the seizure being a secondary phenomenon rather than the primary diagnosis [30]. This creates a diagnostic paradox: the seizure mimics stroke (through postictal deficits),while simultaneously the stroke causes the seizure. Clinical vigilance is required to identify vascular risk factors, sudden onset of symptoms, and neuroimaging evidence of acute infarction. In such cases, management must address both the cerebrovascular event and seizure prophylaxis, with anticoagulation or antiplatelet therapy carefully timed to minimize hemorrhagic risk [30]. 2.2.3 Hemiballismus Mimicking Seizure Focal, violent, Flinging movements of the limbs-hemiballismus-due to infarction or hemorrhage in the contralateral subthalamic nucleus (Luys' body) may be mistaken for seizures [31].

The subthalamic nucleuus, a small diencephalic structure critical for motor control, is supplied by perforating branches of the posterior communicating artery and is highly vulnerable to small vessel disease. The resulting movements are coarse, high-amplitude, and proximal, involving the shoulder and hip in a throwing or kicking motion, typically unilateral [31]. Unlike epileptic seizures, hemiballismus is not associated with loss of consciousness, tongue biting, or postictal confusion. The movements are continuous rather than paroxysmal, and they persist during sleep- features that distinguish them from seizure activity [31].

However, in the hyperacute setting, the violent nature of the movements and the associated agitation may lead to misdiagnosis as convulsive status epilepticus, with inappropriate administration of benzodiazepines and antiepileptic drugs. Neuroimaging typically reveals a small lesion in the subthalamic region, and management focuses on dopamine receptor blockade (haloperidol, tetrabenazine) and treatment of the underlying vascular lesion [31].

2.3 Hemiplegic Migraine

Hemiplegic migraine (HM) represents a particularly treacherous stroke mimic,presenting with reversible unilateral motor weakness that can last hours to days [8].Both familial (FHM) and sporadic (SHM) forms exist, with mutations in CACNA1A,ATP1A2, and SCN1A genes implicated in FHM [9]. The condition is rare, with population prevalence estimated at 0.01%, but its stroke-like presentation makes it clinically significant [9]. The diagnostic differentiator lies in the temporal evolution of symptoms. In HM,aura symptoms develop gradually and sequentially-typically visual phenomena followed by sensory disturbance, then motor weakness, and finally speech impairment [9].

In stroke, these deficits occur simultaneously and suddenly. The ICHD-3 criteria require fully reversible motor weakness and at least one other aura symptom (visual,sensory, or speech) with duration of 5-60 minutes each, though motor weakness may persist longer [10]. A recent case report described a 45-year-old male with chronic hemiplegic migraine misdiagnosed as stroke, receiving tenecteplase before the correct diagnosis was established [8]. The patient had experienced similar episodes approximately ten times monthly, with preceding visual scotomas and photophobia-features that, if elicited in the history, would have strongly suggested migraine [8].

Triptans are contraindicated in HM due to vasoconstrictive properties; management focuses on preventive strategies including calcium channel blockers, lifestyle modification, and avoidance of triggers [9].

2.4 Hypoglycemia

Severe hypoglycemia represents a readily treatable metabolic stroke mimic that must be identified immediately. Blood glucose levels below 50 mg/dL can produce focal neurological deficits including hemiparesis, aphasia, and hemineglect-mimicking ischemic stroke with remarkable fidelity [11]. The mechanism involves asymmetric glucose transporter distribution and differential metabolic vulnerability of brain regions [12]. A Moroccan case series of six diabetic patients presenting with acute hemiplegia and blood glucose<50mg/dL demonstrated complete neurological recovery following intravenous glucose administration [11].

All had been initially misdiagnosed as stroke,with NIHSS scores ranging from 4 to 13. Notably, brain CT was normal in three cases and showed only chronic leukoencephalopathy in the fourth [11]. A more recent case report described a 60-year-old diabetic patient presenting with left-sided hemineglect and CT perfusion abnormalities mimicking acute ischemia; MRI ultimately excluded

stroke, and the patient recovered fully after glucose correction [12]. The clinical imperative is clear: blood glucose measurement must precede neuroimaging in all patients with suspected stroke, particularly those with diabetes or on insulin therapy [11].

The presence of autonomic symptoms (sweating, tachycardia,pallor) may provide a clue, though these are not universal [12]. Focal deficits from hypoglycemia may not resolve immediately after glucose correction, potentially misleading clinicians into pursuing stroke protocols [12].

2.5 Guillain-Barre■ Syndrome and Its Variants

Guillain-Barré syndrome (GBS) and its variants represent critical stroke mimics that can present with acute neurological deficits mimicking cerebrovascular events,particularly in the early stages when the classic ascending paralysis has not yet evolved [26]. The Miller Fisher syndrome (MFS) and Bickerstaff brainstem encephalitis (BBE) variants are especially treacherous, as they present with ophthalmoplegia, ataxia, and altered consciousness—features that overlap significantly with posterior circulation stroke [26]. 2.5.1 Miller Fisher Syndrome MFS is characterized by the classic triad of ophthalmoplegia, ataxia, and areflexia,with anti-GQ1b antibodies present in approximately 90% of cases [27]. The acute onset of diplopia and gait instability can be mistaken for vertebrobasilar stroke,particularly when the patient presents with isolated vertigo or bulbar dysfunction.Unlike stroke, MFS typically evolves over days rather than minutes, and the presence of antecedent infectious symptoms (often Campylobacter jejuni or Haemophilus influenzae) may provide a diagnostic clue [27]. CSF analysis demonstrates albuminocytological dissociation, and nerve conduction studies show reduced sensory amplitudes with preserved motor responses.

MRI is typically normal, though brainstem enhancement may occasionally be seen [27]. Management with intravenous immunoglobulin or plasma exchange is effective, with most patients achieving full recovery within weeks to months [27]. 2.5.2 Bickerstaff Brainstem Encephalitis BBE represents a more severe variant on the GBS spectrum, presenting with altered consciousness, ophthalmoplegia, and ataxia—closely mimicking basilar artery occlusion [28]. The presence of anti-GQ1b antibodies in over two-thirds of cases links BBE to MFS, and some authorities consider them part of a continuous clinical spectrum [28]. The key differentiating feature is the subacute evolution over days, with preceding infectious symptoms in many cases.

Unlike stroke, BBE may show CSF pleocytosis early in the course, and MRI may demonstrate T2 hyperintensity in the brainstem [28]. The prognosis is generally favorable with immunotherapy, though respiratory failure requiring mechanical ventilation occurs in approximately one-third of patients [28]. 2.5.3 Diagnostic Approach The distinction between GBS variants and stroke relies on clinical tempo, presence of antecedent infection, and ancillary investigations. In the hyperacute setting, when diagnostic uncertainty persists, the treat when uncertain principle applies-particularly for BBE, which can be indistinguishable from basilar occlusion [7]. However, the presence of bilateral facial weakness, ophthalmoplegia, or antecedent diarrhea should prompt consideration of GBS variants and urgent neurology consultation [26].

2.6 Other Important Mimics

Multiple Sclerosis (MS): Acute demyelinating relapses can present with unilateral weakness, visual loss, or sensory disturbance, particularly in younger patients. MRI typically demonstrates demyelinating plaques, and cerebrospinal fluid analysis may show oligoclonal bands [1]. Brain Tumors: Sudden presentation of a previously undiagnosed tumor—due to hemorrhage into the mass, seizure, or metabolic disturbance-can mimic stroke. Gradual symptom progression preceding the acute event and imaging characteristics (mass effect, contrast enhancement) facilitate differentiation [1].

Bell's Palsy: Unilateral facial weakness without limb involvement or cortical signs characterizes this peripheral seventh nerve palsy. The forehead is typically affected (unlike central lesions), and associated symptoms such as dysarthria or dysphagia are absent [1].

3. Stroke Chameleons: The Hidden Threat

3.1 Posterior Circulation Stroke Presenting as Acute Vestibular Syndrome

Posterior circulation stroke represents the quintessential stroke chameleon,frequently presenting with isolated dizziness or vertigo in the absence of other focal neurological signs [13]. Dizziness is reported in 47-75% of posterior circulation strokes, and isolated dizziness accounts for approximately 15% of all stroke misdiagnoses [13]. The acute vestibular syndrome (AVS)-defined as acute-onset, continuous vertigo with nausea, nystagmus, and postural instability—can result from either benign peripheral vestibular disease or life-threatening cerebellar/brainstem infarction [14]. The HINTS examination (Head Impulse, Nystagmus, Test of Skew) has revolutionized bedside differentiation of central from peripheral AVS [17].

A positive HINTS test—defined by any of three signs: normal horizontal head impulse, direction- changing nystagmus in eccentric gaze, or skew deviation—strongly suggests central pathology [17]. A systematic review of six studies(n=644) demonstrated pooled sensitivity of 95.5% and specificity of 71.2% for stroke detection, with a negative predictive value of 97.2% [18]. The presence of any HINTS sign confers a 15-fold increased risk of stroke compared to normal examination [18]. However, HINTS has limitations.

Diagnostic accuracy drops significantly when performed by non-neurologists or in patients without true vestibular syndrome (constant vertigo with nystagmus) [18]. HINTS plus—adding horizontal head shaking and hearing assessment—improves detection of posterior inferior cerebellar artery strokes, which may present with isolated vertigo and falsely negative standard HINTS [18]. Recent evidence suggests that combining HINTS with truncal ataxia assessment achieves 100% sensitivity for posterior circulation stroke detection, though this requires further validation [19]. Crucially, transient vestibular syndrome (episodes lasting minutes to hours) may represent vertebrobasilar transient ischemic attacks [13].

Up to 22% of patients with posterior circulation stroke report transient neurological symptoms in the 90 days preceding their event, with isolated vertigo being the only complaint in 21% of vertebrobasilar TIAs [13]. This underlines the importance of vascular risk factor assessment and neuroimaging even in seemingly benign vestibular presentations [14].

3.2 Altered Mental Status and Acute Confusional State

Right middle cerebral artery territory infarction—particularly involving the non- dominant hemisphere-can present as acute confusional state or delirium without obvious motor signs [15].

Similarly, thalamic infarcts produce diverse behavioral abnormalities including apathy, agitation, and memory disturbance [15]. The key diagnostic clue is the sudden onset of these symptoms in a patient with vascular risk factors, though collateral history may be difficult to obtain [15]. 3.2.1 Acute Confusional State as Stroke Chameleon Acute confusional state in the elderly represents one of the most frequently missed stroke chameleons, particularly when it occurs without focal motor deficits [29]. Non- dominant hemisphere infarction, thalamic strokes, and posterior circulation events can all present with delirium, agitation, or profound apathy that is attributed to metabolic disturbance, infection, or psychiatric decompensation [29].

The absence of hemiparesis does not exclude stroke-cortical signs such as inattention, visual field defects, or apraxia may be the only objective findings. A systematic approach including thorough neurological examination, assessment of attention and visuospatial function, and urgent neuroimaging is essential [29]. The prognosis of stroke presenting as confusion is particularly poor, with higher mortality and worse functional outcomes compared to classically presenting stroke, largely due to delayed diagnosis and treatment [29]. Basilar artery occlusion (BAO) represents perhaps the most dangerous stroke chameleon, frequently presenting with syncope or altered consciousness that spontaneously improves, only to progress catastrophically to coma [13].

Unlike syncope, which causes transient loss of consciousness with rapid recovery, BAO produces ischemia of the reticular activating system resulting in progressive deterioration of alertness [13]. The distinction requires meticulous attention to the temporal evolution of symptoms and maintenance of high index of suspicion in patients with posterior circulation risk factors [13]. 3.3 Isolated Sensory or Motor Syndromes Lacunar infarcts in specific territories can produce pure syndromes that mimic peripheral pathology [15]:

· Pure motor stroke: Internal capsule or pontine infarction causing isolated hemiparesis without sensory, visual, or cortical signs [15].

· Ataxic hemiparesis: Pontine or internal capsule lesion causing ipsilateral weakness and incoordination, potentially mimicking cerebellar disease [15].

·Isolated sensory stroke: Thalamic lacune causing hemisensory loss without motor deficits [15].

· Foot drop: Cerebral infarction in the motor cortex leg area can present as isolated foot drop, mimicking peroneal nerve palsy [15]. These presentations highlight the importance of comprehensive neurological examination and neuroimaging even when symptoms appear to localize to peripheral structures [15].

3.4 DWI-Negative Stroke

A particularly challenging radiological chameleon is the diffusion-weighted imaging (DWI)-negative acute ischemic stroke [16]. Despite DWI's 92% sensitivity for acute ischemia, approximately 7% of strokes present with negative scans—particularly small lacunar infarcts in brainstem territories and posterior circulation events [16]. The mechanism involves electric failure without ionic pump failure—reduced blood flow sufficient to cause neurological symptoms but insufficient to produce DWI-visible cytotoxic edema [16]. Patients with DWI-negative stroke typically have mild deficits (NIHSS<5)and posterior circulation involvement [16].

They are five times more likely to have negative DWI than anterior circulation strokes [16]. Importantly, approximately one-third develop DWI positivity on repeat scanning, and the risk of early recurrence is comparable to DWI-positive strokes [16]. This underscores that stroke remains fundamentally a clinical diagnosis: patients with acute focal neurological deficits consistent with vascular syndrome should be considered for reperfusion therapy regardless of initial imaging findings, provided contraindications are absent [16].

4. Practical Diagnostic Framework

4.1 The First 10 Minutes: Critical Bedside Assessment

The hyperacute phase demands rapid, structured assessment:

1. Glucose check: Immediate capillary blood glucose to exclude hypoglycemia [11].

2. Vital signs and rhythm: Blood pressure, heart rate, oxygen saturation, and cardiac rhythm assessment to identify atrial fibrillation or malignant hypertension [1].

3. Stroke severity: NIHSS scoring to quantify deficits and identify posterior circulation features (dysarthria, dysphagia, ataxia, gaze palsy) not captured by FAST [1]. 4. Targeted history: Witnessed seizure activity? Gradual vs. sudden onset? Prior similar episodes? Migraine history? Psychiatric background? Antecedent infection? [2,8, 26]

5. HINTS examination: In patients with AVS, before imaging if neurologist available [17].

4.2 Neuroimaging Strategy

Non-contrast CT remains the initial imaging modality of choice, primarily to exclude hemorrhage [1]. However, CT sensitivity for acute ischemia—particularly posterior fossa-is limited [1]. CT angiography should be performed when large vessel occlusion is suspected, and CT perfusion can identify salvageable tissue in uncertain cases [1]. MRI with DWI is more sensitive for acute ischemia but may be falsely negative in hyperacute posterior circulation stroke [16].

If clinical suspicion remains high despite negative DWI, repeat imaging at 24-48 hours is warranted [16]. Perfusion imaging may theoretically detect electric failure states, though small lesion size limits utility [16].

4.3 The Thrombolysis Dilemma

When diagnostic uncertainty persists within the therapeutic window, the decision to thrombolyse requires careful risk-benefit analysis:

· Benefit of treating true stroke: Substantial, time-dependent reduction in disability and mortality [7].

·Risk of treating mimics: Low. Pooled data demonstrate that stroke mimics treated with intravenous thrombolysis have excellent outcomes with minimal hemorrhagic complications [7].

· Risk of NOT treating true stroke: Catastrophic, with progressive infarction and permanent disability [7]. The evidence supports a treat when uncertain approach, provided absolute contraindications (active bleeding, recent surgery, known structural lesion) are absent [7]. Advanced neuroimaging (CT perfusion, CT angiography) and clinical scores can aid decision-making but should not delay treatment when suspicion is reasonable [7].

5. Conclusion

Stroke mimics and chameleons represent two sides of the same diagnostic coin-both demand clinical vigilance, structured assessment, and informed decision-making under time pressure [1]. Functional neurological disorders, seizures,migraine, hypoglycemia, and Guillain-Barré syndrome variants are the most common mimics, each with distinctive diagnostic features that can be elicited through careful history and targeted examination [2, 6, 8, 11, 26]. Conversely, posterior circulation stroke, altered mental status and acute confusional state from right hemisphere or thalamic infarction, and DWI-negative stroke are the most dangerous chameleons,requiring high index of suspicion and systematic evaluation [13, 15, 16,29]. The HINTS examination has transformed assessment of dizziness, offering high sensitivity for posterior circulation stroke when performed by trained clinicians [17, 18].

The principle of time is brain remains paramount: when diagnostic uncertainty exists, the low risk of thrombolysing a mimic must be weighed against the devastating consequences of missing a treatable stroke [7]. As our understanding of these conditions evolves, so too must our clinical algorithms—integrating advanced imaging, biomarkers, and artificial intelligence to enhance diagnostic precision while preserving the urgency that acute stroke care demands [1]. References

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