

A Narrative Review on Stroke Mimics and Chameleons: Revisiting and Unveiling the Disguise

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ABSTRACT

Stroke refers to a neurological deficit that has an acute onset and a vascular origin. The classical signs and symptoms of a stroke include facial drooping, weakness, and disturbances in talking or speech. Sometimes, these symptoms may also arise from conditions that do not involve a cerebrovascular event, leading to a misdiagnosis of stroke, known as “stroke mimics.” Another interesting category is the “stroke chameleon,” which refers to a class of stroke that occurs in the absence of the typical characteristics suggestive of stroke and instead presents with atypical features that can lead to a false negative diagnosis. A thorough history taking, with an emphasis on the onset of symptoms, is crucial because features of stroke are always acute in onset. Prompt neuroimaging is ultimately necessary to prevent misdiagnosis and to provide emergent thrombolysis within the life-saving window period. In this review, we discuss the various possible stroke mimics and chameleons that have been reported and are available across the web.

Keywords: Altered mental status, Functional disorders, Hypoglycaemia, Migraine, Multiple sclerosis, Seizures, Thrombolysis, Vertigo

INTRODUCTION

Stroke is a medical condition that requires urgent attention and can be defined as a neurological deficit of sudden onset caused by ischaemia or hemorrhage. However, most strokes tend to be ischaemic. The most common clinical manifestations of a stroke include hemiplegia (unilateral weakness of limbs), speech difficulties, facial drooping on one side, severe headache of sudden onset, and confusion. For the identification of these symptoms by first point contacts of patients, such as ambulance staff or professionals in the emergency department, scales like the Face Arm Speech Test (FAST) [1] and Recognition of Stroke in the Emergency Room (ROSIER) [2] have been developed.

Even though these tools assist in the quick identification of stroke, they are not flawless and can lead to false positive or false negative diagnoses [3]. A false positive occurs when clinical presentations similar to those of a stroke are witnessed in other illnesses, such as migraine, seizures, metabolic abnormalities like hypoglycemia, space-occupying lesions, and functional disorders, which do not involve any ischaemic or hemorrhagic event in the brain. These “false positives” are known as “stroke mimics” and account for about 25 percent of cases in emergency departments worldwide [3].

Interestingly, a stroke may also present symptoms that are clinically different from what is expected and cannot be attributed to a single lesion, such as acute psychosis, altered mental status, dizziness, and vertigo. This leads to a diagnosis of false negatives, as these myriad manifestations reflect another condition, referred to as “stroke chameleons.” Due to these mimics and chameleons, stroke is the fourth most prevalent form of misdiagnosed illness [4,5]. This review article aims to provide a detailed account of all the stroke mimics and chameleons typically encountered in the emergency department.

STROKE MIMICS

These false positive strokes are differentiated from actual strokes with the help of radiological imaging, such as Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) scans of the brain [6]. Despite the risks associated with thrombolysis, these risks remain mild when compared to the repercussions one can face if thrombolysis is not provided in a timely manner to a stroke patient.

Therefore, thrombolytic therapy is administered to a suspected stroke patient even if it later turns out to be a stroke mimic [7].

Types of Stroke Mimics

Migraine: Hemiplegic migraine, a type of migraine with aura that also causes weakness in limb movements, is possibly the primary headache disorder most commonly confused with a stroke. It is quite uncommon, with a prevalence estimated at 0.01% [8]. In familial hemiplegic migraine, which typically has a clear family history due to its autosomal dominant inheritance, the initial episodes generally begin before the age of 20. Motor symptoms in people with hemiplegic migraine usually last for three days, but they can also persist for weeks. Most individuals experience the four common types of aura (visual, sensory, aphasic, and motor), and all patients show at least two types during an attack [9]. Thus, a diagnosis of familial hemiplegic migraine cannot be made based solely on motor symptoms. Another significant stroke mimic is a condition known as non-familial Migraine with Unilateral Motor Symptoms (MUMS) [10].

Headache with Neurological Deficits (HaNDL): HaNDL syndrome, which includes headaches, neurological deficits, and Cerebrospinal Fluid (CSF) lymphocytosis, is a rare and non malignant condition that is often underdiagnosed [11]. It is characterised by migraine-like headaches, aphasia, and sensory and motor deficits. Patients experience one or more episodes but are generally symptom-free between episodes. Neurological deficits can persist for up to seven weeks, but recovery is possible. In contrast, a stroke typically presents with sudden-onset, persistent neurological deficits without recurring episodes [3]. The initial diagnostic task is to differentiate HaNDL from Transient Ischaemic Attacks (TIAs), seizures, and meningoencephalitis, which necessitates detailed neuroradiologic and CSF studies in the acute phase [11,12].

Seizures: Postictal Todd's paresis, which accounts for most stroke mimics, can be difficult to distinguish from a stroke. It refers to a temporary motor dysfunction that occurs after partial seizures [13]. Postictal paresis has been attributed to fatigue and depletion of neurons following the great demand for metabolism at the focus of an epileptic seizure and/or proactive suppression near the point of origin [14]. Although distinguishing between a stroke and a seizure is usually simple, in the absence of a thorough medical

history, post-seizure neurological impairments may be misdiagnosed as a stroke. Early diffusion abnormalities in ischaemic stroke are visible on MRI, whereas seizures have less distinct MRI patterns that can change depending on when the examination is conducted. Consequently, the only accepted method for diagnosing a stroke is a detailed medical history and examination backed by neuroimaging, which may show no significant results [15].

Space-occupying lesion: Tumours usually cause gradually progressive deficits, but about 5% can present with stroke-like symptoms. Acute impairments from intracranial mass lesions can result from extrinsic compression of blood vessels due to edema, obstructive hydrocephalus, or Todd's paresis, with bleeding into the lesion being the most common cause of symptoms [15]. Typically, diagnosing intracranial mass lesions presents no challenges; however, low-grade gliomas may occasionally manifest with focal seizures and subsequent postictal Todd's paralysis. Large arterial strokes typically take 24 to 48 hours to cause cerebral edema, hence the occurrence of an extremely early mass effect points to a tumour [16]. Lesions observed on CT and MRI typically do not follow vascular distribution and may be accompanied by surrounding edema. Treatment varies based on the underlying condition, with seizures potentially requiring anticonvulsants [17]. An acute neurological deficit can reveal a tumour, often malignant, due to postictal deficits, intratumoral hemorrhage, or extrinsic vessel compression. Diagnosis is generally straightforward with MRI.

Multiple Sclerosis (MS): Acute stroke presentations can sometimes be mistaken for an acute episode of MS. Factors such as the patient's young age, medical history, and the presence of multiple abnormalities on neurological examination can help differentiate between the two. In some patients, the initial symptoms can resemble an acute pseudo-stroke, with sudden hemiparesis, an inability to speak and hear, closely resembling a stroke [18]. Diagnosing the first attack of MS can be particularly challenging. While a CT scan may appear normal, MS plaques typically present as well-demarcated, homogeneous small ovoid lesions on MRI. Atypical features of MS lesions include sizes larger than 2 cm, mass effect, and edema. Tumefactive MS lesions can mimic intracranial neoplasms, infarction, and infections on radiographs. In such atypical cases, a brain biopsy may be necessary for an accurate diagnosis. It's crucial to remember that, in a patient with MS, the incident might be an acute stroke. The risk of stroke increases by 28% in individuals with MS [19]. In tumefactive MS, several types of enhancement have been observed, such as open-ring and multiple closed-ring enhancing lesions. The absence of enhancement does not exclude an inflammatory demyelinating lesion of the central nervous system. Lack of significant enhancement is another factor to consider when diagnosing a challenging case of tumefactive MS [20].

Hypoglycaemia: Symptoms of hypoglycemia range from mild confusion and dizziness to severe outcomes like coma, seizures, and hemiparesis. It can mimic a stroke, leading to misdiagnosis and delayed treatment. Two key pitfalls contribute to this: normal blood glucose levels due to food absorption or drug metabolism, and transient DWI changes. The 'ABC-DEFG' approach emphasises measuring blood sugar before thrombolysis [21]. A rare form, Hypoglycaemic Hemiparesis (HH), is often misidentified as a stroke, but early intravenous glucose can reverse it quickly, preventing brain damage or death, thus avoiding costly and risky investigations. Typically, hypoglycaemic brain damage presents as diffuse abnormalities, while HH shows distinct bilateral MRI findings affecting the cortex, internal capsule, hippocampus, and basal ganglia, unlike ischaemic stroke [22].

Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like episodes (MELAS): MELAS is a rare hereditary mitochondrial disorder affecting multiple organ systems. It typically presents in the first or second decade of life, although onset can occur later. Symptoms include mitochondrial myopathy, stroke-like episodes, seizures, dementia, and lactic acidosis. Other manifestations may include

ataxia, diabetes, hypoparathyroidism, cardiomyopathy, hearing loss, headaches, vomiting, peripheral neuropathy, ophthalmoplegia, and retinopathy [23]. MELAS should be considered in younger patients with stroke-like symptoms. Early diagnosis, genetic counseling, thorough evaluation, and follow-up are crucial for improving quality of life [24]. Non-ischaemic stroke-like events help distinguish MELAS despite its varied symptoms. Diagnosing MELAS is challenging due to diverse clinical and imaging presentations. "Ragged-red fibers" in muscle tissue are a hallmark finding. Imaging may reveal bilateral basal ganglia calcification, focal lesions, fourth ventricle enlargement, and generalised atrophy, often misdiagnosed as gliomas, infarctions, or viral encephalitis [25].

Hyperglycaemia: Hyperosmolar Hyperglycaemic State (HHS) represents an acute complication associated with Type 2 Diabetes Mellitus, characterised by significant hyperosmolarity, elevated blood glucose levels, and profound dehydration, occurring in the absence of ketosis. There is a documented association between hyperglycemia in HHS and instances of focal seizures, as well as focal neurological deficits that may mimic cerebral infarction. Common neurological manifestations include aphasia, homonymous hemianopia, sensory impairments on one side of the body, hemiparesis, unilateral hyperreflexia, and a positive Babinski sign. Importantly, the lesions resulting from HHS are often reversible and exhibit widespread distribution across both cortical and subcortical regions, aiding in differentiation from infarct-related conditions [26].

Functional disorders: Functional disorders can present as acute motor or sensory deficits that mimic stroke, often triggered by emotional events like panic attacks or dissociative episodes. Diagnosing functional disorders relies on identifying positive features rather than just ruling out organic diseases [4]. These are the stroke mimics that receive thrombolysis most frequently. Patients often present with a history of mental health issues. Repeated testing during physical examinations yields inconsistent results, while imaging is usually normal or shows no Diffusion-weighting Imaging (DWI)-MRI abnormalities. A psychiatry appointment is advised, and the patient is reassured that no structural lesion accounts for their symptoms [27].

Contrast-Induced Encephalopathy (CIE): CIE is a relatively rare condition caused by the administration of iodine contrast in cerebral or carotid angiography procedures. It can also occur post-endovascular aneurysm management. Hypertension and chronic kidney disease are two important risk factors for CIE, being present in 67% and 24% of patients, respectively [28]. The symptoms of CIE usually include cortical blindness, hemiparesis, speech disturbances, lack of coordination, and seizures, which largely mimic an embolic or hemorrhagic event, specifically an anterior circulation stroke [29]. Embolism or hemorrhage after an angiography procedure is also a potential complication; hence, quick neuroimaging to distinguish CIE from a stroke is absolutely necessary. A case report published in 2020 stated that the symptoms induced by iodine contrast in an elderly woman were found to be global aphasia and hemiplegia at the end of the angiography procedure. Typically, the symptoms of CIE begin minutes to hours after the procedure and can last for about 2-3 days. A CT scan generally reveals no ischaemic or hemorrhagic event and might show some degree of cerebral edema [28].

Sepsis: Sepsis can mimic stroke symptoms, especially in cases like urosepsis, which accounts for about 12% of stroke mimics [30]. Sepsis-associated encephalopathy may cause disorientation, restlessness, or drowsiness, resembling a stroke. A detailed history of previous strokes, neurological impairments, medications, and sepsis symptoms is crucial. Unlike the sudden onset of stroke, sepsis symptoms develop gradually. An altered mental status should prompt consideration of other Central Nervous System (CNS) conditions, such as meningitis, encephalitis, or a brain abscess [17].

Coronavirus Disease-2019 (COVID-19): During the global COVID-19 pandemic, infection with Severe Acute Respiratory Syndrome

Coronavirus-2 (SARS-CoV-2) has been widely documented to cause a variety of respiratory and gastrointestinal symptoms. Notably, COVID-19 patients have shown an increased incidence and severity of acute ischaemic stroke, along with instances of stroke-like symptoms. Among these, language impairments have been observed as early indicators in some cases. Reported symptoms include expressive and receptive aphasia, reduced motor and sensory function, temporary confusion, and reversible encephalopathy, appearing both before and after more typical COVID-19 symptoms. Distinguishing between actual strokes and stroke-like symptoms in COVID-19 patients is particularly challenging. Indicators that may suggest a stroke mimic include a lack of typical stroke risk factors, the onset of new seizures, acute or subacute encephalopathy, and a reduced Glasgow Coma Scale (GCS) score [31,32].

STROKE CHAMELEONS

The latest average rate of occurrence of stroke chameleons has been reported as 1.2-12.7% in an article published in 2023 [33]. Such stroke chameleons are more often seen in the younger population and are of greater importance than mimics due to the associated risk of delay in accurate treatment, resulting in higher rates of morbidity and mortality [34]. According to a review article published in 2011, any neurological symptom could indicate a stroke, but the diagnosis becomes contested if the type of deficit is unusual and its timing is ambiguous [35].

The time of onset of stroke symptoms tends to be the most relevant factor in history-taking. Acute and sudden onset of symptoms lends credibility to a stroke diagnosis. For instance, confusion and delirium, which are part of the umbrella term 'stroke chameleons,' can be indicative of a right Middle Cerebral Artery (MCA) stroke if the history suggests sudden onset [36]. Another case in which a stroke could be missed due to a chameleon is thalamic infarctions, which often present with features akin to schizophrenia. Again, the key distinguishing factor to look for is the acuity of onset [37].

Types of Stroke Chameleons

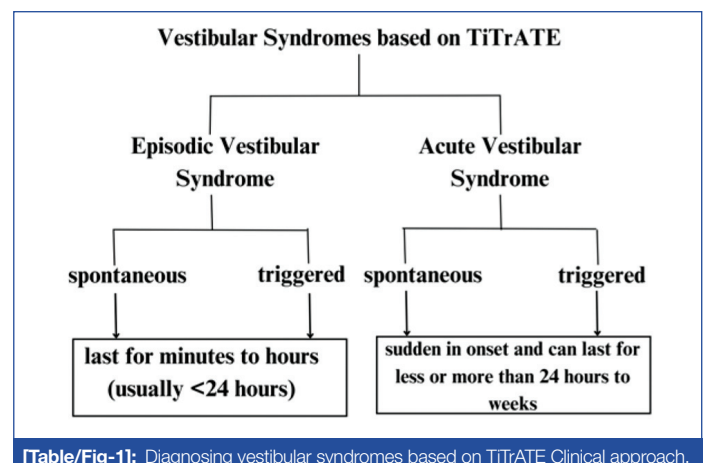
Dizziness and Vertigo: Dizziness is a very common stroke chameleon that is often misdiagnosed when there are no other neurological deficits present. Approximately 47-75% of posterior circulation stroke cases present with dizziness [38]. In the US, about 0.18-0.70% of approximately 1.5 million patients diagnosed with benign peripheral vestibular disorders in emergency departments required hospitalisation for stroke within the next 30 days, with the risk being 50-fold greater within the first seven days after discharge, according to a recent study [39,40].

Dizziness and vertigo are usually mistaken for peripheral vestibular syndrome when they occur in the absence of any other neurological deficit. A detailed examination is essential for diagnosing vestibular syndromes based on a clinical approach called Timing, Trigger, and Targeted Examination (TiTrATE). This concept describes four vestibular syndromes: spontaneous Episodic Vestibular Syndrome (sEVS), triggered EVS (tEVS), spontaneous Acute Vestibular Syndrome (sAVS), and triggered AVS (tAVS) [40]. This is depicted below in [Table/Fig-1].

The HINTS test (head impulse test, nystagmus, and the test for skew deviation), developed by Kattah and colleagues, is useful for diagnosing the central cause of Acute Vestibular Syndrome (AVS) upon first contact. For a confirmatory diagnosis, neuroimaging is indicated [39].

Neuropsychiatric manifestations:

a) Altered mental status: Altered mental status is the second most recognised abnormal presentation of a stroke, accounting for about 20% of errors in stroke diagnosis. These cases are generally misinterpreted as symptoms of metabolic disturbances, CNS or systemic infections, or toxic or hypertensive encephalopathy [3]. Types of strokes that can lead to altered consciousness include:



[Table/Fig-1]: Diagnosing vestibular syndromes based on TiTrATE Clinical approach.

- Occlusion of the distal end of the basilar artery (most common), referred to as the top of the basilar syndrome or rostral brainstem infarction [41].
 - Hemorrhagic stroke associated with a sudden rise in intracranial pressure.
 - Occlusion of the Percheron artery, a variant of the perforating arteries of the thalamus [42].
- b) Delirium:** Delirium can be a presenting symptom in several types of strokes, either alongside other neurological deficits or in the absence of them. It is more commonly associated with hemorrhagic stroke than ischaemic stroke. The brain sites involved in strokes that can manifest as delirium include the right inferior parietal lobe, frontal and occipital lobes, right temporal gyrus, corpus callosum, paramedian nuclei of the thalamus due to vertebrobasilar ischaemia, and the limbic cortex [43].

Hyperkinetic Movement Disorders (HMD): Movement disorders are a rare manifestation of stroke, either as a chief symptom or as a delayed sequela, accounting for 1-4% of stroke cases. Various movement disorders that can occur as a result of stroke include hemichorea-hemiballism, hemidystonia, stereotypies, jerky dystonic unsteady hand, asterixis, bilateral tremor, bilateral jaw myoclonus, hemiakathisia, and dysarthria-dyskinetic hand [44]. An unusual form of HMD reported by some patients is twitching or trembling in one or more limbs triggered by a change in position or exercise, referred to as 'limb shaking TIA.' This is generally associated with a significant degree of occlusion of the contralateral internal carotid artery and requires urgent attention [44]. The locations of infarcts in the brain most commonly associated with HMD are the thalamus, subthalamic nucleus, striatum, and lentiform nucleus of the basal ganglia, as well as the territory of the posterior choroidal artery, which can cause jerky hand dystonic syndrome. Midbrain and pontine strokes may show segmental myoclonus. Nonetheless, irregular movement patterns in stroke patients do not indicate a specific inclination toward a particular vascular region, stroke location, or stroke type [35].

Isolated monoplegia and cortical hand syndrome: Isolated weakness or paralysis of the face, arms, or legs can be mistaken for a peripheral nervous system disorder; however, these symptoms can be a stroke masquerading in less than 1% of cases. Most of these instances are caused by subcortical or cortical lesions in the brain.

The infarction of the MCA territory often leads to paralysis of the contralateral arm, while a subcortical infarct can cause contralateral facial palsy [45]. A special omega or epsilon-shaped area that controls motor functions of the hand, referred to as the 'hand knob area,' is located in the precentral gyrus and is supplied by the rolandic artery, a branch of the MCA. This area has been associated with 'cortical hand syndrome.' If the medial portions of this area are involved, it causes symptoms of ulnar nerve weakness; if the lateral regions are involved, it leads to symptoms associated with radial

weakness. As a result, it can easily be confused with radial or ulnar mononeuropathy [33,46].

Acute Coronary Syndrome (ACS): ACS is one of the most confusing stroke chameleons reported in emergency settings. In the November 2020 issue of the European Journal of Neurology, five cases of stroke presenting as ACS were reported. These patients came in with complaints of acute chest or epigastric pain, which clearly diverted the physicians' attention away from suspecting a stroke. Two out of five patients had minor changes in their ECGs, and only one had elevated troponin levels, which were later found to be of non-cardiac origin [47]. A diagnosis of ACS was made as soon as the patient arrived, as ACS is also a critical condition requiring prompt treatment. A series of cardiac evaluations, including ECG, cardiac biomarkers, and echocardiography, helped rule out a cardiac origin for the symptoms. Furthermore, another factor that suggests a non-cardiac origin of the chest pain is the lack of response to sublingual nitroglycerin or beta-blockers [48,49].

Hence, when encountering cases akin to myocardial infarction with negative biomarkers and ECG results, along with no response to medication, a non-contrast CT scan of the brain could be the next essential step to identify a likely stroke diagnosis. Most often, the region that appears to be involved is the corona radiata.

CNS lymphoma: A rare case was reported in 2023 by Mallio CA et al., describing an elderly man who had a stroke in the artery of Huebner but was incorrectly diagnosed with CNS lymphoma [50]. Symptoms associated with the stroke of the artery of Huebner include face and arm weakness on the opposite side, abulia, akinetic mutism, and neglect. This artery supplies the anterior portion of the caudate nucleus, the internal capsule, and the anterior perforated substance.

Confirmation of an ischaemic stroke could be obtained after performing an MRI two months later, which revealed gliosis and a malacic area, thus confirming a history of ischaemic stroke and dismissing CNS lymphoma [33,50].

Atypical stroke syndromes: Charles Bonnet Syndrome (CBS): This syndrome involves visual hallucinations (such as gardens, animals, or patterns) following a loss of input to the occipital or temporal lobes. Patients recognise the hallucinations as unreal, differentiating them from psychiatric conditions [51].

Anton's Syndrome: This condition results in blindness due to bilateral occipital lobe infarcts, accompanied by denial of vision loss (visual anosognosia). Patients may confabulate and it can coexist with CBS if hallucinations accompany the blindness [33].

Gerstmann Syndrome: An infarct in the dominant parietal lobe can cause agraphia (inability to write), acalculia (difficulty with math), finger agnosia (failure to recognise fingers), and confusion of right-left orientation. A thorough evaluation is required to avoid misdiagnosis [52].

Alien Limb Syndrome: This syndrome involves involuntary, purposeful limb movements coupled with the belief that the limb is not one's own. It occurs more often in the upper limbs, and vascular causes account for approximately 10% of cases [53].

Parinaud syndrome/Dorsal midbrain syndrome/Sylvian aqueduct syndrome: Parinaud syndrome comprises supranuclear vertical gaze palsy, retracted eyelids (known as Collier's sign), diplopia, blurred vision, nystagmus when attempting to look upwards, and impaired pupillary light reflex. The main areas of the brain involved are the dorsal midbrain, pretectal region, or the tegmentum of one side. About 65% of cases of Parinaud syndrome arise from primary midbrain strokes and hemorrhages [54].

Strategies to Improve Accuracy in Differentiating between Stroke Mimics and Chameleons

Distinguishing chameleons from stroke mimics remains a significant challenge. To enhance diagnostic accuracy, medical professionals can adopt several strategies. Artificial Intelligence (AI) can improve

diagnostics by identifying subtle abnormalities in neuroimaging that might escape the naked eye, while predictive modeling can evaluate medical records to estimate stroke probabilities based on patient data patterns. Point-of-care biomarker testing, such as rapid blood tests for inflammation or neuronal injury biomarkers, along with CSF analysis for specific markers, could quickly differentiate strokes from other conditions.

Remote consultation and telemedicine have become essential; virtual consultations with neurologists can expedite diagnoses and treatment decisions, particularly in underserved areas. Mobile stroke units, equipped with portable CT scanners and communication tools, enable early evaluation and treatment before hospital arrival, reducing the incidence of stroke mimics at stroke centers and ensuring timely diagnoses of stroke chameleons. Advanced neuroimaging techniques like functional MRI (fMRI) and Positron Emission Tomography (PET) scans help assess brain activity, cerebral perfusion, and metabolism, offering valuable insights into unusual stroke cases.

Regular simulation training and professional development for emergency and stroke teams can enhance their ability to recognise and manage atypical presentations. Interdisciplinary case reviews can foster collaboration and improve diagnostic approaches. Finally, public awareness campaigns involving local groups can educate communities about stroke symptoms, including atypical ones, to reduce delays in seeking care. By adopting these strategies, healthcare professionals can achieve timely and accurate diagnoses, leading to better patient outcomes [55-57].

CONCLUSION(S)

It is imperative to recognise the various mimics and chameleons of a stroke to facilitate both prompt and accurate diagnosis. A misdiagnosed stroke in the presence of a mimic may result in unnecessary costs associated with the administration of thrombolytics to the patient, as well as significant delays in the diagnosis and treatment of the actual underlying condition. Likewise, the failure to diagnose a stroke due to an atypical presentation, as observed with stroke chameleons, can lead to postponed treatment, which is potentially life-threatening and may result in considerable morbidity and mortality. Therefore, this review seeks to enhance the understanding of clinicians, particularly emergency physicians who serve as the initial point of contact for stroke patients, thereby enabling them to be more vigilant and capable of making accurate and timely assessments and diagnoses.

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