European Journal of Radiology Open 11 (2023) 100533

Contents lists available at ScienceDirect

European Journal of Radiology Open

journal homepage: www.elsevier.com/locate/ejro

Stroke chameleons: Diagnostic challenges

P Candelaresi [a,*], C Di Monaco [a,b], E Pisano [a,b]

[a] Neurology and Stroke Unit, AORN Antonio Cardarelli, Naples, Italy
[b] Department of Neuroscience, Reproductive Science and Odontostomatology, Federico II University, Naples, Italy

1. Introduction

Stroke is a leading cause of death and disability worldwide [1]. An accurate and timely diagnosis is critical to benefit from acute reperfusion treatments and adequate secondary prevention strategies, improving patients' outcome and reducing the risk of recurrency. However, stroke diagnosis is sometimes tricky. According to a recent meta-analysis, approximately 9 % of cerebrovascular events, including ischemic stroke, subarachnoid haemorrhage, and transient ischemic attack, is missed at initial presentation in the emergency department [2]. Stroke misdiagnosis is a meaningful public health problem, resulting in higher rates of morbidity and mortality. Diagnostic errors may consist in stroke overdiagnosis in cases of acute focal neurological signs/symptoms due to different diseases resembling stroke (false positives, the so-called "stroke mimics"), or in stroke underdiagnosis due to atypical stroke presentations (false negatives, the so-called "*stroke chameleons*") [3,4]. The overdiagnosis of stroke may result in unnecessary treatments and increased health care costs, whereas delayed or missed diagnosis may cause failure of timely treatments and accurate secondary prevention, predisposing to worse clinical outcome. Absence or paucity of vascular risk factors, younger age, or subtle clinical characteristics at presentation (such as absence of eye deviation) and posterior circulation localization were showed as independent risk factors of ischemic stroke chameleons. Stroke chameleons rate ranged from 1.2 % to 12.7 %, according to a large series.

2. Clinical stroke chameleons

2.1. Dizziness/acute vestibular syndrome

Dizziness is a common stroke chameleon and a diagnostic challenge for physicians, as possible underlying causes span from benign conditions as vestibular neuritis to life-threatening diseases such as posterior circulation stroke [5]. Large prospective studies reported dizziness as a presenting symptom in 47–75 % of patients with posterior circulation stroke. The risk of misdiagnosis increases when dizziness presents as an isolated and/or a transient symptom in the absence of other focal neurological signs. In a recent meta-analysis, including more than 15,000 acute stroke patients, the rate of stroke misdiagnoses was 9 %, and isolated dizziness accounted for approximately 15 % of them [2]. The clinical and neurological examination plays a crucial role in the diagnosis of vascular vestibular syndrome in the emergency department [6]. Current evidence recommends a thorough assessment of acute vestibular disorders based on clinical algorithms, such as the *TiTrATE* (timing, trigger, and targeted examination) [5]. *TiTraATE* approach describes four main vestibular syndromes according to dizziness timing and triggers: spontaneous or triggered *episodic vestibular syndromes* (s-EVS and t-EVS), and spontaneous or triggered *acute vestibular syndromes* (s-AVS and t-AVS). Vascular dizziness occurs more frequently as an acute vestibular syndrome (AVS), characterized by acute onset vertigo or dizziness with nausea or vomiting, head-motion intolerance, and unsteadiness. AVS may be transient (minutes to < 24 h) or prolonged (≥ 24 h) and may present either as isolated or associated with other focal neurological symptoms. It may be caused by either ischemic or haemorrhagic injury. Cerebellar or brainstem ischemic strokes account for up to 10 % of AVS. The HINTS testing (head impulse test (HIT), gaze and Nystagmus testing, and alternate cover Test for detection of Skew deviation) has been shown to accurately identify central causes of dizziness in patients with AVS [7,8]. The presence of direction-changing or vertical nystagmus and skew deviation, or a negative HIT suggests central causes of vertigo, with a sensitivity of 97 % and specificity of 95 %, when performed by neurologists in cases of AVS. However, the diagnostic accuracy significantly drops down if performed by a non-neurologist or in patients who do not have vestibular syndrome (constant vertigo with nystagmus) [9]. Conversely, diagnostic accuracy increases with HINTS plus examination, adding horizontal head shaking and hearing test with finger rub [8], particularly for posterior-inferior cerebellar artery strokes, in which patients may present isolated vertigo with negative HINTS mimicking acute peripheral vestibular syndromes [9]. In acute transient vestibular syndrome (ATVS), the

* Correspondence: Neurology and Stroke Unit, AORN Antonio Cardarelli, via Cardarelli 9, 80131 Naples, Italy
E-mail address: paolo.candelaresi@aocardarelli.it (P. Candelaresi).

https://doi.org/10.1016/j.ejro.2023.100533
Received 1 June 2023; Accepted 20 October 2023

diagnostic utility of both HINTS/HINTS plus examination and Magnetic Resonance Imaging (MRI) is limited. Though isolated transient vertigo is not considered a symptom of transient ischemic attack (TIA), ATVS may frequently occur in vertebrobasilar TIAs. Transient vestibular syndrome, even when isolated, is the most common clinical presentation of vertebrobasilar TIAs in the days to weeks preceding posterior circulation stroke [10]. In a study of patients with posterior circulation stroke, 22 % reported transient neurological symptoms in the 90 days preceding their stroke, most frequently vertigo. Indeed, isolated vertigo was the only complain in 21 % of patients diagnosed with vertebrobasilar TIAs [11]. A recent study reported the prevalence of stroke is 27 % in ATVS [11,12] The combination of clinical examination and assessment of cerebrovascular risk factors may be useful to detect vascular vestibular syndrome.

2.2. Altered mental status

Altered mental status is a challenging clinical condition for both the emergency and stroke physicians. In a stroke unit–equipped university hospital, the "decreased level of consciousness/coma" was the second most frequent pattern leading to misdiagnosis of stroke, accounting for about 20 % of cases of erroneous diagnoses [14]. Moreover, acute ischemic stroke presenting as pure cognitive alterations, such as confusional state, may be attributed to metabolic disturbances, hypertensive encephalopathy, or systemic infections. Conversely, in younger patients, altered mental status is often attributed to psychogenic disease. Besides mild strokes, very severe strokes may also be missed, above all those patients in stupor or coma: this presentation may mislead physicians to suspect a metabolic, toxic, or anoxic encephalopathy rather than stroke, mostly due to basilar artery occlusion. Particularly challenging is the Percheron artery syndrome. The artery of Percheron is an anatomical variant of the thalamoperforating arteries. Its occlusion can lead to bilateral paramedian thalamic and rostral midbrain infarct presenting as memory loss, fluctuating levels of consciousness, and altered mental status [15].

2.3. Headache

The relationship between stroke and headache is manifold since migraine with aura is a well-established stroke risk factor, aura symptoms can mimic vascular neurological deficits, and migraine may cause cerebral infarction (the migrainous infarction). Furthermore, any cerebrovascular event (e.g., ischemic stroke, intracerebral haemorrhage, venous sinus thrombosis, or cervical artery dissection) may present with isolated headache and may potentially be misdiagnosed as migraine or other primary headache disorders. The clinical manifestations of headache have low sensitivity and specificity for acute cerebrovascular diseases, except for the "thunderclap" onset of headache suggesting subarachnoid haemorrhage. In the emergency setting, headache is among the most common symptoms, accounting for approximately 26 % of cases of cerebrovascular disease misdiagnosis [2]. About 15 % of ischemic stroke patients present with headache at onset [17]. However, nearly 2 % of all patients evaluated for stroke are eventually diagnosed as migraine [18]. In a study based on administrative claims, about 0,5 % of the included 2,101,081 patients discharged from the emergency department with a non-specific headache diagnosis presented with a serious neurological condition within 30 days, including ischemic stroke [19]. According to the International Classification of Headache Disorders-3, Headache attributed to ischemic stroke (HAIS) develops in very close temporal relation to other symptoms and/or clinical signs of ischemic stroke and significantly improves in parallel with stabilization or improvement of other symptoms or clinical or radiological signs of stroke [20]. Alternative diagnostic criteria include a new type of headache or a previous type of headache with altered characteristic, developed within 24 h of other symptoms and/or signs of ischemic stroke. HAIS is typically associated with vertebrobasilar strokes, particularly those involving the cerebellum, and it is common with cortical and larger size lesions (>15 mm). Moreover, it has been more frequently reported after cardioembolic events and large vessel occlusions. Despite most patients experience non-specific pain, resembling tension-type headache, new onset migraine-like headache is more frequent at onset of posterior circulation stroke. In conclusion, a new-onset headache or headache with altered characteristics needs a thorough evaluation, carefully looking for associated subtle symptoms or clinical signs to avoid erroneous false-negative stroke diagnosis.

2.4. Seizures

Stroke is a leading cause of epilepsy, accounting for 30–50 % of all-cause epilepsy in elderly. Seizures following stroke are usually dichotomized into acute and remote. Acute symptomatic seizures occur within 7 days after a stroke, and are likely provoked by the acute inflammatory and metabolic changes following stroke, inducing excitotoxicity. Remote symptomatic seizures are unprovoked seizures occurring more than 7 days after stroke. If the risk of remote seizures is about 30 %, the incidence of acute symptomatic seizures is generally low, and it is higher in intracranial hemorrhage (10–16 %) than in ischemic stroke (2–4 %). The incidence of seizures in the first 24 h or as the presenting symptom of ischemic stroke is even lower, accounting for less than 1 % [21] However, younger age is a recognized risk factor for seizures at presentation: about one-fifth of stroke in pediatric population present with seizures. Importantly, seizures are always accompanied by focal neurologic deficits [22]. Seizures at onset have historically been considered as a contraindication to reperfusion treatments. Indeed, distinguishing a postictal state from stroke may be particularly challenging when confusion or aphasia is present, and perfusion imaging may not be unequivocal if not associated with vessel occlusion. However, seizures are one of the most common mimics of stroke in which thrombolysis has been showed to be safe [23]. Therefore, nowadays, according to guidelines, it is reasonable to consider reperfusion treatments if evidence suggests that residual impairments are secondary to stroke and not a postictal phenomenon [24].

2.5. Isolated monoparesis

Isolated monoparesis may be an uncommon stroke presentation, occurring in less than 1 % of all strokes, and may be misdiagnosed as peripheral neuropathy [25]. It is usually associated to contralateral motor cortical lesions. "Cortical hand" is the most typical clinical subtype, due to lesions involving the hand motor area referred as the "hand knob", an omega- or epsilon-shaped region of the precentral gyrus, supplied by the Rolandic artery, a branch of the M4 segment of the middle cerebral artery [26]. In this cortical area it is possible to distinguish a specific somatotopic distribution in which medial lesions result in ulnar predominant hand weakness whereas lateral lesions result mainly in radial hand weakness. Despite hand knob stroke can mimic radial and/or ulnar neuropathy, a careful physical examination may help differentiate the central or peripheral nervous system involvement, as the pattern of weakness distribution does not match a specific peripheral nerve distribution and nerve conduction studies are normal. Moreover, the presence of multiple cardiovascular risk factors should orientate the diagnostic suspicion towards cerebrovascular disease. In case of ischemic stroke, a thorough evaluation of embolic sources is mandatory. Similarly, "cortical foot", referred as pure foot drop, occurs as a rare stroke presentation and may be confused with by peroneal nerve injury [27,28]. Unlike cortical hand, cortical foot is less generally recognised as clinical entity. It has been reported more frequently associated with high cortical and small infarcts in the anterior cerebral artery territory [16].

2.6. Hyperkinetic movement disorders

Stroke related hyperkinetic movement disorders (HMD) are rare clinical presentations observed in 1–4 % of all strokes [29]. HMD can occur at stroke onset as main and transient symptoms or manifest as delayed permanent sequelae [30]. As HMD are very rare at stroke onset and not included in routine clinical assessment (i.e., National Institute of Health Stroke Scale [NIHSS]), they are frequently overlooked and undertreated. Small vessel disease causing deep infarcts involving basal ganglia, the subthalamic nucleus and thalamus is the most common subtype of stroke leading to HMD [31]. Among HMD, hemichorea-hemiballismus syndrome, asterixis, and episodic paroxysmal dyskinesia commonly occur in the acute phase, whereas dystonia, tremor, vascular parkinsonism, and vascular chorea present as permanent or chronic disorders [31]. There is no definite relation between the type of abnormal movement and the site of stroke: no specific location of lesions reliably predicts the occurrence of a particular movement disorder, and the same movement disorder can be caused by lesions in different parts of the brain [32]. For example, hemiballism, traditionally described in lesions involving the subthalamic nucleus, can also be associated with lesions in caudate nucleus, putamen, and thalamus [33]. Among HMD, limb shaking TIA is an uncommon and hemodynamic form of TIA associated to carotid disease mimicking focal motor seizures [4]. Diagnostic suspicion of limb-shaking TIA should arise in case of recurrent, stereotyped, arrhythmic, and involuntary movements in one or more limbs, described by patients as shaking, trembling, twitching, or flapping, triggered by an orthostatic position change or by exercise. Limb-shaking TIAs require emergent evaluation, as frequently associated to high-grade stenosis or occlusion of the contralateral carotid internal artery leading to decreased cerebral flow and hemodynamic imbalance. Limb shaking TIAs resemble focal motor seizures, from which they differ for lack of Jacksonian march, no facial involvement and for absence of epileptic discharges on EEG [34].

2.7. Radiological stroke chameleons

A particularly challenging scenario for stroke physicians is the diffusion-weighted imaging (DWI)-negative acute ischemic stroke, a full-fledged radiological stroke chameleon.

The sensitivity of DWI for identifying ischemic stroke is 92 %, the specificity 75 %, and the positive predictive value 99.8 %. DWI can identify ischemic stroke within minutes after symptom onset; however, about 7 % of stroke occur with DWI negative scans. DWI-negative stroke patients usually present mild clinical deficits, low NIHSS score (usually < 5), such as isolated internuclear ophthalmoplegia or ataxic hemiparesis [35].

DWI-negative strokes are usually associated with small lacunar ischemic lesions, in the territory of the small perforating arteries of the basilar artery, typically in the brainstem [36]. Patients with posterior circulation stroke are five times more likely to present with a negative DWI scan than patients with anterior circulation ischemia. In fact, only few studies report DWI-negative scans in the anterior circulation, mostly limited to subcortical strokes.

Many hypotheses have been developed on DWI negative strokes, based on duration and degree of hypoperfusion. The reduction in blood flow may be sufficient to cause symptoms (electric failure), but insufficient to cause a DWI lesion (maintained ionic homeostasis). Perfusion imaging may theoretically detect such cases, although very small dimension ischemia limits its utility. Moreover, if the duration of hypoperfusion is relatively short, DWI scans may result in false-negative findings [37]. It was reported that about one third of patients with initial negative scans develop a hyperintense lesion on a second scan. Finally, some technical features of the scanner may be involved: some small lesions may be more easily identified by stronger magnetic fields, or by increasing the b-value and reducing the slice thickness or adding coronal and sagittal DWI scans.

Although DWI is the most sensitive technique for identifying acute ischemia, stroke is still strongly a clinical diagnosis. Therefore, patients with acute focal neurological deficits suggesting stroke should be treated with acute reperfusion treatments even in cases of negative DWI scan, in absence of absolute contraindications.

Moreover, as the risk of recurrence is similar in DWI negative and positive stroke, a thorough etiopathogenetic evaluation is needed to optimize secondary prevention strategies [38].

3. Conclusions

Unusual presentation of stroke is infrequent. However, stroke misdiagnosis carries a high risk of poor outcome and mortality, mainly driven by missed acute reperfusion treatments and secondary prevention strategies. Continuous personnel education is needed to reduce the risk of stroke misdiagnosis and improve patients' outcome.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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