An atypical presentation of Todd's Paralysis

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ABSTRACT

Stroke mimics (SM) such as Todd's paralysis pose a major diagnostic challenge and may be difficult to distinguish from Transient Ischemic Attacks (TIA). The aim of this case presentation was to highlight a case where the presentation made it difficult to ascertain the true cause of patients' quadriplegia.

INTRODUCTION

Transient Ischemic Attack (TIA) is a critical medical condition denoting a transient episode of neurologic dysfunction that is triggered by localized ischemia in the brain, spinal cord, or retina, without acute infarction or tissue damage.[1] Urgent evaluation of TIA is essential, necessitating prompt deployment of advanced imaging and comprehensive laboratory studies to mitigate the risk of subsequent strokes. In light of the increased prevalence of stroke and the expanding availability of sophisticated therapeutic interventions, precise diagnosis within the stipulated timeframe is of paramount importance. Within the diagnostic landscape, discerning 'stroke mimics' (SM) in cases of false positive diagnosis and elucidating 'stroke chameleons' for false negatives are important considerations.[2] The differential diagnoses for suspected stroke encompass seizures, syncope, sepsis, migraine, space-occupying lesions, functional disorders, and metabolic diseases, as outlined extensively in contemporary literature.[3] Todd's Paralysis or Paresis manifests as a syndrome characterized by weakness or paralysis affecting part or of the body immediately following the cessation of an ictal discharge (seizure). While it frequently impacts a single limb or one side of the body, its presentation encompasses a broad spectrum of manifestations.[4] Todd's paresis demands acute differentiation from TIA to facilitate the expedited management of time-sensitive conditions and curtail unnecessary healthcare expenditures, thereby optimizing patient outcomes and resource allocation in the medical domain.

CASE PRESENTATION

We present a case of a 41-year-old female with a 6-year history of seizure disorder being followed up at the neurology outpatient clinic at the San Fernando General Hospital. The patient has had a history of recurrent Todd's paresis with right hemiparesis which usually resolves after 2-3 days. She has been seizure free for the past 3 years and well controlled on lamotrigine 100mg taken twice daily.

The patient was brought to the Accident & Emergency department by relatives due to the onset of global paresis. According to the relatives, the patient initially experienced drowsiness, dizziness, and headaches. Shortly after, the patient had an acute episode characterized by involuntary twitching of the body that started in the right arm. She also experienced eye rolling and was unresponsive to external stimuli that lasted approximately 4 minutes. This was then followed by an episode of urinary incontinence and a loss of awareness lasting approximately 8 hours. On awakening, the patient denied having the ability to move both upper and lower limbs bilaterally. The patient stated that she could not recall any events after initially experiencing drowsiness, dizziness and headaches.

On further questioning the patient denied having any known vascular risk factors, preceding viral illness, head injury, drug or alcohol use or exposure to toxins. Her GCS was assessed as 9/15 E- 4 V- 4 M- 1 with normal vital signs. Clinical examination revealed 0/5 power in all limbs, reduced tone and normal reflexes, with intact sensory and cranial nerve function. Pupils were equally reactive bilaterally. The patient's renal, liver, thyroid functions, Complete Blood Count (CBC), ESR, CRP and urinalysis were normal with negative urinary pregnancy test. Her Covid-19 PCR and HIV tests were negative. Non-contrast CT brain and C-spine, MRI/MRA brain, and whole spine MRI also showed no abnormalities. Her EEG showed moderately abnormal EEG with bilateral sharp activity with mixed theta activity seen throughout the hemispheres without lateralisation. ECG revealed sinus rhythm with nil acute changes. She was diagnosed with Global Todd's paresis and treated with Lamotrigine 125 mg twice daily and Clonazepam 0.5mg.

Her hospital course was uneventful with no further seizure episodes reported by patient or nurses in addition to any deterioration of her condition. She remained stable with gradual return of power in her limbs over the following 4 days and was able to ambulate on day 6 without assistance.

During this case, differential diagnoses were carefully considered including conditions such as hypokalemic periodic paralysis which was deemed unlikely due to unremarkable potassium and thyroid screening results. Similarly, spinal trauma leading to cord compression was considered, however, imaging did not reveal any fractures or compressive lesions to suggest that this may be a potential cause.

DISCUSSION

Distinguishing between conditions with overlapping clinical presentations, such as Todd's paralysis, transient ischemic attack (TIA), and acute ischemic stroke, poses significant clinical challenges. Healthcare professionals, especially those with limited resources, may resort to cognitive biases such as anchoring, availability, and

premature closure, drawing from their clinical experience and mental algorithms to reach a diagnosis. While these strategies are often effective in managing straightforward cases, they can lead to misdiagnosis in complex scenarios like stroke or stroke mimics, potentially impacting patient outcomes. Comparisons of these diagnoses and presentations are found in Table 1. Todd's paralysis, also referred to as Todd's paresis or Todd's palsy, occurs in about 13% of seizures.[5] It affects individuals regardless of gender, age, or race.[6] Diagnosing Todd's Paralysis can be challenging, especially in adults with comorbid conditions like chronic hypertension or diabetes mellitus due to increased risk of intracranial pathology. Misdiagnosing Todd's Paralysis as limb weakness post-acute ischemic stroke (AIS) may lead to unnecessary intravenous thrombolysis that is contraindicated in neurological deficits.[7] Distinguishing Todd's Paralysis from a cerebrovascular accident (CVA) or transient ischemic attack (TIA) in hemiplegic patients without an epilepsy history is clinically demanding.[1] Currently, there are no standardized diagnostic tests for Todd's Paralysis, necessitating a review of symptoms and medical history. MRI and EEG provide structural and functional evidence to identify organic causes.[8]

Factors aiding in Todd's Paralysis differentiation include:

1. Its occurrence after partial or generalized tonic-clonic seizures.[9]
2. Higher incidence in elderly patients or those with prior stroke history.[10]
3. Varying duration from minutes to days.[11]
4. Its multifactorial etiology involving cerebral perfusion abnormalities post-seizure.[12]

With regards to the treatment of this patient, lamotrigine was chosen as the primary treatment for this patient's seizures. This is based on the results of the SANAD trials, which is the largest randomized trials comparing different antiepileptic drugs (AEDs) as monotherapy. These trials demonstrated that lamotrigine is particularly effective in managing both focal and generalized seizures, offering a superior time to treatment failure compared to other AEDs.[13] Despite lamotrigine's strong efficacy as a monotherapy, this patient's epilepsy did not achieve remission with lamotrigine alone. Consequently, clonazepam, an adjunctive treatment known for its effectiveness in cases refractory to other antiepileptic medications,[14] was added to the patient's regimen.

Table 1. Comparisons made between AIS, TIA and Todd's Paralysis

	TIA/AIS	Todd's Paralysis
Causes Include	Impaired oxygen supply to cerebral tissue	Mechanism is likely multifactorial following a seizure
Presenting symptoms	Dependent on ischemia location, size and duration.	Dependent on seizure foci
	Sudden headache, altered mental status, unilateral palsy/paresis, aphasia, visual deficits	Hemiparesis— motor cortex Visual changes—occipital lobe Hemineglect—right parietal cortical lesion
Diagnostic Tests	Computed tomography, MRI, National Institutes of Health Stroke Scale etc.	No single diagnostic tool
Treatment	Thrombolytic therapy (in AIS)	Supportive therapy

CONCLUSION

We report this case as we believe it to be an atypical presentation of the classic Todd's Paresis in a known epileptic. We recommend that physicians be cognizant of this presentation as it can overlap with non-convulsive status epilepsy or a non-organic illness which may lead to difficulty in both the diagnostic and therapeutic aspects of the management of the patient.

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