

A Stroke, is a Stroke... is a  
Stroke....or is it?



## Stroke Mimics and Red Flags



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# Outline of Presentation

- Define “Stroke Mimic” and “Stroke Chameleon”
- Why is it important to discriminate between a mimic and “the real thing”
  - Acute presentation management/ thrombolysis candidate
- Common signs and symptoms and what are the clues to being a “stroke Mimic”
- Common examples of a stroke mimic/ chameleon
  - Complicated Migraine
  - Seizure and Todd’s paresis
  - Vertigo/ BPPV
  - Cerebral tumour
  - Other cerebral pathology
  - Spinal pathology
  - Functional

# Initial Assessment

**Sudden loss of focal brain function in a vascular territory is the main feature of the onset of stroke.**

- Patients with conditions other than brain ischaemia may present in a similar fashion.
- Patients suffering a stroke may present with other serious medical conditions.
- The initial evaluation requires a rapid but broad assessment.

**The goals in the initial phase include:**

- Ensuring medical stability, with particular attention to airway, breathing, and circulation
- Quickly reversing any conditions that are contributing to the patient's problem
- Determining if patients with acute ischemic stroke are candidates for thrombolytic therapy
- Moving toward uncovering the pathophysiologic basis of the patient's neurologic symptoms

# Stroke Mimic

## Mimic

- To resemble closely; simulate
- To take on the appearance of
- To imitate.



Stroke Mimic: a non vascular disease that presents with stroke-like symptoms that are often indistinguishable from stroke. (Long et al, 2016)

- 20-25% of suspected stroke presentations
- 39% mimic – (or non vascular cause) in the TIA Clinic RNSH (July 2011 – June 2013) (O'Brien et al,2016)

# Stroke Mimics

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Graphic 69869 Version 6.0



Migraine aura

Seizure with postictal paresis (Todd paralysis), aphasia, or neglect

Central nervous system tumor or abscess

Functional deficit (conversion reaction)

Hypertensive encephalopathy

Head trauma

Mitochondrial disorder (eg, mitochondrial encephalopathy with lactic acidosis and stroke-like episodes or MELAS)

Multiple sclerosis

Posterior reversible encephalopathy syndrome (PRES)

Spinal cord disorder (eg, compressive myelopathy, spinal dural arteriovenous fistula)

Subdural hematoma

Syncope

Systemic infection

Toxic-metabolic disturbance (eg, hypoglycemia, exogenous drug intoxication)

Transient global amnesia

Viral encephalitis (eg, herpes simplex encephalitis)

Wernicke encephalopathy

# Stroke Chameleons

Strokes with atypical presentations that take on the appearance of other disease processes, for like the chameleon, these disguised strokes may change and evolve with time.

(<http://www.ferne.org/Lectures/strokemimics0302.htm#T> accessed July 2016)



# Stroke Chameleons

(Fernandes et al 2013)

	Presentation
Bilateral Thalamic Stroke	Persistent acute global amnesia
Limb-Shaking TIA	Focal epilepsy
Cortical Stroke	Peripheral nerve lesion
Bilateral occipital strokes	Confusion/ delerium



# Stroke Chameleons

- Atypical presentation of stroke:
  - Movement disorders
  - Acute hemiballismus – can result from an infarct of the subthalamic nucleus
  - Dyskinesias –hyper/hypokinetic –can be found with ischaemic lesions at the motor cortical or subcortical regions
  - Confusion, agitation and delerium – lesions in the limbic cortex or orbitofrontal region
  - Sensory deficits with parasthesia or loss of sensation can occur with parietal and thalamic strokes.

(Long 2016)



# Distinguishable Features and Red Flags



# Red Flags



- **Age** (young less likely for TIA/Stroke but not out of the question)
- **Duration** (rapidly resolving, re-occurring)
- **Nature of the symptoms** “positive” or “negative”
- **Precipitating factors** (alcohol, drugs, risk factors..)
- **Onset and progression** (sudden vs stepwise)
- **Associated symptoms** (headache, loss of awareness/consciousness – during or after)

# Nature of the symptoms-

(Nadarajan et al 2014, Up To Date July 2016)



- Positive symptoms
  - Indicate an excess of central nervous system neurone electrical discharges
    - “visual” – flashing lights, zig zag shapes, lines, shapes, objects
    - “somatosensory”- parasthesia, pain
    - “motor” – jerking limb movements
- Negative symptoms
  - Indicate a loss or reduction of central nervous system neurone function – loss of vision, hearing, sensation, limb power.
- Migraine, Seizure are characterised with having “positive” symptoms
- Whereas TIA / Stroke present with “negative” symptoms.



- **Loss of consciousness**
  - Common in seizures and syncope, uncommon in TIA/ stroke
  
- **Precipitating factors**
  - E.g. Seizure – alcohol, drugs, sepsis, hyperventilation, non-compliance with AEDs
  - Hypoglycaemia- non specific symptoms but can include fatigue, dizziness, visual disturbances, drowsiness, dysarthria and depressed mental state – can proceed to seizures or coma if not corrected.
  - Electrolyte imbalance – hypo/hyperkalaemia, hypo/hypercalcaemia, hypomagnasemia, hypophosphataemia – can cause generalised or focal muscle weakness
  - Sepsis – malaise and generalised weakness

(Nadarajan et al 2014, Up To Date July 2016)

# Predictors of Stroke Mimics (Long 2016)

## Increased odds of stroke

- Abnormal eye movements
- Higher SBP
- Increased DBP >90mmhg
- History of AF or angina

## Increased odds of stroke Mimic

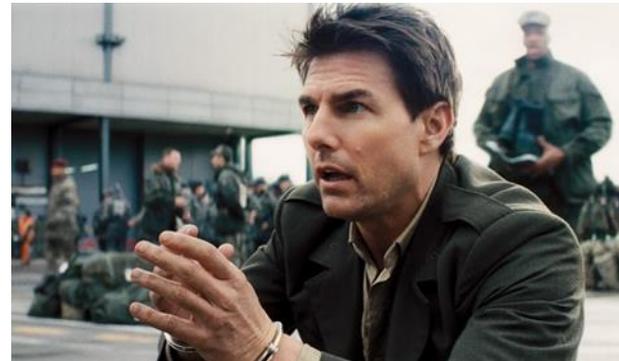
- Decreased LOC
- Normal eye movements
- Cognitive dysfunction
- Female
- Younger age



## Safety of IV Thrombolysis in Stroke Mimics

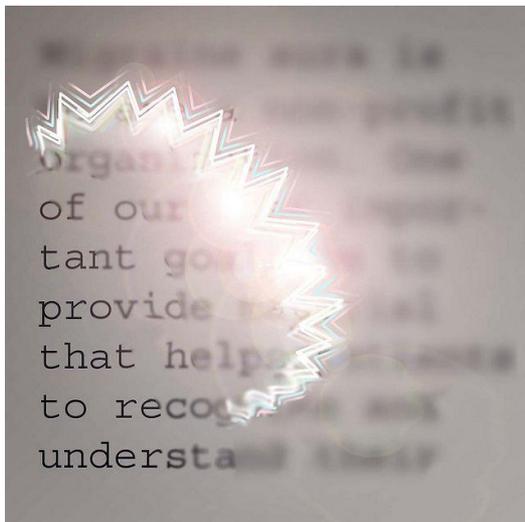
- Safety of tpa was reviewed in a prospective single centre study by Tsivgoulis et al (2015)
  - 14.5% of tpa cases were stroke mimics – with 1.3% (1) fatality (encephalopathy with uncontrolled HTN) vs 1.13% in confirmed Stroke
  - and 2 minor extracranial haemorrhages
- The meta analysis of 8 studies
  - showed a decreased risk of sICH after IV Tpa in Stroke mimics and a favourable functional outcome (mRS 0-1)
- Indicates that IV Tpa does not adversely effect the favourable natural history of stroke mimics
- Treatment benefit would not be counterbalanced by the potential for harm to patients presenting with stroke mimic

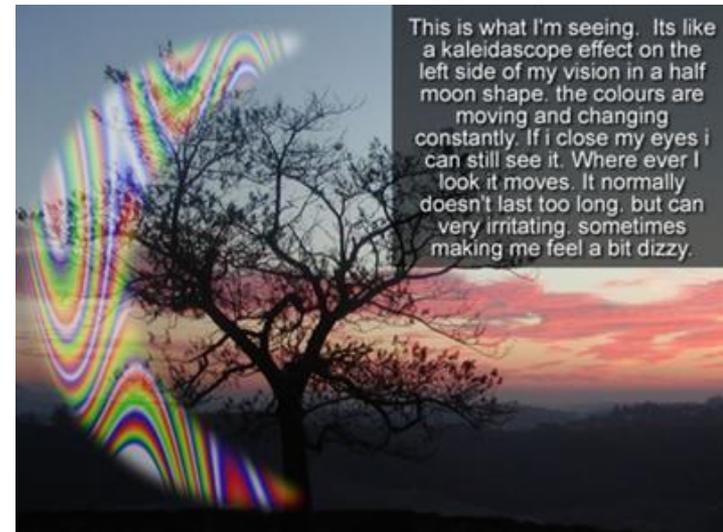
# The Ringleaders!



# Migraine

- **Migraine aura**
  - Progressive neurologic deficit that completely recovers
  - Occur before the onset of migraine headache
    - Can be visual, sensory, motor or speech
  - Headache usually simultaneous or just after the end of the aura – rarely occurs an hour or more after the aura
  - Can have aura with minimal or no headache
- Usually resolve within 20- 30 minutes, lasting seldom more than an hour





- **Migraine aura**

- often progresses slowly in one modality

- i.e.

- scintillations or bright objects move slowly across the visual field

- Parasthesia may progress gradually from one finger, to all, to the wrist, forearm, shoulder, trunk, then face and leg (over minutes)

- After the positive symptoms move they can then be followed by loss of function. Visual scintillations can end in a scotoma or visual field deficit/ parasthesia can leave the areas of skin numb (stroke mimic)

- Recurrent/ stereotyped

# Seizure



Seizure with post ictal hemiparesis (Todd paralysis), aphasia, or neglect

- Can cause neuronal dysfunction that can manifest as weakness, aphasia, confusion or sensory symptoms (stroke mimic)
- Todd's paresis/ unilateral hemiparesis - localised to one side, can last up to 20 minutes but can continue for up to 48 hours. (13% of seizures)
- Abrupt onset with positive features in a single functional neurological domain, recurrent and stereotyped.

# Syncope

- Abrupt and transient loss of consciousness
- Absence of postural tone
- Rapid and usually complete recovery
- Caused by an interruption of energy sources to the brain – usually caused by a sudden reduction of cerebral perfusion

Common types of syncope include:

- Neurocardiogenic (vasovagal) syncope
- Situational syncope (during or immediately after urination, defaecation, swallowing)
- Orthostatic syncope (associated with orthostatic hypotension)
- Syncope related to cardiac ischemia or cardiac arrhythmia



# Vertigo

- Benign Positional Paroxysmal Vertigo
  - Labyrinthitis
  - Vestibular Neuritis
- 
- Isolated dizziness – peripheral vestibular disturbance
    - Only 3% of stroke patients present with dizziness plus additional symptoms
    - 1% with lone dizziness
  - Movement induced
  - Up to a few weeks
  - Normal MRI imaging



# Vertigo

- Positive Hallpike's and Head Impulse test can distinguish between a peripheral lesion and central cause (Fernandes 2013)
  - Nystagmus inducing in peripheral lesions
- Lateral medullary, lateral pontine and inferior cerebellar infarcts can mimic the clinical symptoms of vestibular neuronitis (chameleons).
- Nystagmus in acute cerebellar stroke has the fast phase toward the side of the lesion



# Bleeds

(highlights the importance of rapid assessment and imaging)



- SAH
  - sudden onset of severe headache is the most common presentation
  - Accompanied by LOC, seizures, nausea and vomiting and meningismus
  - **Lateralising signs such as unilateral weakness are uncommon**
- SDH
  - Posterior fossa SDH presents with symptoms of elevated intracranial pressure including headache, vomiting, anisocoria, dysphagia, cranial nerve palsies, nuchal rigidity, and ataxia.
  - cerebral hypoperfusion due to increased intracranial pressure or mass effect may culminate in cerebral infarction.
  - **Global deficits such as disturbances of consciousness are more common than focal deficits after SDH.**

# Tumours and TGA

- Tumours
  - Usually a slowly increasing mass – progressive syndrome
  - Patients with brain tumours (6%) presenting to ED with acute –stroke like symptoms (<1 day) may be more indicative of haemorrhage into the tumour, or those developing obstructive hydrocephalus (Fernandes 2013)
- Transient Global Amnesia (Nadarajan 2014)
  - Characterised by transient loss of anterograde memory
  - Can last for a few hours
  - Filling of old memory and new memories will occur but a gap in memory during the event persists
  - Procedural memory intact – repetitive questions common
  - Vascular risk factors
  - Rarely reoccurs
  - Can see small pinpoint DWI lesions in the hippocampus mainly on the Left but can be bilateral

# Spinal pathology



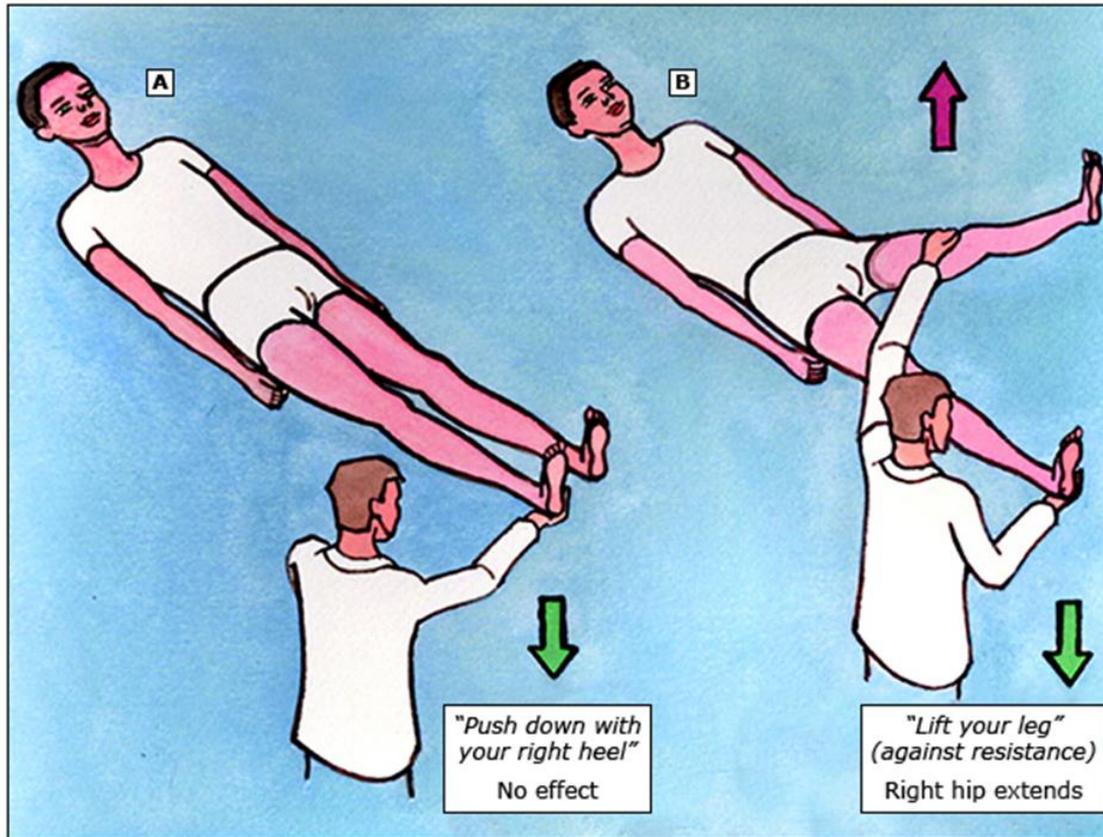
- Presentation with acute flaccid paralysis
  - Bilateral limb weakness
  - Sensory deficits with a discrete level below which sensation is absent or reduced
  - Bladder function impairment
    - Spinal infarcts
    - Anterior spinal artery infarcts (can also present with unilateral weakness)
    - Brown-sequard syndrome
    - Transverse myelitis
- **Compressive myelopathy from neoplasm, epidural or subdural hematoma, or abscess is the most important category of diagnosis to exclude, as these often require urgent surgical decompression.**
  - lesions typically develop over time, sometimes the clinical presentation can be fairly abrupt and mimic spinal cord infarction.

# Functional



- Often manifest as acute weakness or sensory disturbance
- Frequently a trigger
  - Panic attack or dissociative episode
- Guiding principle
  - Inconsistency during physical examination
    - No ankle plantar flexion while lying down, but the ability to stand on tip toes
    - Task dependent weakness - Inability to move arm during examination, but able to use arm to take something out of a bag or put shoes back on
    - A positive Hoover's sign \*
  - Incongruity with recognised neurological disease
- Often can have underlying disease or disease that manifests later

## Hoover's sign for functional leg weakness



(A) Hip extension is weak when tested directly.

(B) Hip extension is normal when the patient is asked to flex the opposite hip.

Reproduced with permission from: Stone J, Zeman A, Sharpe M. Functional weakness and sensory disturbance. J Neurol Neurosurg Psychiatry 2002; 73:241. BMJ Publishing Group Ltd. Copyright © 2002. (Up to Date 2016)

# Functional

Functional facial symptoms with contraction of muscles causing jaw deviation

- Can lead to an appearance of weakness (even though they are caused by muscle overactivity)
- An acute presentation can lead to a diagnosis of stroke

Cortical blindness

- Patient reports being blind but has normal pupillary reflexes and preserved optokinetic nystagmus
  - MRI may reveal bilateral occipital pathology causing cortical blindness
  - A small subset of patients with cortical blindness think they can see when they cannot (Anton's syndrome)

Cognitive symptoms

- Impaired fluency
- Jumbling of words when speaking
- Word finding difficulty
- Marked loss of remote autobiographical memory
- Inability to perform over-learned skills such as reading, spelling, or simple arithmetic
- Ability to perform complex implicit cognitive tasks in the presence of poor performance on simple explicit tasks
- Performance inconsistent with observed behaviour and at different points in the examination or across repeated evaluations

Symptoms may also be attributable to anxiety or depressive disorders

# Management and Ongoing Care

- Prompt diagnostic testing to confirm or negate the diagnosis.
- Explain what they do have “functional weakness – your nervous system is not damaged”
- Indicate you believe the patient
- Explain what they don’t have
- Common/ reversible
- Self help/ written information
- Psychiatric referral
- <http://www.neurosymptoms.org/> (Functional and Dissociative Neurological Symptoms – a Patient Guide)



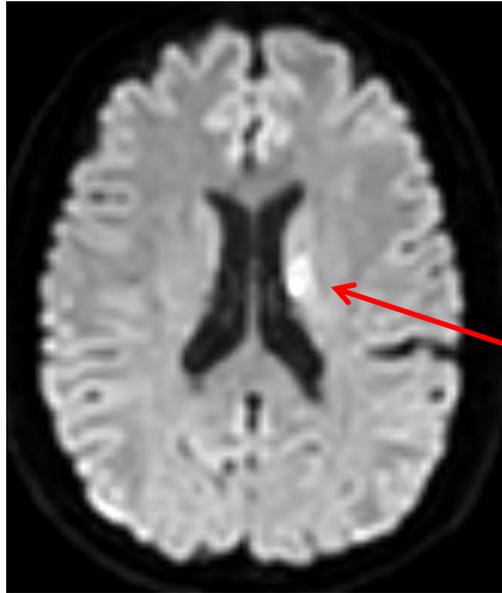
# Case Study 1. TD

- 42 year old female
- acute onset of right arm heaviness – she was unable to pick up the phone.
- She felt flushed and dizzy – pre syncopal –
- ataxic,
- dysarthria and difficulty swallowing.
- lasted 4-6 hours.
- preceded by a migraine that morning relieved with mersyndol and sumatriptan – it however reoccurred later that day. TD had also experienced similar symptoms twice in the previous week lasting 1 ½ and 4 hours.

# Case Study 1. TD

- No hypertension,
- pre-eclampsia with her first child.
- No diabetes.
- Smoker – 15/day.
- Migraines – x2 per week preceded by a visual aura, not cycle related.
- Alcohol intake – 2-3 serves per day, no alcohol free days.
- Stressful work
  
- Impression – Complicated Migraine

# Stroke



- acute to subacute infarction within the deep penetrating branch of left middle cerebral artery.

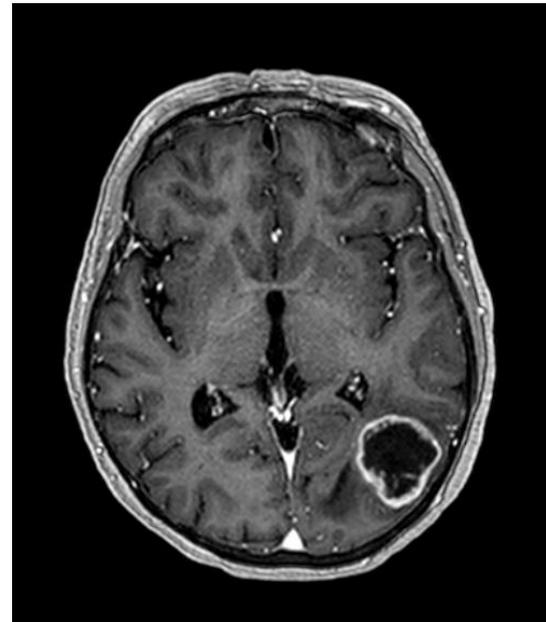
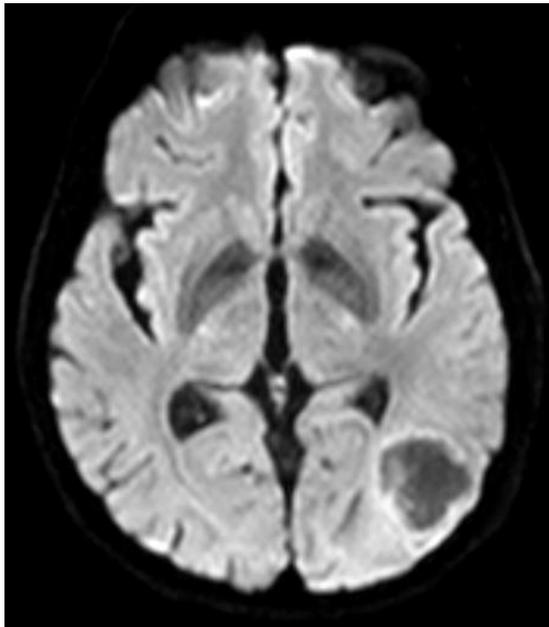
## Case Study 2. CM

- 73yo female
- Episode of expressive/receptive dysphasia. Ongoing mild alexia.
- Odd behaviour

Impression: ?TIA/ ?Functional

## Case Study 2.

- Intra-axial mass lesion in left temporo-occipital region most probably glioma.



# Take Home Messages



- ☺ Discerning review and investigation of presentations to ascertain if stroke or stroke mimic.
  - ☺ Review eligibility for acute treatments
- ☺ Thrombolysis in stroke mimics has a relatively small risk
- ☺ Stroke chameleons can miss out on potentially beneficial stroke treatment if not assessed and managed appropriately.

Thankyou

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