



Episode 6 – Transient Ischemic Attacks

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Definition

Classic definition of TIA is neurologic dysfunction caused by focal brain or retinal ischemia lasting <24hrs. Recent definition has changed to 1hr because up to 50% of classically defined TIAs are actually infarcts as shown on MRI

Risk Stratification Scores

ABCD2 score: Combination of California Score and ABCD Score, and most relevant to Emergency Physicians because of 2-day risk of stroke (as opposed to 90 days or 7 days for the others)

Age >60 (1 point)	Score 0-3 = low risk (1% risk of stroke at 48hrs)
Blood pressure >140 systolic or >90 diastolic (1 point)	
Clinical features: Unilateral weakness (2pts), Speech disturbance (1pt)	Score 4-5 = moderate risk (4% at 48hrs)
Duration of symptoms: >60min (2pts), 10-60min (1pt), <10min (0pt)	
Diabetes (1 point)	Score 6-7 = high risk (8% at 48hrs)

Key historical features

Abrupt onset of *focal* neurological symptoms, with the features identified by the ABCD2 score;

Other relevant features: multiple episodes (eg, crescendo), associated headache or trauma, history of atrial fibrillation or risk for cardioembolic source (mural thrombus, carotid stenosis, patent foramen ovale – PFO)

TIA/CVA Mimics

Partial seizure: context is crucial because a Todd's paresis will occur in the setting of a focal seizure (or generalized with focal onset) with pre-existing CNS lesion, stroke or tumor (which should be seen on a CT scan)

Migraine: migraine-equivalent visual disturbances may occur – migraine aura without the associated headache; the symptoms are usually positive (scintillating scotomas, shimmering) and asynchronized (the migraine “marches”, with visual disturbance then a speech or weakness appears) in visual phenomena, versus negative (actual visual loss, obscuration) and synchronized (both visual and motor disturbances simultaneously) in TIA

Other diagnoses to consider: Hypoglycemia (it CAN cause focal symptomatology), subdural hematoma, brain tumor, labyrinthitis/neuritis, transient global amnesia, severe postural hypotension, arrhythmia, cervical disk disease, cerebral venous thrombosis, conversion disorder



Key physical exam findings

Most important are facial droop, pronator drift and abnormal speech

Head and neck: pupillary size and EOMs, facial droop and abnormal speech, level of alertness

Cardiovascular: heart rate and RHYTHM (eg, a.fib), blood pressure and neck bruits

Motor exam (and reflexes, Babinski): Grip strength and big motor groups are too gross examinations; use also finger extension and pronator drift for >10sec

Romberg test and gait (including tandem gait)

TIA classification and tests (rule of 4s – 4 types and 4 investigations)

<u>Types of TIA</u>	<u>Appropriate investigations</u>
Cardioembolic (¼ of TIAs)	ECG: Look for a.fib (12-16% of stroke) and other cardiac problems that might cause embolic phenomena from the heart: CHF, recent MI, left ventricular aneurysm, rheumatoid heart disease, valvular disease); if the ECG looks like acute MI and a severe headache is present, think of ICH and SAH
Lacunar (small arteries) TIAs (¼ of TIAs) Classically: pure motor and pure sensory, or mixed-motor-sensory but without cortical findings (below)	Unenhanced CT head: To rule out TIA mimics (tumor, SDH, ICH, SAH – especially given that ASA will be given), and see old strokes
Large arteries in neck, brain (¼ of TIAs) Cortical findings: aphasia (sometimes mistaken for confusion) means left-hemispheric involvement; neglect means right-hemispheric involvement; visual disturbances (field cuts)	Vascular imaging: Organize for carotid Doppler in the neck 1-3d maximum, or consider adding CT-angiogram to CT head (which can assess for intracranial arteries) – see discussion below
Other TIAs (¼ of TIAs) Intracranial hemorrhages (15%), cryptogenic, clotting disorders, PFOs	Blood: Accucheck and blood work (CBC, lytes and blood sugar, BUN, Cr, INR)

Other important causes of TIA:

Carotid dissection: young patient with no risk factors, especially if associated with trauma or sudden, rapid movement of the neck, neck pain (anterior pain in carotid dissection, posterior pain in vertebral dissection), vertigo or headache

Endocarditis: unwell for weeks, flu-like symptoms, recurring fever, weight loss, and headache



Vascular Imaging (NASCET trial)

Essential to organize early imaging (1-3d max) given that carotid endarterectomy (CEA) performed within 2wks of TIA gives an absolute risk reduction (ARR) of 30%, or a number needed to treat (NNT) of 3 – unparalleled in terms of benefit compared to other treatments we can offer; early treatment is crucial given that the ARR drops to 16% if CEA is performed 2-4wks after TIA, and close to 0% if after 4 weeks; interestingly, the benefit of early CEA seems to drop in women after 3wks, emphasizing the need for prompt treatment

Consider CTA if there is no renal dysfunction nor allergies to contrast, especially in a convincing TIA story or concern for dissection (the vascular imaging will be done immediately and admission will be considered, as opposed to a few-days outpatient management); carotid Doppler, although still good, cannot assess intracranial circulation and is an imprecise measure in certain cases

Echocardiography: lower priority test as the yield is low, especially if there is no known cardiac disease; consider it in patients for which there is no explanation of the TIA/stroke

Acute TIA management

Maximize cerebral blood flow by rehydrating dehydrated patients, and by resisting the urge to rapidly lowering the blood pressure

Antiplatelet medications (decreases the risk of stroke – and MI – by ¼)

ASA should almost always be the first option, and given as soon as there is no bleed as confirmed by CT head, with loading dose of 160-325mg NON-enteric coated chewed, then 81mg daily

In severe ASA allergy, Clopidogrel is the only choice

If ASA failure (ensure compliance first), Plavix™ and Aggrenox™ are equally effective and at least as good as ASA, but Plavix™ has a better side-effects profile

Plavix™ (Clopidogrel) should be given either as a loading dose of 300mg (onset of action 6-7hrs) then 75mg daily, or overlapped with ASA for 3-4d if no loading dose is given (onset of action 3-4d); do not combine ASA + Clopidogrel for extended periods as the risks of major bleeding and ICH outweigh the benefits (MATCH trial)

Aggrenox™ (dipyridamole 200mg + ASA 25mg) should be given BID, but patients should be warned of the frequent severe headaches accompanying the medication, and therefore should be directed to take acetaminophen with the first few doses

Heparin should probably never be started by the EP (even in the setting of a.fib), except possibly in crescendo TIAs with suspected critical carotid artery stenosis or tight basilar artery occlusion in consultation with a neurologist

Disposition

Admit high-risk patients with suspicion of large vessel disease, especially if other “soft factors” that would delay care are present: non-English speaking, no means of transportation, unreliable patient or family, impoverished); other patients can be referred to stroke clinic for prompt assessment



Medications for home

Reduction in blood pressure leads to lower events in the long run, but can be dangerous if performed too rapidly and acutely; it is therefore reasonable to let the patient know to discuss anti-hypertensive medications with their GP; alternatively, for hypertensive patients in the ED, low-dose medications can be started (eg, Hydrochlorothiazide 12.5mg, Ramipril 2.5mg or Telmisartan 40mg daily – follow up with the GP for electrolyte monitoring is essential)

Coumadin to prevent stroke in patients with Atrial Fibrillation:

Risk stratification is essential: CHADS Score (CHF, HTN, Age>75, DM, previous Stroke) ≥ 2 should get warfarin given the benefits – 70% risk reduction in embolic events – and the risks – moderate bleeds (10-12%), or severe bleeds (2-3%) requiring hospitalization, transfusion, or causing ICH

Remember that older age means higher risk for embolic event, so a 92y.o. woman who everyone is afraid of giving warfarin to is probably the person who would most benefit from it

Vertigo

4 types of dizziness (there’s always one that predominates):

Syncope / pre-syncope: feeling of passing out	Vertigo: hallucination of rotation or linear movement; spinning
Disequilibrium: cannot walk properly, staggering	Non-specific light-headedness

4 types of vertigo (based on duration of symptoms):

Less than 60sec	Positional event (MARKEDLY worse with movement)
Minutes (few to 30)	If not positional: migraine (in young, low-risk patients) or TIA/CVA (in older, at-risk patients)
Many hours	Vestibulopathy, Ménière’s disease
Days	Labyrinthitis, or stroke



Approach to vertigo:

The easiest way to rule out an ominous central cause is by ruling in a benign peripheral cause:

1. BPPV: <1min, normal in between attacks, Dix-Hallpike positive (see below), Epley maneuver cures it in 50% of cases (see youtube)
2. Vestibular neuronitis: acute severe constant vertigo, positive head-thrust manoeuvre (see below)
3. Ménière's disease: >20mins to hrs in combination with tinnitus, ear fullness or decreased hearing

History:

Features suggestive of posterior circulation ischemia: diplopia, ataxia (especially between episodes), dysarthria (slurred speech) and dysphagia – and central cause: non-positional or bidirectional nystagmus, inability to ambulate, focal neurological deficit and cerebrovascular risk factors

Note: all causes of vertigo can be worsened by head movement

Physical examination:

Unidirectional horizontal nystagmus is usually peripheral in nature, whereas vertical, pure torsional and/or bidirectional nystagmus, limb ataxia and pinprick sensation asymmetry are usually central

Dix-Hallpike test:

Useful in ruling in BPPV only if CLASSIC response: brief latency of 3-5 seconds, dramatic response with vertical or rotatory eye movements that stop after 30-60sec, and fatigability if the test is repeated

Head thrust test:

Abnormal test (unilateral latency in eye repositioning when the head is thrust) is indicative of peripheral lesion (eg, vestibular neuronitis with acute, severe and constant vertigo) almost always

Medications:

Antihistamines (eg, dimenhydrinate), antidopaminergic (prochlorperazine - Stemetil™), and/or anticholinergic (atropine 0.4-0.6mg IM or Scopolamine 0.5mg patch) may be tried

Serc™ (betahistine) should ONLY be prescribed for Ménière's disease