MONTHLY FEATURE BEST PRACTICE STATEMENT SUMMARY

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Key Words: Emergency department (ED), transient ischemic attack (TIA), stroke

Scope of Guideline: Not specified; presumably all clinicians evaluating TIA in the ED.

Inclusion/Exclusion: Not specified

Key Guidance:

1) Clinical Evaluation:

- **a.** Definition of TIA = acute onset of focal neurological symptoms followed by complete resolution/return to baseline.
- **b.** TIA mimics are summarized in Table 1 below.

2) Diagnostic Evaluation:

- **a. Laboratory/Cardiac Testing**: The usual ED work-up for query TIA/CVA are applicable (eg. ECG, POCT glucose, CBC, chemistry, HBA1C, troponin and fasting lipid profiles). For suspected temporal arteritis (age>50), consider adding CRP and ESR levels. Infection and toxicology tests can be added as clinically warranted.
  - **i.** Initial ECG can detect atrial fibrillation in 7% of ED TIA/CVA patients, but longer telemetry could have higher detection rates.
- **b. Risk Stratification Tools**
  - **i.** Assess for usual CVA/CVS vascular risk factors (eg. smoking, HTN, hyperlipidemia, diabetes, family history).
  - **ii.** The different ABCD risk score variants have different levels of validation in ED settings. The $ABCD^2$ score has high sensitivity but low specificity to risk-stratify low- (score 0-3) vs high- (sore 6-7) risk patients (meta-analysis of 33 studies, >16000 patients); low-risk early stroke occurred in 2.1% of patients, whereas high-risk patients had 7% stroke incidence.
  - **iii.** Patients with dual/crescendo TIAs (2+ episodes of TIA symptoms within 1 week, increasing duration/frequency/severity of events) are higher risk and may warrant admission for expedited workup for ipsilateral carotid stenosis (ie. "unstable angina of the brain.")
- **c. Imaging:** Summary characteristics of DI modalities are summarized in Table 2 below. In the ED setting, NCCT is not very sensitive to rule out small acute strokes, but can help rule out TIA mimics. MRI (with DWI) is the preferred modality for acute small infarcts, but may not be readily accessible in ED settings. NCCT and CTA may be useful to evaluate intra-cranial hemorrhages or symptomatic stenoses. Risk of contrast-nephropathy is low with CTA, and worth the risk in cases of suspected stroke/bleed.

3) **Disposition:** It may not be practical to complete a comprehensive TIA workup in the ED setting. As such, creating an ED discharge protocol with expeditious follow-up for TIA patients is critical to reduce the risk of early post-TIA stroke (ideally within 48hrs). Use of structured ED TIA diagnostic pathways have been shown to reduce time to imaging, ED LOS, admissions and cost-savings without sacrificing short term strokes or mortatlity. Many of these pathways have been developed/validated in certified stroke centers, so generalizability to non-stroke ED centers may not be valid. The **Figure** below outlines the key elements of a successful ED TIA pathway, and requires all elements to be functioning cooperatively with appropriate partners (eg. radiology, neurology outpt clinics).

Table 4 provides suggestions for post ED discharge secondary prevention. For patients with $ABCD^2$ >4pts (higher risk), they should be started on ASA 81mg PLUS clopidogrel 75mg daily (dual antiplatelet therapy; DAPT) until further f/u and consolidation to monotherapy. For lower risk patients <4, one can consider ASA 81mg, clopidogrel 75mg daily OR dipyramidole 200mg BID. For patients with new-onset Afib, initiation of oral anticoagulants are likely warranted (warfarin, DOACs). If ED-available, use of DWI-MRI can be useful to identify micro-bleeds that may influence use of antithrombotic agents. For patients with severe HTN, then starting/advancing anti-hypertensive therapy may be necessary to control future risk. Similarly, severe hyperglycemia for diabetics (with high HbA1C levels) may warrant immediate intervention, and/or admission to hospital for risk factor control. Initiation of statins for hyperlipidemia is less likely an urgent ED intervention, and could be deferred for outpt f/u discussion.

Simple counselling for behaviour/lifestyle modification should include the benefits of physical exercise, healthy diets (Mediterranean, DASH), limiting alcohol intake and smoking cessation.

CLINICAL COMMENTARY: TIAs comprise about 250K visits to US EDs, and can be a strong predictor of subsequent stroke. The 90day risk of stroke can be as high as 17.8%, and almost 50% of these can occur within 2days. Usual vascular risk factors can be applied for risk stratifying ED TIA patients. Unfortunately, Black Americans have 1.4x greater risk of completed early stroke than Caucasians, and men have a substantially lower rate of TIA/stroke than women (suggesting common inequities in ED vascular care).

Benefits of Recommendations: This document provides guidance for key elements of ED management of TIA, including lab investigations, imaging options, risk stratification scores, and disposition decision-making. Suggestions can be adopted/adapted into ED workplaces, and operationalized quite readily.

Harms/Adverse Effects of Recommendations: It is not clear how to advance antithrombotic Rx in patients who are already on single-agents and are still experiencing TIA symptoms (treatment failures?).

Facilitators of Uptake:

1) Useful tables for TIA vs mimics, imaging test performance, and risk stratification scores are included.
2) A care pathway is included in the Figure, which can be adopted/adapted for ED TIA implementation.
3) Creation of regional TeleStroke programs can be beneficial in linking under-resourced/ rural ED physicians with regional expert stroke supports, especially for those patients

presenting with acute strokes within thrombolytic windows. These programs may also be leveraged for ED TIA risk stratification and expedited admission vs. follow-up care.

Barriers to Uptake:

1) Access to DWI-MRI or acute phase vascular imaging may be problematic in ED settings.
2) Various ABCD scores have limitations for ED settings. $ABCD^2$ does not account for "posterior" circulation symptoms (ataxia, hemianopsia, dysmetria), TIA mechanisms, recurrent/crescendo TIAs, nor presence of large vessel disease. The latter three have higher risk of recurrent stroke or neurologic worsening. Lack of access to carotid imaging or advanced brain imaging preclude the use of $ABCD^3$ or $ABCD^3$-I scores.
3) Extensive counselling/behaviour modification for lifestyle change may be inaccessible in most ED settings; ideally this would be available in outpt clinic settings and with primary care providers.
4) Under-served/racialized/rural patients may have specific social determinant barriers that need to be addressed in order to provide equitable and rapid ED TIA care

Prior Guideline Recommendations/Relevant Evidence: A comprehensive review by Perry *et al* (CMAJ 2022; doi: 10.1503/cmaj.220344) make similar recommendations for ED TIA work-up, risk stratification and disposition decisions. Salient points are listed as follows:

1) TIA and minor stroke are less likely to be diagnosed in women, even though there are similar cerebral ischemia rates with men.
2) These authors (from Ottawa) recommend the use of the **Canadian TIA Score**, derived/validated to predict 7d stroke risk as Low (<1%), Moderate (1-5%) or High (>5%); available online free calculator at https://emottawablog.com/canadian-tia-score/, and listed on MedCalc. Scoring tool summarized in figure below.
3) The Canadian Stroke Best Practices Guidelines call for **early CT head** for all patients with TIA/early stroke to assess stroke mimics; any abnormal findings can increase the subsequent 3mo stroke risk considerably. They also report that patients with transient resolved symptoms may still have abnormal MRI findings (13.5%); it is not clear, however, what impact widespread MRI would have on management changes, subsequent strokes, ED over-crowding and resource costs (likely last two would be much higher).
4) After disposition for expedited neuro/stroke clinic follow-up, decisions re: Holter monitoring, echocardiography and vascular imaging can be made later.
5) Secondary prevention with **ASA 81mg (Low risk) or DAPT (Mod/High risk) is warranted** for **at least 21days** until further outpt follow-up visits. **Give a loading dose in ED prior to discharge (ASA 160mg or clopidogrel 300-600mg)**. Substituting ticagrelor for clopidogrel is also reasonable. **Patients with AFib on ECG may also warrant starting a DOAC** (assuming no active bleeding on CT head); 66% risk reduction for future stroke. HTN control (optimal target BP unknown?) is important, as uncontrolled HTN contributes 40% to stroke burden. All other recommendations (statins, behaviour/lifestyle modification) are congruent with those presented in this AHA Statement.

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***Conflicts of Interested*:** Reported; fully disclosed for writers/reviewers (pg e10).

Methodological Threats to Validity: *This Best Practice Statement (BPS) does not meet basic requirements for traditional clinical practice guidelines (eg. focused questions, robust evidence*

reviews/ quality assessments, formulation of recommendations). Nonetheless, this document summarizes best current practices for ED TIA care.

Table 1. Factors Suggestive of TIA Versus TIA Mimic

Factors	TIA	TIA mimic
Demographics	Older age	Younger patient without vascular risk factors
Medical history	Presence of vascular risk factors (hypertension, diabetes, coronary artery disease, peripheral artery disease, smoking, obesity, hyperlipidemia, atrial fibrillation, previous stroke, obstructive sleep apnea)	History of epilepsy, migraines, brain tumor
Symptomatology	Abrupt onset Maximal symptoms at onset Duration typically <60 min Preserved mentation Localizing/focal neurological symptoms corresponding to a vascular territory: dysarthria/aphasia, facial droop, hemiparesis, hemibody numbness Dizziness paired with cranial neuropathies, vision loss/diplopia, difficulty with coordination or gait/truncal ataxia, severe nausea/vomiting may suggest posterior circulation process Hypertensive on presentation Headache with ptosis and miosis might indicate dissection	Symptoms that spread/march from site of onset might suggest seizure Altered mentation Migraine headache Presence of signs or symptoms suggesting an alternative diagnosis (ie, positive visual phenomena, seizure-like activity, positional vertigo without localizing/focal symptoms)

This table is meant as a guide to approaching a patient with neurological symptoms and should not be the sole determinant of ultimate diagnosis. Patient-specific factors must also be considered.

Table 2. Advantages and Limitations of Noninvasive Techniques Available to Assess Patients With TIA[22,23]

Noninvasive techniques	Sensitivity for 50%–69% carotid stenosis, %	Specificity for 50%–69% carotid stenosis, %	Sensitivity for 70%–99% carotid stenosis, %	Specificity for 70%–99% carotid stenosis, %	Advantages	Limitations	Considerations
Doppler ultrasonography	36	91	89	84	Low cost No intravenous contrast	Operator variability Results might be affected by patient body habitus or vessel anatomy Only assesses cervical vessels Insensitive for dissection Not available in the emergency department, but potentially available in observation units	
Computed tomography angiography	67	79	87	95	Widely available as a STAT scan in emergency departments Can be performed simultaneously with noncontrast head computed tomography Can assess cervical and intracranial vessels Sensitive for cervical and intracranial atherosclerosis, dissection, other vasculopathies	Requires intravenous contrast Radiation exposure Limitations in interpretation in the setting of significant calcification	Should be ordered as computed tomography angiography head and neck
Magnetic resonance angiography	77	97	88	84	Can be performed simultaneously with magnetic resonance imaging of brain Can assess cervical and intracranial vessels Sensitive for dissection No radiation Can be performed without contrast	Contraindicated in some patients with implants and devices Costly compared with computed tomography and Doppler ultrasonography Longer time to schedule, perform, can rarely perform acutely May overestimate	Should be ordered as he and neck Contrast-enhanced magr resonance angiography a for higher-quality images less motion artifact Food and Drug Administ tion warning on gadoliniu because of retention in th body and brain, especiall the case of repeated inje tions

Table 3. Comparison of ABCD², ABCD³, and ABCD³-I Scores

Components	ABCD² score	ABCD³ score	ABCD³-I score	ABCD³-I (d, c/i) score
Risk factor				
Age ≥60 y	1	1	1	1
Blood pressure ≥140/90 mm Hg	1	1	1	1
Diabetes	1	1	1	1
Clinical features				
Unilateral weakness	2	2	2	2
Language disturbance without weakness	1	1	1	1
Symptom duration, min				
≥60	2	2	2	2
10–59	1	1	1	1
<10	0	0	0	0
>10	N/A	N/A	0	0
Dual transient ischemic attack	N/A	2	2	2
Imaging				
Ipsilateral ≥50% stenosis of internal carotid artery	N/A	N/A	2	N/A
Ipsilateral ≥50% stenosis of internal carotid artery and major cerebral artery	N/A	N/A	N/A	2
Acute diffusion-weighted imaging hyperintensity	N/A	N/A	2	2
Total points	0–7	0–9	0–13	0–13
ABCD² score	2-d risk (%)	7-d risk (%)	90-d risk (%)	
Low (0–3)	1.0	1.2	3.1	
Moderate (4–5)	4.1	5.9	9.8	
High (6–7)	8.1	11.7	17.8	

ABCD² indicates age/blood pressure/clinical features of transient ischemic attack/duration/diabetes score; ABCD³, ABCD² plus Dual TIA; c, carotid stenosis; d, diffusion-weighted image; I, imaging; i, intracranial stenosis; and N/A, not applicable.

Table 4. Secondary Prevention Checklist for Patients With Suspected Transient Ischemic Attack[17]

Care component	ABCD² <4 (low risk)	ABCD² ≥4* (high risk)	ABCD² ≥6 and symptomatic ipsilateral intracranial stenosis*
Antiplatelet (should be started within 12–24 h of symptom onset)	Aspirin 50–325 mg daily OR Clopidogrel 75 mg daily OR Aspirin 25 mg/extended release dipyridamole 200 mg twice daily	Aspirin 81 mg plus clopidogrel 75 mg daily for 21–90 d† THEN transition to single therapy	Aspirin 81 mg plus clopidogrel 75 mg daily for 21–90 d OR Ticagrelor 180 mg load followed by 90 mg twice daily plus aspirin 75–100 mg daily for 30 d‡ THEN transition to single therapy
Antihypertensives (long-term goal blood pressure <130/80 mm Hg)	Angiotensin-converting enzyme inhibitor, angiotensin II receptor blockers, thiazide diuretic. Calcium channel blockers can be considered for patients who need additional options.		
Anticoagulation (for patients with atrial fibrillation or other indications§)	Apixaban, dabigatran, edoxaban, rivaroxaban, warfarin		
Lipid lowering (goal low-density lipoprotein cholesterol <70 mg/dL)	3-Hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors (first line), and ezetimibe then PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitor (if needed for very-high-risk patients)		
Nutritionist consult	Encourage a low sodium or Mediterranean diet. For patients with diabetes, start conversation and consider referral to a nutrition specialist.		
Counsel regarding modification of lifestyle factors in an individualized, culturally sensitive manner	Smoking cessation Physical activity Alcohol moderation		
Follow-up appointment	Expedited transient ischemic attack/neurology and primary care clinics		

*When possible, strongly consider hospital admission.

†Based on CHANCE trial (Clopidogrel in High-Risk Patients With Acute Non-Disabling Cerebrovascular Events) and POINT trial (Platelet-Oriented Inhibition in New TIA and Minor Ischemic Stroke) protocols.[33,34]

‡Based on THALES trial (Acute Stroke or Transient Ischaemic Attack Treated With Ticagrelor and ASA for Prevention of Stroke and Death) protocol.[50]

§In patients with moderate to severe mitral stenosis or mechanical heart valve, warfarin is preferred.

ABCD² indicates age/blood pressure/clinical features of transient ischemic attack/duration/diabetes score; and TIA, transient ischemic attack.

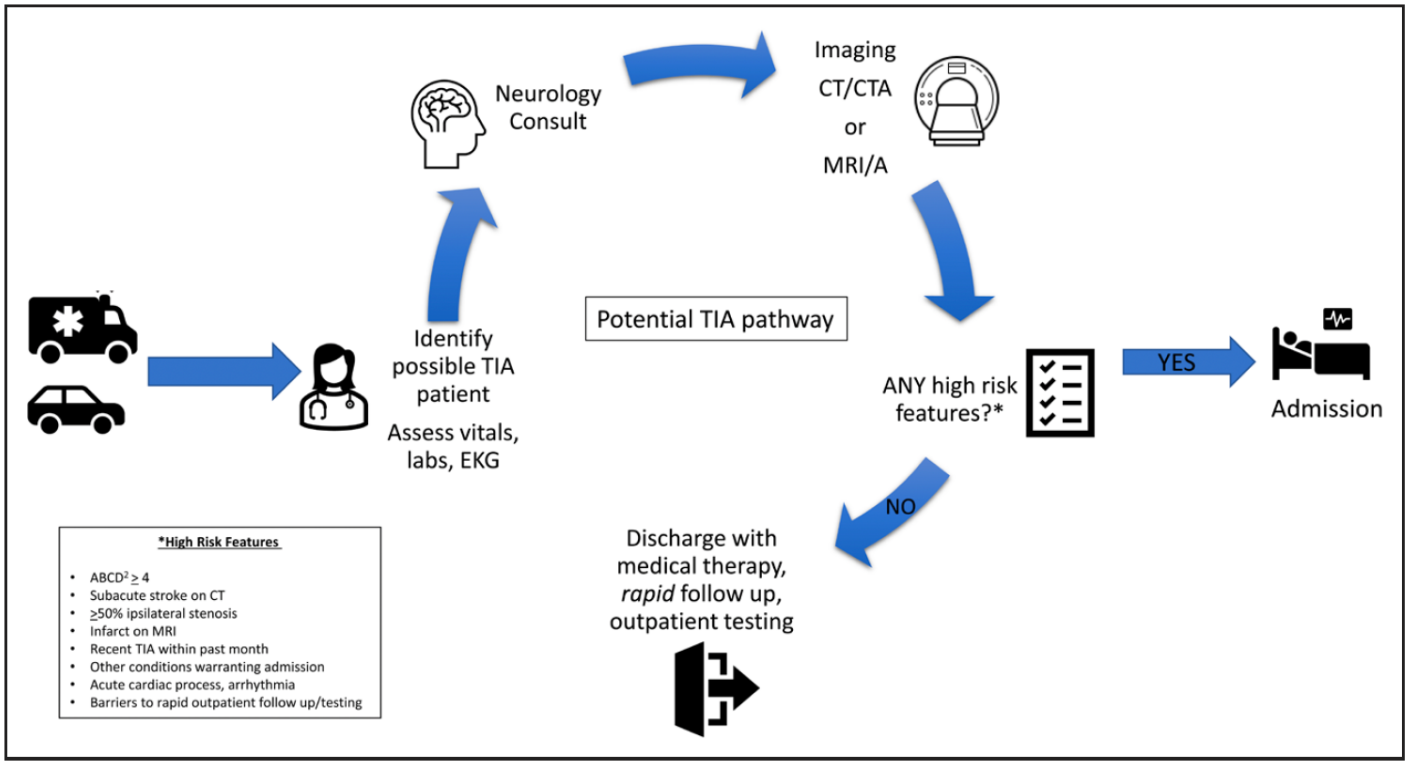

Figure. A potential TIA pathway that incorporates clinical evaluation, imaging, and risk stratification to guide disposition decisions.

Modifications are expected when rapid neurology consultation or MRI are not available. CT indicates computed tomography; CTA, computed tomography angiography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; and TIA, transient ischemic attack.

*High Risk Features

- $ABCD^2 \geq 4$
- Subacute stroke on CT
- ≥50% ipsilateral stenosis
- Infarct on MRI
- Recent TIA within past month
- Other conditions warranting admission
- Acute cardiac process, arrhythmia
- Barriers to rapid outpatient follow up/testing

*May also need to consider patient who live in under-serviced areas, or have transportation issues for outpatient f/u clinic visits that preclude a safe discharge.

Canadian TIA score

OTTAWA

Clinical Findings	Points
First TIA (in lifetime)	2
Symptoms ≥ 10 minutes	2
Past History of **Carotid Stenosis**	2
Already on **Antiplatelet Therapy**	3
History of **Gait Disturbance**	1
History of **Unilateral Weakness**	1
History of **Vertigo**	-3
Initial Triage **Diastolic BP ≥ 110 mmhG**	3
Dysarthria or Aphasia (History or Physical)	1

Investigations in the ED	Points
Atrial Fibrillation on ECG	2
Infarction (new or old) on CT	1
Platelet count ≥ 400 x 10^9/L	2
Glucose ≥ 15 mmol/L	3

Calculated Risk Score

Risk of Stroke or Carotid Revascularization in 7 days

Low Risk (0.5%)	**-3 to 3**
Medium Risk (2.3%)	**4 to 8**
High Risk (5.9%)	**≥ 9**