REVIEW ARTICLE

Rapid Triage and Secondary Prevention in Transient Ischemic Attack: A Narrative Review

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ABSTRACT

Transient ischemic attack (TIA) requires urgent evaluation and treatment to prevent disabling stroke. This review provides an overview of TIA management including clinical presentation, risk stratification, diagnosis, site of care decisions, key diagnostic tests, imaging, acute treatment, hospitalization criteria, medical therapy for secondary prevention, and treatments targeting specific TIA mechanisms. TIA patients face up to a 20% 90-day recurrent stroke risk. Critical management components include prompt specialist assessment, brain/vascular imaging, cardiac monitoring, early antithrombotics, aggressive vascular risk factor control, patient education, and tailored secondary prevention strategies aimed at the underlying TIA mechanism. Expedited TIA protocols facilitate rapid intervention and are associated with reduced stroke rates compared to delayed management. With appropriate systems of care, lower-risk TIA patients can undergo outpatient evaluation. Higher-risk patients often warrant hospitalization for closer monitoring and access to advanced treatments if symptoms recur.
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INTRODUCTION

Transient ischemic attack (TIA) is defined as a transient episode of focal neurological dysfunction caused by brain, spinal cord, or retinal ischemia without acute tissue infarction (1). TIA manifests as one of the several transient neurologic deficits including unilateral limb weakness, speech impairment, vision loss, sensory loss, ataxia, vertigo, or diplopia, typically lasting less than one hour before resolving completely (1–3). An estimated 240,000 people experience a TIA annually in the United States, with incidence increasing with age (4). TIA is associated with up to a 20% 90-day risk of recurrent stroke, with nearly half occurring in the first 48 hours after symptom onset (3–7). Among the strongest predictors of impending stroke following TIA are symptom duration over 60 minutes, weakness or speech impairment, diabetes, age over 60 years, elevated blood pressure at presentation, and dual events within the past week (5,8–10). Compared to stroke, management strategies for secondary stroke prevention may be more effective when initiated early after TIA since there is no residual neurological injury (11). However, early recurrence risk and variable clinical presentation make TIA management challenging (12). This article provides a comprehensive, evidence-based review focused on key diagnostic and therapeutic strategies aimed at improving patient outcomes following TIA.

RISK STRATIFICATION

Clinical risk stratification aids in estimating a patient's short-term risk of stroke following TIA and helps guide management decisions (10,13,14). The ABCD2 score (Age, Blood pressure, Clinical features, Duration of symptoms, Diabetes) is the most widely used, allowing prognostic classification as high risk (6-7 points) or low risk (0-3 points). High risk patients have a 7-day stroke risk of 8% versus 2% in low-risk patients (8,15). However, ABCD2 has limitations including a lack of key predictors like large artery stenosis and atrial fibrillation, thus risk estimates should be considered in context of each patient's clinical scenario (8,15). The presence of diffusion-weighted imaging (DWI) lesions on MRI is a significant predictor of recurrent stroke risk within 90 days, increasing the risk over 6-fold, and should be considered alongside the ABCD2 score for comprehensive risk assessment (19). While clinical scores provide a benchmark, optimal risk assessment

combines clinical judgment with multimodal imaging (20).

SITE OF CARE

The central management decision for TIA patients revolves around determining the optimal site of care. The sense of urgency following TIA presentation relates directly to the subsequent high stroke risk rather than the location of the initial evaluation (21,22). Specialized TIA clinics and emergency department-based observation units permit expedited diagnosis and intervention on par with inpatient units (6,14,23,24). Consequently, lower-risk TIA patients without concerning clinical, laboratory or imaging findings may undergo an initial outpatient evaluation without requiring hospital admission. However, higher-risk patients including those with symptomatic carotid or intracranial stenosis often benefit from hospitalization to enable closer clinical monitoring and rapid administration of stroke treatments if symptoms recur (16,25–27). Telemedicine systems also improve access to vascular neurology expertise which may aid triage decisions (28). Regardless of setting, newly diagnosed TIA should be considered a true neurological emergency warranting focused evaluation and management.

DIAGNOSTIC EVALUATION

Urgent neuroimaging, vascular imaging, cardiac monitoring, and laboratory tests are recommended in all patients with suspected TIA to confirm diagnosis, determine aetiology, exclude mimics, and guide targeted secondary prevention (1,16).

NEUROIMAGING

Brain MRI with DWI within 24 hours of symptom onset is the preferred initial neuroimaging modality for TIA, having greater sensitivity in detecting small ischemic lesions compared to CT (16, 20, 29, 30). DWI-positivity often indicates the presence of an ischemic event rather than a transient ischemic attack (TIA), as it confirms ischemic stroke in nearly 40% of cases regardless of symptom duration (19, 31, 32). This highlights the importance of DWI in accurately diagnosing the underlying condition. (19, 31, 32). DWI lesions also signify higher recurrent stroke risk guiding inpatient triage decisions (16, 19). CT brain is a reasonable alternative when MRI is unavailable and helps exclude haemorrhage and structural lesions (16). However, while CT imaging is commonly used in the initial assessment, it lacks the sensitivity to detect small ischemic lesions that may be present in TIA patients whose symptoms have resolved, underscoring the value of MRI with DWI for a more definitive diagnosis F(33).

VASCULAR IMAGING

Cervical carotid imaging should be performed routinely in suspected TIA patients as part of the initial ED workup or on an expedited basis for those undergoing outpatient evaluation. (1, 16). Carotid duplex ultrasonography, CTA or MRA aid detection of obstructive atherosclerotic lesions amenable to surgical revascularization like carotid endarterectomy (1,34). Intracranial vascular imaging is also recommended when knowledge of a stenosis will influence management. Detecting stenosis can significantly impact clinical decisions, including the need for intervention, such as angioplasty or stenting, and the initiation of more aggressive medical therapy to prevent recurrent ischemic events.. CTA has highest sensitivity for intracranial stenosis while extracranial MRA is preferred for carotid evaluation (35–37).

CARDIAC EVALUATION

Cardiac monitoring, echocardiography and ECG are warranted to exclude cardioembolic sources like AF which warrant anticoagulation over antiplatelets (7, 16). Prolonged rhythm monitoring beyond the initial ECG improves AF detection, which is relevant for TIA patients as atrial fibrillation is a significant risk factor for recurrent ischemic events (38).

LABORATORY TESTING

Basic laboratory tests screen for prothrombotic conditions and reversible causes of neurological deficits like hyponatremia or anaemia. Candidate conditions warranting hypercoagulability testing are outlined in guidelines (39). Point-of-care glucose measurement is mandatory to rule out hypoglycaemic symptoms. Early vascular risk factor assessment with fasting lipids is also recommended. Inflammatory marker measurement aids giant cell arteritis diagnosis in older adults (16).

EARLY SPECIALIST ASSESSMENT

Vascular neurology input, in-person or via telemedicine, improves diagnostic and therapeutic decision-making in TIA patients, whether they undergo inpatient or expedited specialty clinic evaluation. Neurology consultation in the ED is associated with a significant reduction in post-TIA mortality, with studies showing a reduction of up to 50% (40) (40). For centres lacking onsite expertise, developing rapid neurology follow-up protocols is essential given the dynamic post-TIA stroke risk (7).

ACUTE ANTIPLATELET THERAPY

Antiplatelet therapy is recommended immediately following TIA once haemorrhagic transformation is excluded on imaging (7,41,42). Dual antiplatelet therapy (DAPT) with aspirin plus clopidogrel is utilized for early secondary prevention in high stroke risk TIA cohorts (ABCD2 ≥4 points) with number needed to treat as low as 10 to prevent one stroke at 90 days based

on CHANCE and POINT trial data (21, 24, 43, 44). The shorter acting P2Y12 inhibitor ticagrelor is an alternative to clopidogrel (45). High-risk patients on pre-TIA aspirin monotherapy may benefit more from the addition of a second antiplatelet agent, such as clopidogrel, rather than switching to or increasing the dose of aspirin. This dual therapy approach has been shown to reduce the risk of recurrent ischemic events more effectively than aspirin alone. (7). Low risk TIA patients (ABCD2 <4) can be managed with aspirin monotherapy (25). Anticoagulation with a vitamin K antagonist or direct oral anticoagulant is recommended for most patients with TIA and atrial fibrillation (7).

HOSPITALIZATION CRITERIA

Inpatient admission is appropriate for high-risk TIA patients, although criteria vary between protocols. Commonly adopted admission triggers include:

- ABCD2 score ≥4 points (16, 23, 26)
- Age >60 years (16)
- Symptoms >60 minutes (16)
- New DWI lesion on MRI brain (16, 23)
- Primary motor weakness (26)
- Dual TIA events (46)
- Active cardiac arrhythmia [e.g. atrial fibrillation]
- Symptomatic carotid or vertebral stenosis >50% (16, 26)
- Symptomatic intracranial stenosis (25)
- Uncontrolled hypertension or hyperglycaemia
- Inability to complete outpatient workup within 24-48 hours (26)
- Social barriers limiting follow-up access (47)

INTENSIVE MEDICAL THERAPY FOR SECONDARY PREVENTION

All patients with TIA warrant comprehensive medical therapy for secondary stroke prevention. Coordinated, multicomponent interventions addressing glycaemic control, lipid management, blood pressure targets <130/80 mmHg and promoting Mediterranean diet, smoking cessation and exercise reduce recurrence by up to 80% (11,48-50). High intensity statin therapy produces a 16% reduction in stroke and 22% reduction in recurrent events demonstrating vascular protective effects extending beyond pure cholesterol lowering (48,51). Formal stroke education programs also significantly reduce TIA recurrence and mortality by improving patient awareness and medication compliance (27, 28).

TARGETED SECONDARY PREVENTION BASED ON TIA MECHANISM

Once the underlying TIA mechanism is determined, targeted therapies can be implemented (Table I) (7). For suspected large artery atherosclerotic sources, symptomatic extracranial carotid stenosis warrants surgical revascularization with carotid endarterectomy or stenting (29, 34, 52). DAPT is utilized peri-procedurally (53, 54). Symptomatic intracranial stenosis is managed medically with DAPT followed by single antiplatelet therapy (29). Cardioembolic TIA related to atrial fibrillation is treated with oral anticoagulation (7, 29). Cryptogenic TIA warrants antithrombotic therapy tailored to patient risk factors like patent foramen ovale, along with aggressive control of modifiable vascular risks (29). Small vessel lipohyalinotic disease responds best to intensive hypertension treatment and lifestyle changes (7, 29).

Table I: Targeted Secondary Prevention Based on TIA Mechanism

TIA Mechanism	Secondary Prevention Strategy
Large Artery Atherosclerosis	- Carotid endarterectomy or stenting for symptomatic extracranial carotid stenosis - Dual antiplatelet therapy (DAPT) peri-procedurally
Intracranial Atherosclerosis	- Initial DAPT followed by single antiplatelet therapy - Intensive risk factors control
Cardioembolic (e.g., Atrial Fibrillation)	- Oral anticoagulation (e.g., warfarin, DOACs) based on CHA2DS2-VASc score
Cryptogenic TIA	- Antithrombotic therapy tailored to patient risk factors (e.g., patent foramen ovale) - Aggressive vascular risk factor modification
Small Vessel Disease (Lipohyalinosis)	- Intensive risk factors control - Lifestyle modifications (diet, exercise)

EXPEDITED TIA MANAGEMENT PATHWAYS

Specialized TIA clinics and ED observation units successfully facilitate rapid evaluation and treatment initiation following TIA (6, 14). In the Erlanger EXPRESS TIA Pathway, patients undergo protocolized neuroimaging, cardiac telemetry monitoring and serial NIH stroke scale assessments enabling over 70% to be discharged within 23 hours (14). Implementation of the regional Ontario TIA Algorithm allows 75% of TIA patients to be managed as outpatients with low rates of subsequent hospitalization, procedures, or 90-day recurrent stroke (23). Both studies demonstrate feasibility of lower-risk TIA patient evaluation in the ED observation unit setting. Direct admission to stroke units is also appropriate for higher-risk patients requiring hospitalization. Regardless of care location, standardized TIA order sets enhance evidence-based decision making and adherence to guideline recommendations.

CONCLUSION

TIA requires focused specialist assessment given the substantial recurrent stroke risk, highest within 48 hours of symptom onset. All management pathways should emphasize expedited brain and vascular imaging, ECG monitoring, prompt antiplatelet therapy, aggressive control of modifiable vascular risks, patient education, and tailored secondary prevention strategies. With

specialized systems of care, it is feasible to manage low-risk TIA patients in an expedited manner as outpatients. However, hospitalization remains appropriate for higher-risk patients requiring closer clinical monitoring, access to neurological expertise, advanced imaging modalities, intravenous thrombolysis, or endovascular therapy should symptoms recur. Ongoing research aims to clarify uncertainties around neuroimaging priorities, risk scoring methods and optimal duration of combination antiplatelet regimens.

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