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TRANSIENT ISCHEMIC ATTACK COMPREHENSIVE MANAGEMENT AND EVIDENCE BASED APPROACH

Akhmadov Jakhongir Akmal ugli

Department of Preclinical Sciences, Asia International University, Bukhara, Uzbekistan.

E-mail: neurosurgeryjahongir@gmail.com

ORCID: https://orcid.org/0009-0009-5743-3185

Abstract

Background: Transient ischemic attack (TIA) is a neurological emergency because the risk of ischemic stroke is front-loaded into the first hours and days after symptom onset. Contemporary tissue-based diagnosis, rapid neurovascular imaging, and mechanism-directed prevention have transformed care. Objective: To synthesize current evidence for the diagnosis, risk stratification, acute treatment, disposition, and secondary prevention of TIA. Methods: A targeted structured review of PubMed/MEDLINE and major professional guidance was performed for English-language publications from January 2009 through 12 September 2026. Priority was given to American Heart Association/American Stroke Association and European Stroke Organisation guidance, randomized trials, meta-analyses, and large prospective cohorts involving adults with TIA or minor ischemic stroke. Results: TIA should be defined as transient focal neurological dysfunction caused by cerebral, spinal, or retinal ischemia without acute infarction. Clinical scores may support triage but must not replace brain and vascular imaging or specialist assessment. Magnetic resonance imaging with diffusion-weighted sequences is preferred, while computed tomography combined with head and neck angiography is an effective rapid alternative. For eligible high-risk non-cardioembolic TIA, aspirin plus clopidogrel started early and continued for 21 days provides the most favorable balance of ischemic benefit and bleeding risk. Cardioembolic events require anticoagulation; symptomatic carotid stenosis requires expedited revascularization assessment; and all patients need intensive control of blood pressure, lipids, diabetes, smoking, diet, and physical inactivity. Conclusion: The decisive therapeutic principle is not the disappearance of symptoms but the urgency of identifying the mechanism and starting targeted prevention. A 24-hour diagnostic pathway integrating imaging, cardiac evaluation, and specialist review can prevent a substantial proportion of early strokes.

Keywords: transient ischemic attack; ischemic stroke; diffusion-weighted imaging; dual antiplatelet therapy; carotid stenosis; atrial fibrillation; secondary prevention

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1 Introduction

Transient ischemic attack is a brief episode of focal neurological dysfunction produced by cerebral, spinal cord, or retinal ischemia without acute infarction. This tissue-based definition replaced the older rule that symptoms resolving within 24 hours automatically represented TIA, because modern diffusion-weighted magnetic resonance imaging (DWI-MRI) can demonstrate infarction despite rapid clinical recovery [1]. Accordingly, a transient deficit with an acute ischemic lesion is classified as ischemic stroke, even when the patient appears normal at assessment.

The apparent reversibility of TIA can create false reassurance. In emergency-department populations, the 90-day risk of stroke may be as high as 17.8%, and almost half of subsequent strokes occur within 48 hours [2]. However, prognosis is strongly modified by the speed and quality of care. In the EXPRESS study, urgent assessment and immediate preventive treatment reduced the 90-day recurrent stroke rate from 10.3% to 2.1%, an adjusted relative reduction of approximately 80% [5]. Risk also persists after the acute period: a 2025 meta-analysis of 171,068 patients estimated cumulative stroke risks of 5.9% at 1 year, 12.8% at 5 years, and 19.8% at 10 years [23]. TIA is therefore best understood as an unstable neurovascular syndrome and a time-sensitive opportunity for both immediate and sustained prevention.

This review aims to provide a practical, evidence-based framework for clinicians managing suspected TIA. The objectives are to clarify diagnosis and mimics, define the minimum urgent investigation, interpret risk scores appropriately, summarize antithrombotic and revascularization evidence, and integrate mechanism-specific secondary prevention into a coherent pathway suitable for emergency departments, stroke units, and rapid-access TIA clinics.

2 Materials and Methods

2.1 Review design and information sources

A targeted structured narrative review was undertaken. PubMed/MEDLINE and the guideline repositories of the American Heart Association/American Stroke Association (AHA/ASA) and European Stroke Organisation (ESO) were searched for publications available from January 2009 through 12 September 2026. The starting date was chosen to capture the formal tissue-based TIA definition and subsequent changes in imaging and secondary prevention. The search combined the terms transient ischemic attack, minor stroke, diagnosis, diffusion MRI, computed tomography angiography, risk stratification, ABCD2, dual antiplatelet therapy, anticoagulation, atrial fibrillation, carotid stenosis, lipid lowering, and secondary prevention.

2.2 Eligibility and evidence prioritization

English-language guidance documents, randomized controlled trials, pooled analyses, meta-analyses, and large prospective cohorts involving adults with TIA or minor ischemic

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stroke were prioritized. Studies were selected when they informed immediate diagnosis, early stroke risk, antithrombotic therapy, cardiac monitoring, revascularization, or long-term vascular risk reduction. Pediatric studies, isolated case reports, non-ischemic transient neurological syndromes, and studies focused exclusively on established disabling stroke were excluded. Because the objective was clinical synthesis rather than estimation of a single pooled effect, formal meta-analysis and certainty regrading were not performed.

2.3 Data synthesis

Evidence was organized into five clinically sequential domains: recognition and differential diagnosis; urgent brain, vascular, cardiac, and laboratory assessment; early risk stratification and disposition; immediate antithrombotic treatment; and mechanism-directed secondary prevention. Recommendations were interpreted according to the populations enrolled in the supporting trials. Absolute event rates are reported where they materially affect bedside decisions.

3 Results

3.1 Clinical recognition and diagnostic boundaries

The diagnosis remains clinical. The strongest pattern is a sudden-onset, maximal-at-onset, anatomically plausible focal deficit. Typical anterior-circulation manifestations include unilateral weakness or numbness, aphasia, dysarthria, and monocular visual loss. Posterior-circulation ischemia may produce diplopia, dysarthria, dysphagia, unilateral or bilateral weakness, crossed sensory findings, severe gait ataxia, or a consistent combination of vertigo with other focal brainstem signs. Isolated loss of consciousness, generalized weakness, confusion, incontinence, or nonfocal dizziness is less typical and should prompt consideration of an alternative diagnosis.

Negative symptoms, such as loss of power, sensation, language, or vision, favor ischemia. Positive spreading symptoms, such as scintillating visual phenomena or marching paresthesia, favor migraine aura; rhythmic jerking, forced head or eye deviation, automatisms, or postictal confusion favor seizure. Nevertheless, elderly patients and those with vascular risk factors can have atypical events, and a mimic cannot be declared solely from one clinical feature. A witness history, precise time course, prior stereotyped episodes, medication review, and bedside glucose measurement are essential.

Table 1. Clinical features that influence the probability of TIA

Onset	Abrupt; deficit maximal within seconds or minutes	Gradual spread over minutes; fluctuating symptoms linked to position, anxiety, or hyperventilation
Symptom type	Loss of motor, sensory, language, or visual function	Positive visual phenomena, marching paresthesia, rhythmic movements, isolated confusion

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Anatomic pattern	Deficit fits a cerebral arterial, retinal, or brainstem territory	Multiple symptoms without a coherent vascular territory
Associated clues	Vascular risk factors, atrial fibrillation, carotid bruit, recent vascular procedure	Headache with prior aura, postictal state, hypoglycemia, syncope prodrome
Recurrence	Crescendo focal episodes may indicate unstable arterial disease	Highly stereotyped frequent events may suggest seizure, migraine, or functional disorder, but recurrent TIAs remain possible

3.2 Immediate stabilization and exclusion of ongoing stroke

Any patient whose deficit has not fully resolved should enter an acute stroke pathway. Airway, breathing, circulation, oxygen saturation, temperature, capillary glucose, blood pressure, last-known-well time, neurological examination, and National Institutes of Health Stroke Scale score should be documented without delaying imaging. Persistent disabling symptoms may represent acute ischemic stroke and require immediate assessment for intravenous thrombolysis and endovascular thrombectomy. Thrombolysis is not indicated for a completely resolved event, but waiting for spontaneous resolution when a disabling deficit persists can forfeit reperfusion eligibility.

Marked blood pressure elevation is common. In a fully resolved TIA, abrupt empiric reduction is generally avoided until intracranial hemorrhage, large-vessel stenosis, and hemodynamic mechanisms have been considered. Hypotension, hypoxemia, fever, and hypoglycemia should be corrected. Antithrombotic treatment is initiated after hemorrhage and major contraindications have been excluded.

3.3 Urgent diagnostic investigation

Brain imaging should be obtained urgently, preferably within 24 hours. MRI with DWI, fluid-attenuated inversion recovery, gradient-echo or susceptibility-weighted imaging, and vascular sequences is preferred because it identifies small acute infarcts and alternative diagnoses. When MRI is unavailable, delayed, or contraindicated, noncontrast computed tomography (CT) should be combined with CT angiography (CTA) of the head and neck whenever feasible. A normal noncontrast CT does not exclude ischemia; its immediate value is primarily the detection of hemorrhage, mass lesions, and established infarction.

Vascular imaging is not optional in a complete TIA evaluation. CTA or magnetic resonance angiography should assess extracranial and intracranial arteries. Carotid duplex ultrasonography is useful for screening and surveillance but an apparent stenosis of 50% or greater should generally be confirmed with CTA or magnetic resonance angiography before revascularization planning [3]. Early detection of symptomatic carotid or intracranial stenosis changes both disposition and treatment.

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A 12-lead electrocardiogram should be performed promptly, and telemetry is appropriate when the patient is admitted or observed. If no cause is found and a cardioembolic mechanism remains possible, extended ambulatory monitoring increases detection of paroxysmal atrial fibrillation. In EMBRACE, 30-day event-triggered monitoring detected atrial fibrillation more often than repeat 24-hour monitoring in patients with cryptogenic stroke or TIA [15]. Transthoracic echocardiography is reasonable when structural heart disease is suspected; transesophageal echocardiography is reserved for questions such as left atrial appendage thrombus, valvular pathology, aortic arch atheroma, or selected patent foramen ovale evaluation.

Table 2. Minimum urgent diagnostic package for suspected TIA

Focused clinical assessment	Confirm focal syndrome; establish last-known-well; identify ongoing deficit, mimics, bleeding risk, and mechanism clues	Immediately
Capillary glucose and vital signs	Identify reversible mimic or physiological instability	Immediately
Brain imaging	MRI with DWI preferred; noncontrast CT if MRI is not immediately available	As soon as possible, ideally within 24 hours
Head and neck arterial imaging	Detect carotid, vertebral, intracranial stenosis, occlusion, or dissection	During initial assessment
ECG and rhythm monitoring	Detect atrial fibrillation, ischemia, or conduction disease	ECG immediately; telemetry or ambulatory monitoring according to risk
Laboratory studies	Complete blood count, electrolytes, renal function, glucose/HbA1c, lipid profile; coagulation tests when indicated	Initial visit
Echocardiography	Identify selected structural or embolic cardiac sources	Urgent or early outpatient based on suspected mechanism

3.4 Risk stratification and disposition

The ABCD2 score assigns points for age, blood pressure, clinical features, symptom duration, and diabetes. It is simple and may communicate risk, but it has limited sensitivity for dangerous mechanisms. Patients with low scores may still have atrial fibrillation, DWI-positive infarction, or critical carotid stenosis. ESO guidance therefore recommends against using ABCD2 alone to determine urgency, admission, or treatment [3]. ABCD3-I improves discrimination by adding recurrent events and imaging findings, but it still supplements rather than replaces specialist evaluation [19,20].

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Features favoring hospital admission or monitored observation include an ongoing or recurrent deficit, crescendo TIAs, DWI-positive lesions, symptomatic extracranial or intracranial stenosis, atrial fibrillation or another major cardiac source, severe uncontrolled hypertension, metabolic instability, high bleeding risk during treatment initiation, diagnostic uncertainty requiring serial examination, and inability to complete imaging and specialist assessment rapidly as an outpatient. A rapid-access TIA clinic is safe only when the full evaluation and treatment can be delivered reliably within 24 hours and the patient can return immediately if symptoms recur.

3.5 Immediate antithrombotic therapy

For suspected non-cardioembolic TIA, aspirin should be given promptly after intracranial hemorrhage has been excluded and no contraindication exists. A loading dose of 160-325 mg is commonly used, followed by a maintenance dose of 75-100 mg daily. If aspirin is contraindicated, clopidogrel monotherapy is a reasonable alternative. Empiric anticoagulation is inappropriate before a cardioembolic indication is established.

Short-course dual antiplatelet therapy (DAPT) is the principal evidence-based escalation for high-risk non-cardioembolic TIA. CHANCE randomized 5,170 patients within 24 hours and found 90-day stroke rates of 8.2% with clopidogrel plus aspirin versus 11.7% with aspirin alone, without an increase in moderate or severe hemorrhage [8]. POINT confirmed fewer major ischemic events with DAPT in a broader international population but demonstrated more major hemorrhage over 90 days [9]. A pooled time-course analysis showed that most net benefit occurs in the first 21 days [10]. Therefore, aspirin plus clopidogrel should usually be started within 12-24 hours in eligible patients and continued for 21 days, followed by single antiplatelet therapy. Routine prolonged DAPT is avoided because bleeding risk accumulates while incremental ischemic benefit declines.

Ticagrelor plus aspirin for 30 days is an alternative in selected patients, including some with severe high-risk presentations, clopidogrel nonresponse, or CYP2C19 loss-of-function alleles. In THALES, stroke or death occurred in 5.5% with ticagrelor-aspirin and 6.6% with aspirin alone, while severe bleeding increased from 0.1% to 0.5% [11]. This regimen requires careful assessment of bleeding risk, cost, adherence, dyspnea, and drug interactions. It is not a routine substitute for clopidogrel-based DAPT in every TIA.

Table 3. Landmark evidence for early antiplatelet treatment

CHANCE [8]	Minor stroke or high-risk TIA within 24 h; clopidogrel for 90 d plus aspirin for 21 d	Stroke at 90 d: 8.2% vs 11.7%; HR 0.68	Moderate/severe hemorrhage 0.3% in both groups; conducted in China
POINT [9]	Minor stroke or high-risk TIA within 12 h; clopidogrel plus aspirin for 90 d	Major ischemic events: 5.0% vs 6.5%; HR 0.75	Major hemorrhage: 0.9% vs 0.4%; supports short rather than

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			prolonged DAPT
Pooled CHANCE/ POINT [10]	Individual-patient pooled analysis	Benefit concentrated mainly in first 21 d	A 21-d course maximizes net clinical benefit for most eligible patients
THALES [11]	NIHSS 5 or less or high-risk TIA within 24 h; ticagrelor plus aspirin for 30 d	Stroke/death: 5.5% vs 6.6%; HR 0.83	Severe bleeding: 0.5% vs 0.1%; use selectively

3.6 Mechanism-directed prevention

3.6.1 Cardioembolic TIA

When atrial fibrillation is established, oral anticoagulation is substantially more effective than antiplatelet therapy for prevention of cardioembolic stroke. A direct oral anticoagulant is preferred for most patients with nonvalvular atrial fibrillation; warfarin remains indicated for mechanical heart valves and moderate-to-severe rheumatic mitral stenosis. After a true TIA with no infarction or hemorrhage on imaging, anticoagulation can often begin immediately or within 24 hours, provided bleeding risk and diagnostic certainty are acceptable. The timing should be individualized if imaging shows infarction, hemorrhagic transformation, uncontrolled hypertension, or another bleeding hazard. Antiplatelet therapy is not routinely added to anticoagulation unless there is a separate compelling indication.

If the initial cardiac evaluation is negative, prolonged rhythm monitoring should be matched to the probability that occult atrial fibrillation would change management. Both 30-day external monitoring and insertable cardiac monitors have higher diagnostic yield than short conventional monitoring [15,16]. By contrast, anticoagulation should not be prescribed empirically for an embolic event of undetermined source; NAVIGATE ESUS found rivaroxaban did not reduce recurrent stroke compared with aspirin and increased bleeding [17].

3.6.2 Large-artery atherosclerosis

Patients with ipsilateral symptomatic carotid stenosis require urgent vascular-neurology and surgical assessment. Carotid endarterectomy provides substantial benefit for 70-99% stenosis without near-occlusion and a smaller, patient-dependent benefit for 50-69% stenosis [13]. Benefit is greatest when surgery is performed early, ideally within 14 days of the last retinal or cerebral ischemic event [14]. Age, sex, comorbidity, anatomy, procedural risk, and local expertise influence the choice between endarterectomy and stenting. Revascularization does not replace intensive medical therapy.

For symptomatic severe intracranial atherosclerotic stenosis, aggressive medical therapy is first line. This includes antiplatelet therapy, high-intensity lipid lowering, blood pressure control after the hyperacute phase, smoking cessation, and lifestyle modification.

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Selected patients with recent stroke or TIA caused by 70-99% intracranial stenosis may receive aspirin plus clopidogrel for up to 90 days under specialist supervision. Intracranial stenting is not routine initial treatment.

3.6.3 Other etiologies

Cervical artery dissection should be considered in younger patients, after neck trauma or manipulation, or when pain, Horner syndrome, cranial neuropathy, or posterior-circulation symptoms accompany the event. Antiplatelet therapy or anticoagulation may be used for a limited course, commonly three to six months, with selection individualized to anatomy, infarct burden, bleeding risk, and specialist judgment. Giant cell arteritis must be excluded urgently in older patients with transient monocular blindness, headache, jaw claudication, constitutional symptoms, or elevated inflammatory markers. Hypercoagulable and autoimmune testing should be targeted rather than routine, guided by age, history, venous thrombosis, pregnancy morbidity, malignancy, systemic features, and absence of conventional causes.

3.7 Intensive vascular risk reduction

Secondary prevention begins at the index encounter. For most patients with hypertension, the long-term target is below 130/80 mm Hg, while the regimen and pace of reduction should be individualized for frailty, orthostatic symptoms, critical arterial stenosis, and hemodynamic events [4]. Patients with atherosclerotic TIA should receive high-intensity statin therapy unless contraindicated. An LDL cholesterol target below 70 mg/dL (1.8 mmol/L) is supported by the Treat Stroke to Target trial; ezetimibe and, in very-high-risk patients, a PCSK9 inhibitor can be added when maximally tolerated statin therapy is insufficient [4,18].

Diabetes management should integrate glucose control with therapies that reduce cardiovascular risk; for many adults, a glycated hemoglobin target near 7% is appropriate, but age, hypoglycemia risk, comorbidity, and life expectancy require individualization. Smoking cessation should combine brief clinician advice with behavioral and pharmacological support. A Mediterranean-style or DASH-type dietary pattern, sodium reduction, weight management, regular aerobic and resistance activity, moderation of alcohol, and evaluation for obstructive sleep apnea support risk reduction. Medication reconciliation and adherence assessment are essential because recurrence often reflects untreated mechanism or failure to sustain therapy.

Table 4. Etiology-specific prevention after TIA

Non-cardioembolic high-risk TIA	Early aspirin plus clopidogrel for 21 d, then single antiplatelet therapy	Exclude hemorrhage; assess bleeding risk; avoid routine long-term DAPT
Atrial fibrillation	Direct oral anticoagulant for most nonvalvular AF;	Do not routinely

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	warfarin for mechanical valves or rheumatic mitral stenosis	combine anticoagulant and antiplatelet therapy
Symptomatic carotid stenosis	Expedited carotid endarterectomy evaluation; ideally treat within 14 d when indicated	Confirm stenosis and consider procedural risk and anatomy
Severe intracranial stenosis	Aggressive medical treatment; selected patients may receive DAPT up to 90 d	Intracranial stenting is not first-line routine therapy
Cervical artery dissection	Individualized antiplatelet or anticoagulant therapy, commonly 3-6 mo	Consider intracranial extension, infarct burden, and bleeding risk
Atherosclerotic risk	High-intensity statin; target LDL-C below 70 mg/dL; BP generally below 130/80 mm Hg	Individualize in frailty, orthostasis, and hemodynamic disease

3.8 Follow-up and safety-netting

Before discharge, the patient and family should receive a clear diagnosis or working diagnosis, medication plan, pending-test plan, and explicit instructions to call emergency services for recurrent weakness, numbness, speech disturbance, visual loss, imbalance with focal signs, or any persistent neurological symptom. Driving and occupational restrictions should follow local law and the functional consequences of the event. Follow-up should occur within days, not weeks, and should confirm imaging results, determine the final mechanism, adjust antithrombotic therapy, review blood pressure and lipid treatment, assess adverse effects and adherence, and arrange carotid or cardiac interventions when indicated.

4 Discussion

The evidence supports a shift from a symptom-duration model to a tissue- and mechanism-based model of TIA care. The practical consequence is that symptom resolution does not end the emergency. DWI-positive lesions, symptomatic arterial stenosis, atrial fibrillation, and recurrent focal events identify biologically unstable disease that is incompletely captured by a bedside score. The most reliable pathway combines immediate clinical assessment, brain imaging, head and neck vascular imaging, cardiac evaluation, and treatment initiation within 24 hours.

DAPT illustrates the importance of matching treatment intensity to both timing and mechanism. CHANCE and POINT established that platelet inhibition should be intensified early in eligible high-risk non-cardioembolic events, but the pooled time course shows why treatment should usually be de-escalated after 21 days. THALES offers another option, but its modest absolute benefit and higher severe bleeding rate argue for selective rather than universal ticagrelor use. None of these regimens should delay anticoagulation for

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confirmed atrial fibrillation or carotid revascularization for symptomatic high-grade stenosis.

Service design is as important as drug selection. EXPRESS demonstrated that rapid access to ordinary evidence-based interventions produced a larger clinical effect than many individual medications. Hospitals without a dedicated TIA clinic should establish a protocol that guarantees same-day imaging, stroke-specialist review, immediate antithrombotic treatment, and reliable completion of cardiac and vascular studies. Where these elements cannot be delivered promptly as an outpatient, observation or admission is safer.

Several uncertainties remain. TIA is a clinical diagnosis without a single definitive biomarker, DWI may be negative despite genuine ischemia, and the optimal intensity of rhythm monitoring varies by patient. Evidence for DAPT is strongest in trial-defined high-risk TIA and minor stroke populations and should not be indiscriminately extrapolated to low-risk or uncertain events. Optimal management of unusual mechanisms, recurrent events despite therapy, and patients with simultaneous high ischemic and bleeding risk requires specialist judgment.

4.1 Limitations

This structured narrative review did not use a preregistered protocol, duplicate screening, or formal meta-analysis and may therefore be affected by selection bias. Recommendations were synthesized across healthcare systems with different access to MRI, vascular imaging, stroke specialists, and revascularization. Trial populations may underrepresent frail older adults, patients with severe renal or hepatic disease, and those at high bleeding risk. Drug doses and service pathways must be adapted to national formularies, regulatory approvals, and local stroke systems.

5 Conclusion

TIA is a preventable-stroke emergency. The standard of care is urgent, mechanism-oriented evaluation rather than reassurance after symptom resolution. MRI with DWI is preferred, but rapid CT and CTA provide an effective alternative when MRI is not immediately available. Risk scores support communication but should never replace imaging and specialist assessment. Early 21-day aspirin-clopidogrel therapy is appropriate for selected high-risk non-cardioembolic TIA, whereas atrial fibrillation requires anticoagulation and symptomatic carotid stenosis requires expedited revascularization assessment. Sustained control of blood pressure, lipids, diabetes, smoking, diet, and physical activity completes secondary prevention. A coordinated 24-hour pathway offers the greatest opportunity to reduce early recurrent stroke and long-term disability.

Declarations

Ethics approval: Not applicable. This article is a review of previously published evidence and did not involve human participants or identifiable patient data.

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