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Clinical Assessment and Emergency Management of Transient Ischemic Attack

Faleh Turki Hammad Alanazi [(1)] , Hussain Ali Jubran Shafei [(2)] , Omar Mahmoud Abduljawad [(3)] , Nooh Thawab Almotiri [(4)] , Abdullah Hussain Ghazi Alghazi [(5)] , Ibrahim Mohammed Ibrahim Otudi [(5)] , Ayoub Ali Mohamed Aldarbi [(6)] , Faisal Saud Almutaiti [(7)] , Eman Mohammed Almadan [(8)] , Anfal Abdulrazaq Alabdulwahed [(8)] , Safer Atif Alqahtani [(8)] , Itidal Zaid Hassan Alnahari [(9)] , Manal Ali Hassan Makin [(9)]

(1) Red Crescent Authority – Tabuk, Saudi Arabia,
(2) Emergency Management, Ministry of Health, Saudi Arabia,
(3) Badr General Hospital, Ministry of Health, Saudi Arabia,
(4) Emergency and Ambulance Services, Ministry of Health, Saudi Arabia,
(5) Emergency Coordination and Care Department, Jazan Health Cluster, Ministry of Health, Saudi Arabia,
(6) Emergency Coordination and Care Department, Jazan Health Cluster, Saudi Arabia,
(7) Wathilan Hospital – Ministry of Health, Saudi Arabia,
(8) Imam Abdulrahman Al Faisal Hospital Riyadh, Ministry of Health, Saudi Arabia,
(9) King Fahad Hospital, Ministry of Health, Saudi Arabia

Abstract

Background: Transient ischemic attack (TIA) is a neurological emergency characterized by temporary focal ischemia without permanent infarction. It serves as a critical warning for impending stroke, with the highest risk occurring within 48 hours post-event.

Aim: To review the clinical assessment, emergency management, and preventive strategies for TIA, emphasizing early recognition and intervention to reduce stroke risk.

Methods: This review synthesizes current evidence and guidelines on TIA evaluation and management, including neuroimaging, vascular and cardiac assessment, laboratory investigations, and risk stratification using the ABCD² score. It also examines therapeutic approaches such as antiplatelet therapy, anticoagulation, and lifestyle modification.

Results: Early and comprehensive evaluation significantly reduces stroke risk. MRI with diffusion-weighted imaging is preferred for diagnosis, while vascular imaging and cardiac monitoring identify treatable sources. Dual antiplatelet therapy for 21–30 days post-TIA, followed by monotherapy, is effective in non-cardioembolic cases. Carotid endarterectomy benefits patients with high-grade stenosis, and anticoagulation is indicated for cardioembolic TIA. Integrated strategies combining pharmacologic therapy and lifestyle changes can reduce recurrent stroke risk by up to 80%.

Conclusion: TIA requires urgent, multidisciplinary management. Rapid diagnosis, risk stratification, and targeted interventions are essential to prevent subsequent ischemic events. Evidence supports a multifaceted approach involving imaging, pharmacologic therapy, and risk factor modification to optimize outcomes.

Keywords: Transient ischemic attack, stroke prevention, neuroimaging, ABCD² score, antiplatelet therapy, carotid endarterectomy, anticoagulation.

Introduction

A transient ischemic attack (TIA) represents a neurological emergency that demands prompt recognition and intervention. TIAs are defined as brief episodes of neurological dysfunction resulting from focal ischemia in the brain, spinal cord, or retina without causing acute infarction or permanent tissue injury. The conceptual understanding of TIA has shifted from a purely time-based framework, traditionally defined as lasting less than 24 hours, to a tissue-based definition emphasizing the absence of irreversible brain damage. Most TIAs resolve within minutes, rarely exceeding one hour, yet their clinical significance is profound, as they serve as a critical warning sign for an impending ischemic stroke. The risk of stroke following a TIA is particularly high within the first 48 hours, underscoring the need for urgent assessment and intervention.[1] Clinical presentation of TIA typically involves sudden-onset focal neurological deficits that correlate with specific vascular territories. Common manifestations include unilateral weakness or numbness, speech disturbances, visual deficits, and coordination impairments. Differentiating a TIA from other transient neurological events or mimicking conditions, such as seizures, migraine auras, or metabolic disturbances, is essential for accurate diagnosis and management. The hallmark of TIA is its abrupt onset and rapid resolution, often with complete recovery within minutes. Prompt identification allows clinicians to

*Corresponding author e-mail: ftft3303@gmail.com (Faleh Turki Hammad Alanazi).
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implement strategies aimed at preventing subsequent ischemic events, which are otherwise associated with substantial morbidity and mortality.[2] Evaluation of suspected TIA requires a comprehensive, urgent approach combining neuroimaging, vascular studies, and laboratory assessments. Imaging modalities, particularly diffusion-weighted magnetic resonance imaging (MRI), are used to rule out acute infarction and identify high-risk vascular lesions. Laboratory investigations help uncover metabolic or hematologic contributors to cerebrovascular risk, including glucose abnormalities, lipid disorders, and coagulation defects. Risk stratification tools, such as the ABCD² score, enable clinicians to estimate the likelihood of recurrent TIA or early stroke and prioritize the urgency of interventions.[2][3] Therapeutic strategies following a TIA are multifaceted and aimed at reducing the risk of recurrent ischemic events by targeting modifiable vascular risk factors. Immediate interventions include tight blood pressure control, initiation of high-intensity statin therapy, and antiplatelet therapy. Glycemic management, dietary modifications, and structured physical activity are integral components of secondary prevention. Additionally, addressing the underlying etiology, such as carotid artery stenosis, atrial fibrillation, or hypercoagulable states, is crucial for individualized patient care. When these interventions are implemented promptly and comprehensively, studies indicate that the risk of recurrent TIA or ischemic stroke can be reduced by up to 80%, emphasizing the importance of rapid, coordinated, and evidence-based management in emergency settings.[1][2][3]

Etiology

The etiology of transient ischemic attack (TIA) reflects the underlying mechanisms of ischemic stroke, with subtypes classified according to pathophysiological processes. The primary categories include large artery atherothrombosis, cardiac embolism, small vessel disease, cryptogenic TIA, and less common causes such as arterial dissection, vasculitis, or other vascular abnormalities. Large artery atherothrombosis involves atherosclerotic plaque formation in major intracranial or extracranial arteries, which can result in local thrombosis or artery-to-artery embolization, leading to transient cerebral ischemia. Cardioembolic TIAs occur when clots form within the heart, typically in the left atrium, and subsequently embolize to cerebral arteries; atrial fibrillation is the most frequent contributor to this subtype. Small vessel or lacunar TIAs arise from occlusion of penetrating arteries due to lipohyalinosis or arteriolosclerosis, often linked to chronic hypertension, diabetes, and advancing age. Cryptogenic TIAs, also referred to as embolic stroke of undetermined source (ESUS), are characterized by cortical ischemic patterns without an identifiable cardiac or large artery origin. Rare causes include arterial dissections, vasculitis, hypercoagulable states, and other structural or inflammatory vascular pathologies. Multiple risk factors influence the likelihood of TIA occurrence. These factors are both modifiable and nonmodifiable. Hypertension represents the most critical risk factor on both individual and population levels, as elevated blood pressure contributes to arterial wall injury and promotes atherothrombosis and small vessel disease. Other established risk factors include diabetes mellitus, advanced age, smoking, obesity, sedentary lifestyle, excessive alcohol consumption, poor dietary habits, and chronic psychosocial stress. A prior history of TIA or stroke significantly increases the risk of recurrence, highlighting the cumulative nature of cerebrovascular vulnerability. Additionally, the interaction of risk factors is synergistic; for example, the coexistence of diabetes and hypertension markedly elevates cerebrovascular risk. Understanding the etiology of TIA is essential for targeted interventions, as therapy can be directed toward the underlying mechanism, whether it involves anticoagulation for cardioembolism, antiplatelet therapy for large artery disease, or aggressive risk factor modification for small vessel disease.[4][5] The heterogeneity of TIA etiology underscores the need for comprehensive evaluation, including detailed history, physical examination, neuroimaging, cardiac assessment, and vascular studies. Such an approach allows clinicians to categorize TIA accurately, assess risk of recurrent events, and implement personalized preventive strategies. Failure to identify the underlying cause may result in recurrent TIA or progression to a full ischemic stroke, emphasizing the critical importance of early and thorough etiological assessment.

Epidemiology

Estimating the incidence and prevalence of transient ischemic attack presents inherent challenges due to the transient nature of symptoms, frequent underreporting, and the presence of mimicking conditions such as migraine, seizure, or syncope. Nevertheless, epidemiological studies provide insight into population-level burden. In the United States, approximately 500,000 TIAs occur annually, translating to an incidence of roughly 1.1 per 1,000 individuals. The overall prevalence among adults is estimated at around 2%, though rates are higher among populations with preexisting cerebrovascular disease or traditional vascular risk factors. Importantly, individuals with a history of prior stroke demonstrate an elevated prevalence of TIA, reflecting the shared pathophysiology and cumulative risk. Several longitudinal studies have highlighted that a substantial proportion of patients presenting with their first ischemic stroke experienced prior TIA symptoms, often unrecognized or untreated, reinforcing the concept of TIA as a critical warning event. Age, sex, and comorbidity patterns influence epidemiological trends. The incidence of TIA increases with advancing age, reflecting the progressive accumulation of

atherosclerotic burden, arterial stiffness, and atrial arrhythmias. Men generally demonstrate slightly higher incidence rates than women, though this gap narrows in older populations. Coexisting conditions, particularly hypertension, diabetes, dyslipidemia, and atrial fibrillation, significantly elevate the population-attributable risk. Geographic and ethnic variations in incidence are observed, likely due to differences in lifestyle, socioeconomic status, and access to healthcare services. Epidemiologic surveillance emphasizes that early recognition and intervention after TIA could markedly reduce the incidence of subsequent stroke, which remains a major contributor to morbidity, mortality, and healthcare utilization.[4] Accurate epidemiologic assessment relies on standardized definitions and diagnostic criteria, including neuroimaging confirmation. Transitioning from time-based to tissue-based definitions has refined TIA classification, facilitating more precise epidemiologic measurements. Prospective cohort studies incorporating MRI, vascular imaging, and cardiac monitoring yield more reliable estimates than retrospective analyses based solely on clinical evaluation. Understanding the epidemiology of TIA informs public health strategies, resource allocation, and prioritization of preventive measures, including population-wide blood pressure control, lifestyle modification programs, and community education on early recognition of neurologic symptoms.

Pathophysiology

The pathophysiology of transient ischemic attack varies according to the underlying subtype, but the unifying feature is a transient disruption of cerebral blood flow leading to temporary neuronal dysfunction. In large artery atherothrombosis, stenotic lesions or unstable plaques in intracranial or extracranial arteries may result in either hemodynamic compromise distal to the lesion or artery-to-artery embolization. The latter mechanism is more common and is often responsible for cortical TIAs presenting with motor, sensory, or speech deficits localized to specific vascular territories. Small vessel TIAs, also referred to as lacunar, involve occlusion of penetrating arterioles due to lipohyalinosis or arteriolosclerosis. Chronic hypertension, diabetes, and age-related vascular changes contribute to vessel wall thickening and luminal narrowing, producing transient ischemia in subcortical regions such as the internal capsule, basal ganglia, or pons. Cardioembolic TIAs arise from thrombi forming in the heart, most frequently in the left atrium or left atrial appendage secondary to atrial fibrillation. Embolization of these thrombi into cerebral arteries causes sudden focal neurological deficits. Cryptogenic TIA, or embolic stroke of undetermined source (ESUS), is characterized by cortical ischemic events without an identifiable cardiac or large artery origin, highlighting the possibility of undetected paroxysmal arrhythmias, microemboli, or occult hypercoagulable states. Less common etiologies include arterial dissection, which can occur spontaneously or following trauma, and vasculitis, which leads to inflammation and narrowing of cerebral vessels. Hypercoagulable states, either inherited or acquired, predispose to thrombosis and subsequent transient ischemia. Across all subtypes, the fundamental pathophysiologic mechanism involves a temporary mismatch between cerebral oxygen delivery and metabolic demand, resulting in reversible neuronal dysfunction. Rapid restoration of blood flow prevents permanent infarction, distinguishing TIA from completed ischemic stroke. Understanding these mechanisms is critical for guiding diagnostic evaluation, secondary prevention, and tailored therapeutic strategies, as interventions vary according to the underlying cause and vascular territory affected.[4][5]

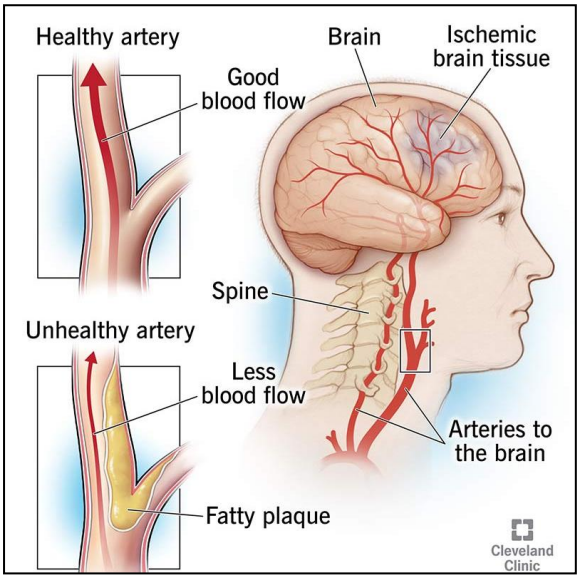

Fig. 1: Transient Ischemic Attacks.

History and Physical

The evaluation of a patient with a transient ischemic attack (TIA) requires a meticulous and structured approach because symptoms frequently resolve before presentation, making clinical history the cornerstone of diagnosis. A detailed history of the present illness should capture the exact onset, duration, timing, and sequence of neurological symptoms. It is essential to document the complete spectrum of transient deficits, including motor weakness, sensory disturbances, speech difficulties, visual changes, and coordination problems. Clinicians must inquire about associated features such as headache, vertigo, or syncope, as well as any aggravating or relieving factors. The temporal pattern, including the rapidity of onset and resolution, provides important diagnostic clues distinguishing TIA from mimicking conditions such as migraine, seizure, or peripheral neuropathies. In addition to the presenting symptoms, a comprehensive history should explore cardiovascular and cerebrovascular risk factors. Established risk factors, including hypertension, diabetes mellitus, dyslipidemia, obesity, smoking, and substance abuse, significantly increase the probability

of cerebrovascular events. Personal and family histories of stroke, TIA, or hypercoagulable disorders are critical for assessing susceptibility. Specific etiological clues may indicate the likely TIA subtype; for example, a history of atrial fibrillation, recent myocardial infarction, or valvular heart disease suggests a cardioembolic source. Transient monocular visual disturbances, such as the perception of a descending or rising curtain, point toward ipsilateral internal carotid artery pathology. Cortical symptoms, including aphasia, alexia, or visual field deficits, indicate involvement of cortical territories, distinguishing them from lacunar syndromes that typically present with subcortical motor or sensory deficits.

The physical examination is focused on detecting residual neurological deficits and identifying clues to the TIA's etiology. Focal neurological deficits may be subtle or absent at the time of evaluation, emphasizing the need for a systematic neurological assessment. Cranial nerve examination should evaluate vision, ocular motility, facial symmetry, auditory function, swallowing, and tongue movements. Monocular blindness or diplopia may reveal involvement of the optic or oculomotor pathways, while facial droop or dysarthria indicates cortical or subcortical involvement. Motor assessment should include evaluation of strength, tone, reflexes, and coordination in both upper and lower extremities. Subtle asymmetries in strength, abnormal reflexes, or evidence of clonus may reflect prior transient ischemia. Cardiovascular and vascular assessment is equally essential. Carotid auscultation may detect a bruit, indicative of stenosis and a potential source of cerebral emboli. Cardiac examination should focus on arrhythmias, valvular abnormalities, and signs of heart failure. Ophthalmologic examination, including fundoscopy, may reveal hypertensive or diabetic vascular changes, or Hollenhorst plaques, which are cholesterol emboli that signal carotid artery disease. These findings not only provide diagnostic insight but also guide urgent intervention to prevent stroke recurrence. In summary, the history and physical examination in TIA evaluation are intricately linked, with each component providing valuable diagnostic and prognostic information. Clinicians must integrate symptom chronology, risk factors, and detailed neurologic and cardiovascular assessments to identify the underlying etiology, stratify stroke risk, and initiate timely interventions. A careful and thorough assessment enhances the ability to implement targeted therapies, including antiplatelet or anticoagulant strategies, risk factor modification, and urgent referral for neuroimaging or vascular interventions, thereby reducing the risk of subsequent cerebrovascular events.

Evaluation

The evaluation of a transient ischemic attack (TIA) is a critical process aimed at confirming the cerebrovascular origin of the patient's symptoms, excluding alternative nonischemic causes, identifying the underlying vascular mechanism, and stratifying the risk of recurrent events. Prompt and thorough evaluation allows clinicians to select the most effective strategies for secondary prevention, reduce the likelihood of progression to ischemic stroke, and improve patient outcomes. Establishing a vascular origin may be achieved directly, by detecting evidence of hypoperfusion or acute infarction, or indirectly, by identifying potential sources such as large-vessel stenosis or cardiac emboli. Concurrently, clinicians must remain vigilant for conditions that mimic TIA, including migraine, seizure, metabolic disturbances, or peripheral neuropathies, which require a distinct diagnostic and therapeutic approach. Determining the pathophysiological mechanism underlying the TIA is fundamental for targeted management. Subtypes include large-vessel atherothrombosis, cardioembolic events, and small-vessel lacunar ischemia, among other less common etiologies. Identification of the mechanism informs the choice of secondary prevention measures, including antiplatelet therapy, anticoagulation, and revascularization procedures. The evaluation also provides prognostic information, allowing clinicians to categorize patients according to their likelihood of subsequent ischemic events, thereby guiding decisions regarding hospitalization and intensity of monitoring.

Imaging plays a pivotal role in TIA evaluation. The 2009 AHA/ASA guidelines emphasize neuroimaging within 24 hours of symptom onset, with MRI using diffusion-weighted imaging (DWI) preferred for its superior sensitivity in detecting small infarcts compared to CT. If MRI is unavailable or contraindicated, head CT with CT angiography serves as an alternative. Evaluation of the cervicocephalic vasculature is essential to identify treatable atherosclerotic lesions and may be performed using carotid ultrasonography, transcranial Doppler, magnetic resonance angiography, or CT angiography. For patients considered candidates for carotid endarterectomy, vascular imaging should be completed within one week of symptom onset to optimize timing of intervention. Cardiac assessment is equally important, particularly to identify potential cardioembolic sources. Electrocardiography (ECG), transthoracic echocardiography (TTE), or transesophageal echocardiography (TEE) can detect atrial fibrillation, valvular heart disease, intracardiac thrombus, patent foramen ovale, or aortic atherosclerosis. Ambulatory cardiac monitoring, including Holter or prolonged rhythm monitoring, is appropriate in patients with cortical infarcts of unclear origin, primarily to detect paroxysmal atrial fibrillation that may otherwise be missed. Routine laboratory investigations, including complete blood count, coagulation profile, comprehensive metabolic panel, fasting blood glucose, lipid panel, erythrocyte

sedimentation rate, and urine drug screen, provide additional information to identify contributing factors and rule out mimicking conditions.

Risk stratification is guided by the ABCD2 score, which predicts the likelihood of subsequent TIA or stroke based on age, blood pressure, clinical presentation, symptom duration, and diabetes status. Age over 60 years, blood pressure ≥140/90 mmHg, focal weakness (2 points) or speech impairment without weakness (1 point), symptom duration of 60 minutes or longer (2 points) or 10–59 minutes (1 point), and the presence of diabetes mellitus (1 point) constitute the scoring criteria. Two-day stroke risk correlates with ABCD2 scores: 0–1 points correspond to 0%, 2–3 points to 1.3%, 4–5 points to 4.1%, and 6–7 points to 8.1%. Patients with scores of 4 or higher are typically admitted for expedited evaluation and observation, while lower-risk patients still benefit from prompt assessment and intervention. In conclusion, TIA evaluation is multifaceted, integrating neuroimaging, vascular and cardiac assessment, laboratory testing, and clinical risk stratification. Early and comprehensive evaluation ensures accurate diagnosis, elucidates the underlying pathophysiology, identifies treatable sources, and allows timely implementation of preventive strategies. A structured and systematic approach reduces the risk of subsequent ischemic events and supports evidence-based decision-making for optimal patient outcomes [5][6][7][8][9].

Treatment / Management

The primary goal in the management of transient ischemic attacks (TIA) is to reduce the risk of subsequent cerebrovascular events, particularly ischemic strokes. The period immediately following a TIA is critical, as the risk of stroke is highest within the first 48 hours, with nearly 20% of patients experiencing a stroke within three months, and about half of these occurring in the first two days after symptom onset. Early recognition, evaluation, and initiation of treatment significantly reduce this risk. Prompt assessment of vascular status, including carotid and intracranial arteries, and identification of cardiac sources such as atrial fibrillation, are essential steps. Early detection of these risk factors allows for targeted interventions that can substantially mitigate the likelihood of recurrent cerebrovascular events [10][11][12]. Evidence from studies conducted in the early 2000s emphasized the benefits of expedited evaluation and the use of combined therapeutic strategies. The EXPRESS study conducted in the United Kingdom demonstrated that rapid assessment and implementation of interventions could reduce the risk of stroke following TIA by up to 80%, compared with standard care. This highlighted the importance of urgent management in the post-TIA period [13]. Similarly, a meta-analysis by Hackam et al. in 2007 indicated that a combination of lifestyle modification, including diet and physical activity, together with pharmacologic therapy—such as antiplatelet agents, statins, and antihypertensive medications—could reduce the risk of subsequent stroke by 80% to 90% [14]. These findings underscore the importance of an integrated, multifaceted approach to TIA management rather than reliance on single interventions.

Antiplatelet therapy is central to secondary prevention, particularly in non-cardioembolic TIA. Recent trials, including the CHANCE trial in China and the multinational POINTE trial, support the use of dual antiplatelet therapy with aspirin and clopidogrel for three weeks to one month following a TIA, followed by long-term monotherapy with a single antiplatelet agent. This regimen has been associated with reduced recurrent ischemic events without significantly increasing the risk of major bleeding [15][16]. The choice of antiplatelet therapy should be individualized based on the patient's risk profile, bleeding risk, and underlying etiology of the TIA. Management strategies must also address the underlying vascular pathology. For patients with symptomatic high-grade stenosis of the cervical internal carotid artery (≥70%), revascularization through carotid endarterectomy is recommended. The decision to perform surgical intervention in patients with moderate stenosis (50–69%) depends on the surgical complication rates and overall risk-benefit assessment, particularly given the improvements in aggressive medical management strategies. Endovascular stenting, such as Wingspan stenting for intracranial major artery stenosis (70–99%), has not demonstrated superiority over medical therapy alone, as evidenced by the SAPPRIS trial, highlighting the continued importance of optimized medical management [17]. For patients with TIA caused by cardioembolic sources, particularly atrial fibrillation, oral anticoagulation is indicated to prevent further thromboembolic events. Warfarin and direct oral anticoagulants (DOACs) are commonly employed depending on patient-specific factors, including renal function, bleeding risk, and comorbidities. Continuous monitoring and follow-up are essential to ensure therapeutic efficacy and safety. In conclusion, the management of TIA requires rapid evaluation, risk stratification, and the initiation of targeted therapeutic strategies. Early intervention, comprehensive secondary prevention measures, and individualized treatment based on underlying etiology are critical to reducing the risk of recurrent TIA and ischemic stroke. The integration of lifestyle modification, antiplatelet or anticoagulant therapy, and revascularization procedures where appropriate ensures a patient-centered approach that maximizes safety and efficacy while minimizing the risk of recurrent cerebrovascular events [10][11][12][13][14][15][16][17].

Differential Diagnosis

Differentiating transient ischemic attacks (TIA) from other neurological or systemic conditions is critical because the management, urgency, and prognosis differ significantly. Several conditions can mimic TIA, and a careful evaluation of patient history,

physical examination, and supportive diagnostic testing is necessary to establish the correct diagnosis. Carotid artery dissection is an important differential, particularly in younger patients or those presenting with neck pain, headache, or focal neurological deficits. Dissections may result in embolic phenomena leading to transient ischemia, and imaging such as CTA or MRA of the cervical vessels is required for diagnosis. Meningitis, including bacterial and viral etiologies, can present with headaches, confusion, and focal neurological signs that overlap with TIA symptoms. Meningococcal meningitis specifically requires prompt recognition due to its rapid progression, with additional signs such as fever, neck stiffness, photophobia, and a petechial rash. Lumbar puncture and cerebrospinal fluid analysis remain diagnostic standards. Multiple sclerosis is another condition that can produce transient neurological deficits mimicking TIA, often with a relapsing-remitting course, and diagnosis relies on MRI findings demonstrating demyelinating plaques in the central nervous system. Ischemic and hemorrhagic strokes are both key differentials, with strokes generally resulting in persistent deficits beyond 24 hours, whereas TIAs resolve spontaneously. Subarachnoid hemorrhage may present with sudden-onset headache, nausea, vomiting, and neurological deficits, sometimes mimicking TIA but often accompanied by meningeal signs. Syncope, often cardiovascular in origin, can produce transient neurologic or sensory symptoms but is generally associated with loss of consciousness, distinguishing it from TIA. Other mimics include vestibular disorders, seizure activity, migraine with aura, hypoglycemia, and electrolyte disturbances, which may require laboratory testing, EEG, or metabolic evaluation to rule out. Accurate differentiation is essential to prevent unnecessary interventions and to prioritize early TIA-specific management to prevent subsequent strokes [18][19].

Staging / Risk Stratification Scores

Risk stratification is a fundamental component of TIA management because it identifies patients at high risk for early stroke and guides the intensity of monitoring and intervention. The ABCD2 score is one of the most widely used clinical tools for this purpose. It incorporates age, blood pressure, clinical features, duration of symptoms, and presence of diabetes. Specifically, patients older than 60 years receive one point, and those with systolic blood pressure ≥140 mmHg or diastolic ≥90 mmHg receive one point. Clinical features are weighted based on severity: speech impairment without weakness scores one point, while focal weakness, with or without speech disturbance, scores two points. Symptom duration of 10–59 minutes scores one point, and duration ≥60 minutes scores two points. A history of diabetes adds one point. The ABCD2 score provides a stratified estimate of short-term stroke risk. Scores of 6–7 indicate an 8% risk of stroke within 48 hours, while scores below 4 correspond to a risk of approximately 1% in the same period. This scoring system helps clinicians prioritize urgent evaluation and intervention, particularly for patients who may benefit from hospital admission, early imaging, and initiation of secondary prevention therapies. It is important to note, however, that some patients with critical carotid stenosis or high embolic risk may have low ABCD2 scores, and clinical judgment must supplement scoring to avoid underestimating risk. Additional risk assessment may incorporate imaging results, cardiac evaluation for embolic sources, and laboratory studies evaluating coagulation or metabolic contributors. Integration of these findings with ABCD2 scores enables a more accurate individualized risk profile, guiding decisions about inpatient versus outpatient management, timing of revascularization, and intensity of pharmacologic therapy. These scores also serve as communication tools among multidisciplinary teams, facilitating consensus on urgency and prioritization of interventions to prevent recurrent TIA or ischemic stroke [18][19].

Other Issues

Accurate differentiation of TIA from mimics is critical because misdiagnosis may delay preventive treatment and increase stroke risk. Conditions such as vertigo, seizure, migraine with aura, Bell's palsy, drug withdrawal, dementia, electrolyte imbalances, syncope, or acute infections can mimic the transient neurological deficits of TIA. A detailed history, careful neurologic examination, and appropriate use of imaging or laboratory studies help reduce diagnostic errors. Clinicians should be aware that TIA symptoms are often fleeting, and physical findings may be absent by the time of evaluation. Early recognition and management can significantly reduce the risk of a subsequent stroke, highlighting the importance of clinical vigilance [18][19].

Enhancing Healthcare Team Outcomes

Management of TIA requires an interprofessional approach involving emergency physicians, neurologists, nurses, and allied health professionals. The triage nurse plays a critical role in early recognition, ensuring timely neurologist consultation and initiation of urgent diagnostic evaluation. Education of patients regarding stroke symptoms, blood pressure control, smoking cessation, and adherence to a heart-healthy diet is crucial to secondary prevention. The early stroke risk following TIA ranges from 4% to 9% within 90 days, while the long-term risk may reach 20–30% within five years. Patients often share common cardiovascular risk factors, necessitating comprehensive evaluation and management that addresses both cerebrovascular and cardiac health. Coordinated care, patient education, and continuous risk monitoring are essential to improving outcomes and reducing morbidity associated with recurrent strokes. The integration of multidisciplinary expertise ensures timely

intervention, optimal secondary prevention, and patient-centered care [18][19].

Conclusion:

Transient ischemic attack represents a pivotal opportunity for stroke prevention. Its transient nature often leads to underestimation, yet the risk of progression to ischemic stroke is highest within the first 48 hours. Comprehensive evaluation—including neuroimaging, vascular studies, and cardiac assessment—enables accurate diagnosis and identification of underlying etiologies. Risk stratification using tools like the ABCD² score aids in prioritizing urgent interventions, though clinical judgment remains indispensable. Management strategies must be individualized, addressing both the immediate and long-term risk of cerebrovascular events. Antiplatelet therapy, particularly short-term dual therapy, has proven effective for non-cardioembolic TIA, while anticoagulation is essential for cardioembolic sources. Surgical interventions such as carotid endarterectomy remain critical for patients with significant stenosis. Beyond pharmacologic and procedural measures, lifestyle modification—including blood pressure control, smoking cessation, and dietary improvements—plays a central role in secondary prevention. Ultimately, TIA care demands a coordinated, multidisciplinary approach to ensure timely intervention and patient education. By integrating evidence-based strategies, clinicians can reduce recurrent stroke risk by up to 80%, transforming TIA from a harbinger of disability into an opportunity for prevention.

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