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Transient ischemic attack: A literary review of cerebral ischemia

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ABSTRACT:
A transient ischemic attack (TIA) is a medical emergency. It is defined as a transient episode of neurologic dysfunction due to focal brain, spinal cord or retinal ischemia without acute infarction or tissue injury. Up to a 10% risk of recurrent stroke exists after a TIA, and up to 80% of this risk is preventable with urgent assessment and treatment. Imaging of brain and intracranial and extra cranial blood vessels using CT, CT angiography, Carotid Doppler Ultrasound, and MRI is an important part of the diagnostic assessment. Treatment options include anticoagulation for atrial fibrillation, carotid revascularization for symptomatic carotid artery stenosis, antiplatelet therapy, and vascular risk factor reduction strategies.Sudden neurologic dysfunction caused by focal brain ischemia with imaging evidence of acute infarction defines acute ischemic stroke (AIS), while an ischemic stroke with neurologic deficits but without acute infarction defines transient ischemic attack (TIA). TIAs are at high risk of early stroke, and their risk may be stratified by clinical scale, vessel imaging and diffusion magnetic resonance imaging. However, recent findings suggest that having a TIA correlates with deficits that can persist beyond the resolution of clinical symptom even in the absence of imaging evidence of ischemic tissue injury. These deficits may be result of subtle perturbations to brain structure and/or function that are not easily appreciated using the standard clinical and imaging tools that are currently employed in practice. An estimated 7.5% to 17.4% of patients with TIA will have a stroke in the next 3 months. Patients presenting with non disabling AIS or high risk TIA, who do not have severe carotid stenosis or atrial fibrillation, should receive dual antiplatelet therapy with aspirin and clopidigrel within 24 hours of presentation.

Keywords: Spinal cord, CT angiography, Anticoagulation, Revascularization, Ischemic stroke, Antiplatelet therapy.

I. INTRODUCTION:

Transient ischemic attack (TIA) was originally defined as self-resolving focal cerebral ischemia with symptoms lasting less than 24 hours. The newer definition added the limitation that there should be no evidence of acute brain tissue infarction, to recognize that the acute injury to brain can result from ischemia of less than 24 hours duration. A transient ischemic attack is a medical emergency. It is defined as a transient episode of neurologic dysfunction due to the focal brain, spinal cord or retinal ischemia, without acute infarction or tissue injury. The definition of a TIA has moved from time based to tissue-based. A TIA typically lasts less than an hour, more often minutes. TIA can be considered as a serious warning for an impending ischemic stroke; the risk is highest in the first 48 hours following a transient ischemic attack. The subsequent risk of TIA or ischemic stroke can be stratified with a simple clinical measure. Immediate multimodality therapeutic interventions should be initiated. These will include aggressive treatment of blood pressure, high dose stain, antiplatelet therapy, blood sugar control, diet and exercises. This treatment scheme may substantially reduce the risk of recurrent strokes or future TIA by at least 80%. [1] [2]

A transient ischemic attack (TIA), also known as a mini stroke, occurs when blood supply to the brain temporarily stops. The reduced blood supply does not usually last for longer than 5 min., but a TIA is still a medical emergency. It may be a warning of a major stroke to come. Many people do not seek help for a TIA because the symptoms pass quickly. However, the Center forDisease Control and Prevention (CDC) note that more than One third of people who do not receive treatment for a TIA have a major stroke within a year. Currently it can be argued that approach to investigate transient ischemic attack and minor stroke is a compromise between timeliness of investigation and accuracy of diagnosis.While the new definitions of TIA and stroke are much more accurate regarding the biological consequences of cerebral ischemia [80] [81] [83].

The advanced newer, neuroimaging informed operational definitions of TIA such as “a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction” [43]. However, with rare exceptions [44], the newer definitions have not yet been formally considered

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for endorsement or rejection by authoritative organizations.

Transient ischemic attack and minor ischemic stroke are associated with brain dysfunction in a circumscribed area caused by a regional reduction in blood flow (i.e. ischemia), resulting in either transient or minor observable clinical symptoms. Identification of ischemia is important as 20% of patients with ischemic stroke presents with a TIA in a hours to days preceding the stroke. [20] [21]. Up to 80% of strokes after TIA are preventable; thus early diagnosis and treatment isthekey. The clinical definitions of TIA and ischemic stroke are based on focal neurologic signs or symptoms are referable to known cerebral arterial distribution without direct measurement of blood flow or cerebral infarction. This is important to note that TIA and stroke represent different ends of ischemic continuum from the physiologic perspective, but clinical management is similar. The historical time based definition of TIA was based on full resolution of all symptoms within 24 hours of onset. It is also relevant that the diagnosis of TIA and minor stroke are commonly used interchangeably and recorded as such in medical records. Treatment to prevent ischemic stroke following TIA and treatment to prevent recurrent stroke following minor ischemic stroke are also similar. Very early assessment of these patients also makes the distinction between TIA and minor ischemic stroke difficult. A limitation of clinical definition of stroke and TIA is that they rely on presumed cause of symptoms: ischemia. Because patients vary in reporting the events they have experienced, even an astute physician may find it challenging to make a certain diagnosis based on the history and physical examination alone. Even experts do not agree about which clinical events are in fact TIAs. [22] [23] [24].

Symptoms:

The symptoms of TIA will depend on which part of the brain is not receiving adequate blood flow. As with a major stroke, the acronym FAST (face, arms, speech, time) can help people remember the symptoms to look for:

F = Face: The eye or mouth may droop on one side, and the person may be unable to smile properly.

A = Arms: Arm weakness or numbness might make it hard to raise one or both arms or keep them raised.

S = Speech: The person's speech may be slurred or garbled.

T = Time: Someone should call the emergency services at once if a person has one or more of these symptoms.

The person may also have:

- Numbness or weakness, especially on one side of the body
- Sudden confusion
- Difficulty understanding what others are talking about
- Vision problem
- Dizziness
- Problem with co ordination
- Difficulty walking
- A very bad headache
- A loss of consciousness, in some cases.

TIA symptoms are temporary. They can last for a few minutes to a few hours, and they usually disappear completely after 24 hours. The same factors that lead to temporary insufficiency of blood flow in a TIA can cause a stroke due to longer lasting blood flow reduction, which can lead to permanent brain damage.

TIA subtypes, classified according to the pathophysiological mechanisms are similar to ischemic stroke subtypes. They include large artery atherothrombosis, cardiac embolism, small vessel (lacunar), cryptogenic and uncommon subtypes such as vascular dissection, vasculitis etc. The common risk factor for all TIA include diabetes, hypertension, age, smoking, obesity, alcoholism, unhealthy diet, psychosocial stress, and lack of regular physical activity. A previous history of TIA or stroke will increase substantially the subsequent risk of recurrent stroke or TIA. [4] [5]. Among all risk factors, hypertension is the most important one for an individual as well as in a population. A TIA is a clinical syndrome characterized by the sudden onset of focal neurologic deficit presumed to be on a vascular basis. Imaging can support the diagnosis, but TIA is primarily a clinical diagnosis. Descriptors such as "numb", "dead", "heavy" or "weak" may have different meanings for different patients and require clarification, similar to the different meanings patients may have for "dizzy". The most important clinical determination is whether the neurologic symptoms are focal or non focal. Focal neurologic symptoms usually affect one side of the body. Non focal neurologic symptoms include generalized weakness, light headedness, fainting, blackouts and bladder or bowel symptoms. Loss of consciousness is only very rarely a symptom of stroke or TIA.

TIA symptoms are often resolved by the time the patient presented to the doctor or

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emergency department. The history of present illness should include onset, duration, timing, complete neurological symptoms, associating symptoms or any aggrevating or relieving factors. Some have indeed an ischemic event but others have had symptoms related to a stroke or TIA mimic, such as migraine, epilepsy, multiple sclerosis or peripheral nerve entrapment. Motor and speech symptoms may have a higher likelihood of brain ischemia as the cause of the symptoms because the differential diagnosis for such clinical presentations is much narrower, and patients who present with motor or speech symptoms are known to be at high risk for recurrent stroke [25]. However, patients who present with symptoms other than motor and speech symptoms (e.g. sensory symptoms and dizziness) have a more uncertain etiology [26]. Posterior circulation ischemia can pose an additional diagnostic challenge as symptoms are more variable than those that occur with hemispheric ischemia [27]. Although the proportion of patients with true ischemia is lower in those without motor or speech symptoms, it is important not to miss patients with true TIAs and minor ischemic strokes [27] [28]. The diagnosis of TIA remains largely clinical and based on taking an accurate history. This contributes to the variability in the diagnosis of TIA, with high rates of disagreement seen even between neurologists [22]. The clinician should also try to identify the associated risk factors such as coronary artery disease, smoking, drunk abuse, obesity, diabetes mellitus, dyslipidemia and hypertension, as well as personal family history of hypercoagulability disorder, stroke or TIA. Physical examination should focus on identifying focal neurological deficit, and speech disturbances which are the most common presenting symptoms in patients with TIA. Cranial nerve examination can yield findings of monocular blindness, disconjugate gaze, facial droop, hemianopia, diplopia, abnormal tongue movement, difficulty swallowing, and auditory dysfunction. Cardiac examination and carotid auscultation for a carotid bruit are very important. Fundoscopy is important to look for any fundoscopic evidence of vascular changes as result of hypertension or diabetes. It may also show a Hollenhurst plague which will indicate an underlying internal carotid artery disease.

In candidates for carotid endarterectomy, carotid imaging should be performed within 1 week of onset of symptoms. Cardiac assessment should be done with ECG, Echocardiogram/TEE to find a cardioembolic source and the presence of patent foramen ovale, valvular disease, cardiac thrombus and atherosclerosis. Routine blood tests including complete blood count (CBC), PT/INR, CMP, FBS, lipid panel, urine drug screen, and ESR should be considered.[6][7][5].

The ABCD2 score was derived from providing a more robust prediction standard. The ABCD2 score includes factor including age, blood pressure, clinical symptoms, duration and diabetes.

Age: older than 60 years (1 point)

Blood pressure greater than or equal to 140/90 mmHg on first evaluation. (1 point)

Clinical symptoms: a focal weakness with the spell (2 points) or speech impairment without weakness (1 point)

Duration greater than 60 min (2 points) or 10 min to 59 min (1 point)

Diabetes mellitus (1 point)

The 2-day risk of stroke was 0% for scores of 0 or 1, 1.3% for 2 or 3, 4.1% for 4 or 5, and 8.1% for 6 or 7. Most stroke centers will admit patients with TIA to the hospital for expedited management and observation if score is 4 or 5 or higher. This expedited approach has been proven to improve the outcome. [9]

Causes:

- Atherosclerosis
- Blood clots due to heart disease, cardiovascular disease or an irregular heart rhythm
- Blood clots due to a blood condition, such as sickle cell disease
- An air bubble in blood stream

Risk factors:

The risk factors for a TIA are similar to those for a stroke.

- Having a family history of stroke or TIA
- Being 55 years or above
- Being assigned male at birth
- Having high blood pressure
- Having cardiovascular disease, diabetes, high cholesterol levels
- Smoking tobacco
- Eating a diet that is high in unhealthy fats and salts
- Having high homocysteine levels
- Having over weight or obesity
- Having a type of heart beat known as atrial fibrillation

Investigations:

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A full neurologic and cardiac examination should be completed on all patients with suspected TIA. CT was the first tool to identify acute ischemic structural injury to brain after transient symptoms lasting less than 24 hours [82]. Blood pressure, pulse rate and oxygen saturation should be obtained, and ECG should be performed to evaluate for arterial fibrillation. Many patients will also require an echocardiogram and some form of extended cardiac monitoring if no definitive cause is found for the TIA. Over the last decade, substantial new diagnostic advances have occurred, including the widespread availability of MR angiography (MRA) and Computed Tomography (CT) angiography (CTA), the recognition that diffusion MR frequently shows abnormalities in classic TIA patients and the development and the validation of risk stratification algorithms that identify TIA patients at higher and lower risk of early stroke. Numerous studies also have shown that the short term risk of stroke is particularly high, with most studies finding risks exceeding 10% in 90 days [45] [48] [50] [53-62]. Ischemic stroke appears to carry a lower short-term risk of subsequent ischemic stroke than TIA, with reported 3-month risks generally ranging from 4% to 8% [57-59] [61] [63-76].

Prognosis:

About 10% of patients present with TIAs or minor strokes will have a stroke within the next 90 days [25] [31] [32] with the highest risk period being the first 24 hours [33]. Wide consensus exists that TIA and minor ischemic stroke are medical emergencies that necessitate immediate management [34]. For instance, the cardiovascular health study estimated a prevalence of TIA in men of 2.7% for 65 to 69 years of age and 3.6% for 75 to 79 years of age. For women, TIA prevalence was 1.6% for 65 to 69 years of age and 4.1% for 75 to 79 years of age [46]. The percentage varies, depending on such factors as how TIA is defined, which stroke subtype is evaluated, and whether the study is a population based series [48] [49].

Clinical /Event features and scores:

Certain clinical features have been associated with recurrent stroke after TIA. These include diabetes mellitus [25], hypertension [35] [36], symptoms duration, and weakness or speech disturbance [25] [36]. The Rotterdam Study [37] followed patients with transient neurologic attacks for 10 years and found an increased risk of stroke not only in patients with focal symptoms (i.e. possible TIAs) but also in patients who had transient episodes of non specific symptoms. Posterior circulation events, in particular, can cause non specific symptoms [38]. Evidence of an acute infarct on a non contrast CT alone has been shown to be predictive of recurrent stroke in patients with TIA (i.e. patients whose symptoms had resolved), although the proportion of patients with evidence of acute infarcts was small (4%). [39].

Rapid advances in imaging technology in the past 25 years have contributed significantly to our understanding of pathophysiology of TIAs. MRI is not as widely available as CT and is generally more expensive. In study of TIAs evaluated in emergency departments in Ontario, Canada, from May to December 2000, only 3% received MRI within 30 days. With respect to frequency of identifying brain infarcts in patients with TIAs, one needs to analyze whether the infarcts reported are new or old, whether they are in a clinically relevant vascular territory or not, and whether infarcts are cortical or in a perforator territory. Across various studies, MRI has shown at least 1 infarct somewhere in cerebrum in 46% to 81% of TIA patients [77] [78]. The prevalence of intracranial disease is much higher in nonwhite populations. Reports found that 51% to 77% of Asian patients with TIA had intracranial stenosis or occlusion [79] [80].

Patients with minor ischemic stroke and TIAwho are at the highest risk of recurrent events and disability can be identified using non invasive CT angiography (CTA) [40]. Evidence of 50% or greater stenosis or occlusion in a symptom relevant vessel in the intracranial or extra cranial circulation puts a patient at high risk of a recurrent stroke [40]. Brain imaging using MRI is very sensitive way of assessing for brain ischemia. Diffusion-weighted imaging (DWI), which shows the abnormal diffusion of water in the setting of focal brain ischemia, is the most helpful sequence. Most studies of recurrent stroke after TIA have shown an increased risk of short-term recurrent stroke in the presence of a lesion seen on DWI. Most stroke neurologists would agree that patients who have a negative DWI but have truly had TIAs clearly exist, and thus they will treat patients for TIA even with a negative DWI. DWI has better sensitivity than CT for identifying ischemic injury to brain [85] [86] [87], and it can detect relevant ischemic lesions in approximately 50% of individuals who have transient neurological symptoms lasting <24 hours [88] many of whom are CT negative. Unfortunately, up to two-thirds of individuals who are acutely DWI-positive will no longer show

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evidence of infarction at 21 days [89]. Individuals who had lesion free TIAs may also have an increased chance of experiencing fatigue [90].

Treatment/Management:

Stroke and TIA are on a spectrum of serious conditions involving brain ischemia. Both are markers of reduced cerebral blood flow and an increased risk of disability and death. However, TIAs offer an opportunity to initiate treatment that can forestall the onset of permanently disabling injury [51] [52].

The recognition and management of TIA offers the greatest opportunity to prevent disabling stroke. Studies have shown up to an 80% reduction in the risk of stroke after TIA with the early implementation of secondary stroke prevention strategies [28] [29], including revascularization of patients with symptomatic carotid artery stenosis, anticoagulation of patients with atrial fibrillation, treatment with antiplatelet agents, treatment with statins for most patients management of hypertension, and lifestyle interventions, such as smoking cessation or weight loss. Another recent study identified that gait may be abnormal in people who have TIA or minor stroke for up to 5 months after the index event. Deep neural networks are starting to be used with some success to identify individuals at risk of tissue injury after stroke and to predict treatment effects.

The main aim of treatment of TIA is to decrease the risk of subsequent stroke or TIA. Early treatment after a TIA can significantly reduce the risk of stroke within 3 months has been reported to be around 20%, with approximately 50% of these strokes occurring within the first 2 days after initial presentation. It is extremely important to evaluate the vessel status and look for atrial fibrillation when a patient comes with TIA. This will significantly reduce future strokes. Management of TIAs should focus on treating underlying etiologies. [10] [11] [12]. The express study in the UK has shown the importance of early intervention versus regular treatment by reducing 80% of the stroke risk. [13] More recent studies in China (CHANCE trial) and the multinational POINTE trial also confirmed dual antiplatelet with aspirin and clopidogrel for 3 weeks to 1 month followed by a single antiplatelet agent is the best scheme for antiplatelet therapy. [15] [16] Revascularization is a recommended for symptomatic cervical internal carotid artery stenosis of 70%or higher.Whether to operate on patient with 50-69% stenosis will depend more on surgeon's complication rates given the vast improvement in efficacy of aggressive medical therapy. Wingspan stenting of intracranial major arterial stenosis of 70-99% stenosis is not better than aggressive medical therapy alone. [17]

For most patients, it will be a single antiplatelet agent, usually aspirin mono therapy (81mg/d to 325mg/d). Other options include 75mg/d clopidogrel or a combination of 25mg aspirin and 200 mg extended-release dipyridamole 2 times a day [41]. The Fast Assessment of Stroke and Transient Ischemic Attack to prevent Early Recurrence (FASTER) trial compared the effectiveness of 3 months of treatment with 81mg aspirin and 75mg clopidogrel commenced within 24 hours of onset versus aspirin alone in patients with minor strokes/TIAs [42].

In some cases, a doctor may recommend surgery to remove a blockage or part of damaged artery.

Diagnosis:

Anyone experiencing signs or symptoms of a TIA needs an immediate medical assessment to find out why it happened and how to prevent recurrence or more severe event.

Differential diagnosis of TIA includes but is not limited to vertigo, dizziness, seizures, headaches, bell's palsy, drug withdrawl, dementia, electrolyte disorder, acute infections, syncope and alcoholism. The early risk of stroke varies from 4-9% within 90 days and without treatment, the risk of stroke within 5 years varies from 20-30%. At the same time, these patients also have the same risk factors for adverse cardiac events. Once the TIA has been diagnosed, the patient must been referred to a neurologist. At the same time, the patient should be educated about the importance of the blood pressure control, discontinuing smoking and eating a healthy diet. Finally the patient should be educated about the symptoms of the stroke and when to seek immediate medical assistance. [18] [19] (level V)

Possible tests include:

- Blood tests to check blood pressure, cholesterol levels and clotting ability
- An electrocardiogram to measure electrical activity and rhythms of heart
- An echocardiogram to check the pumping activity of heart
- A CT scan to reveal any sign of an aneurysm, bleeding or changes to blood vessels in the brain
- A MRI scan to help identify damage to the brain

II. DISCUSSION:

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TIA offers the greatest opportunity to prevent stroke that physicians encounter. A TIA should be treated as a medical emergency, as up to 80% of strokes after TIA as preventable.Differentiating transient ischemic attack from other mimicking condition is important.Symptoms of TIA include weakness and numbness on one side of the body, drooping on one side of the face, and difficulty speaking. Transient ischemic attacks are usually associated with a focal neurologic deficit and / or speech disturbance in a vascular disease. Among all risk factors, hypertension is the most important one for an individual as well as in a population. The estimated overall prevalence of TIA among adults in the United States is approximately 2%. Few studies have shown that the majority of people who presented with initial stroke had prior TIA symptoms [4]. The 2009 AHA/ASA guidelines include "neuroimaging within 24 hours of symptoms onset and further recommend MRI and diffusion-weighted MR imaging as preferred modalities." Brain MRI with diffusion-weighted imaging has a greater sensitivity than CT for detecting small infarcts in patients with TIA. The ABCD2 score is very important for predicting subsequent risks of TIA or stroke.Early treatment after a TIA can significantly reduce the risk of stroke within 3 months has been reported to be around 20%, with approximately 50% of these strokes occurring within the first 2 days after initial presentation. Hackam et al did a meta-analysis in 2007 showing that combination of diet, exercise, antiplatelet, statin and antihypertensive therapy may reduce the subsequent stroke by 80-90% [14]. Stroke is the fifth leading cause of death and a leading cause of disability in the United States, affecting nearly 800000 individuals.

The early risk of stroke varies from 4-9% within 5 years varies from 20-30%. At the same time, these patients also have the same risk factors for adverse cardiac events. As many as 60% of patients referred to a TIA clinic will not have a final diagnosis of TIA[29] [30]. Identification of possible TIA mimics is an important stage in the assessment of patients with transient neurologic symptoms. Many stroke neurologists find MRI particularly helpful in cases in which the diagnosis is not 100% clear based on the history. MRI and DWI have become the methods of choice in TIA and minor stroke diagnosis however, DWI has key limitations that make it problematic to rely upon it as a sole diagnostic tool.Recurrent stroke was seen in 8.2% of patients in the clopidogrel-aspirin group, as compared with 11.7% of those in the aspirin-only group. The risk of heamorrhage was not different in the two groups. Combined aspirin and clopidogrel for 3 weeks followed by single antiplatelet therapy reduces stroke risk from 7.8% to 5.2%.

Additional statistics suggest that 20% of those have a TIA have a stroke within 3 months, and half of these will happen within 2 days of the TIA. Diagnostic recommendations include: TIA patient should undergo neuroimaging evaluation within 24 hours of symptom onset, preferably with magnetic resonance imaging, including diffusion sequences, non-invasive imaging of cervical vessels should be performed and non-invasive imaging of intracranial vessels is reasonable; electrocardiography should occur as soon as possible after TIA. MRI, including diffusion sequences, should now be considered a preferred diagnostic test in investigation of the patient with potential TIAs.

Ideally, patients with TIA should be evaluated expeditiously with tests assessing the extra cranial and intracranial circulation. The prevalence of intracranial disease is much higher in non-white populations. Reports found that 51% to 77% of Asian patients with TIA had intracranial stenosis or occlusion [79] [80]. Robotics provide a means by which to investigate behaviors in a more sensitive way than is possible with standard clinical tools and allows the behavioral correlates of neurological injury to be investigated.

III. CONCLUSION:

The assessment of TIA is all about making the correct diagnosis, and taking the good history is the key. Once the TIA diagnosis has been made, cardiac and neurovascular imaging can help inform the potential etiology and guide initiation of evidence based secondary stroke preventive strategies. Ideally, obtaining the history, imaging, and identifying the etiology occur on the same day as presentation to reduce risk of recurrent cerebral ischemia. Across various studies, MRI has shown at least 1 infarct somewhere in the cerebrum in 46% to 81% of TIA patients. Unfortunately, up to two-thirds of individuals who are acutely DWI-positive will no longer show evidence of infarction at 21 days.

TIA incidence in a population is difficult to estimate due to other mimicking disorders, but TIA incidence in the United States could be around half a million per year, and estimates are about 1.1 per 1000 in the United States population. Presence

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of any cortical symptom such as language disturbance or visual field loss will point to a cortical TIA rather than a lacunar syndrome. The 2-day risk of stroke was 0% for scores of 0 or 1, 1.3% for 2 or 3, 4.1% for 4 or 5, and 8.1% for 6 or 7. Early treatment after a TIA can significantly reduce the risk of stroke within 3 months has been reported to be around 20%, with approximately 50%of these strokes occurring within the first 2 days after initial presentation. Patients with an ABCD2 score of 6-7 have an 8% risk of stroke within 48 hours. Patients with an ABCD2 score of less than 4, have a 1% risk of stroke within 48 hours. Even though, scales are important in the evaluation of TIA, be aware that patients with critical carotid artery stenosis may sometimes present with a very low ABCD2 score. The main criteria used for TIA are the clinical history and objective findings on neurologic examination consistent with focal neurologic dysfunction at some point of evaluation and imaging of the brain.

Evidence of 50% or greater stenosis or occlusion in a symptom relevant vessel in the intracranial or extra cranial circulation puts a patient at high risk of a recurrent stroke. Atrial fibrillation is a common cause of transient ischemic attack and ischemic stroke. Finding out why a transient ischemic attack occurred, is the key to prevent a recurrent stroke. Among patients who present with stroke, the prevalence of prior TIA has been reported to range from 7% to 40%. Recognition and management of transient ischemic attack offers the greatest opportunity to prevent disabling stroke. In patients presenting with acute ischemic stroke (AIS)and disabling deficits interfering with activities of daily living, intravenous anteplase improves the likelihood of minimal or no disability by 39% with intravenous recombinant tissue plasminogen activator Vs 26% with placebo when administered within 3 hours of presentation and by 35.3% with IV right PA VS 30.1% with placebo when administered within 3 to 4.5 hours of presentation. Statistics suggest that TIAs affect around 2% of the population of the United States.

The current approach to TIA and minor stroke investigation is focused appropriately on immediate threats to health such as recurrent stroke. Additionally the ability to identify tissue injury rapidly remains incomplete. Research on tools such as MEG, EEG, robotics, and various MRI based tools to study brain function after TIA and minor stroke is still a work in progress and presently has no prognostic value. Importantly, there is increasing recognition that there are other consequences to TIA and minor stroke beyond the presence or absence of DWI lesions.

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