Diagnosing and evaluating patients with a transient ischaemic attack

In this series, we present authoritative advice on the investigation of a common clinical problem, specially commissioned for family doctors by the Board of Continuing Medical Education of the Royal Australasian College of Physicians.

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A transient ischaemic attack (TIA) is not a benign event and should under some circumstances be treated as a medical emergency. Failure to adequately recognise and evaluate this warning sign could mean missing an opportunity to prevent permanent disability or death. The risk of stroke after a TIA is up to 10% at seven days and 15% at 30 days, with 50% of strokes occurring in the first 48 hours.[1,2] Recent evidence shows that by using a simple scoring scheme (the ABCD^2 score),[3] those patients at high risk of stroke can be identified and should be referred for emergency assessment.

A case study

A 69-year-old man, Mr L, consults his GP one hour after recovering from a 45-minute episode of difficulty speaking and weakness of the right side of his face and right arm. He has a history of hypertension and hypercholesterolaemia.

What is the appropriate management of this patient?

Definition and clinical presentation

A TIA is a clinical syndrome characterised by the sudden onset of focal neurological symptoms and/or signs that last less than 24 hours. These symptoms or signs are presumed to have been caused by focal cerebral or retinal ischaemia as a result of either arterial thrombosis or embolism associated with arterial, cardiac or haematological disease.

The vast majority of TIAs are brief, with symptoms lasting less than one hour. Focal neurological and ocular symptoms and signs common in TIA are outlined in Table 1. In general, a TIA presents as a syndrome rather than any one sign or symptom.

About 15 to 20% of patients who have a stroke report a preceding TIA, and a similar proportion

IN SUMMARY

- **A transient ischaemic attack (TIA) is a clinical syndrome characterised by the sudden onset of focal neurological symptoms and/or signs that last less than 24 hours.**
- **A focused and prompt history and physical examination are important for all patients with acute transient neurological symptoms to confirm the diagnosis of the cerebral ischaemic syndrome and to localise the vascular territory affected.**
- **All patients with an ABCD^2 score of 4 or greater should be sent to an emergency department for urgent evaluation; those with a score of less than 4 can complete their investigations as outpatients.**
- **Failure to adequately recognise and evaluate a TIA could mean missing an opportunity to prevent permanent disability or death.**

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have a preceding minor stroke.[1] These 'warning' events provide an opportunity for therapeutic intervention. A recent study shows that urgent assessment of patients in a rapid response TIA clinic and early initiation of a combination of existing preventative treatments can reduce the risk of early recurrent stroke by about 80%.[4]

Primary care (pre-emergency department)

For all patients with acute transient neurological symptoms it is important to take a focused and prompt history and perform a physical examination to confirm the diagnosis of the cerebral ischaemic syndrome and localise the vascular territory affected. This can be important in directing investigations and determining the potential cause of the TIA.

A TIA affecting the carotid artery territory usually results in contralateral motor, sensory and higher cortical symptoms. Amaurosis fugax is strongly associated with internal carotid artery stenosis, because the ophthalmic artery is the first branch of the internal carotid artery. A TIA affecting the posterior circulation can manifest in a multitude of symptoms and signs, including vertigo, imbalance, ataxia and cranial nerve dysfunction (see Table 1).

All patients with ongoing symptoms suggestive of cerebral ischaemia should be immediately sent to a hospital emergency department via ambulance since there is no reliable way to determine if the neurological deficits represent ischaemia without subsequent brain damage (TIA) or if ischaemia will result in permanent damage to the brain (stroke). Patients presenting to the emergency department within three hours of onset of symptoms may be considered for thrombolytic therapy in centres capable of administering this treatment. More than 50% of patients with symptoms lasting longer than one hour but less than 24 hours (i.e. clinically classified as a TIA) will have imaging evidence of infarction if acute MRI scanning is performed.

Differential diagnosis

The diagnosis of a TIA can be problematic because it is often based on clinical history alone and specifically on the recollection of the patient who was neurologically impaired during the event. The differential diagnosis of TIA is wide and includes metabolic, structural and vascular disorders.

The most common mimics of TIA-like symptoms are:

- seizure with Todd's paralysis
- migraine headache with aura
- glucose derangements
- global cerebral ischaemia from hypotension

Investigating transient ischaemic attacks

The short-term risk of stroke in patients after a transient ischaemic attack (TIA) is high and relatively predictable. Identification of TIAs thus provides an important opportunity for the clinician to intervene and prevent strokes. For all patients who have had acute transient neurological symptoms, a focused and prompt history should be taken and physical examination performed to confirm the diagnosis of cerebral ischaemic syndrome and localise the vascular territory affected.

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Table 1. Focal neurological and ocular symptoms and signs common in TIAs

Affected function	Symptoms and signs	Localisation
Motor function	Weakness or paralysis of one side of the body, in whole or in part (hemiparesis)	Carotid artery territory, posterior circulation
	Simultaneous bilateral weakness	Brainstem
	Difficulty swallowing (dysphagia)	Brainstem
Co-ordination	Imbalance (ataxia) with standing or walking	Cerebellum, brainstem
	Clumsy arms or legs	Cerebellum, brainstem
Sensory function	Altered feeling on one side of the body, in whole or in part (hemisensory disturbance)	Carotid artery territory, posterior circulation
Vestibular function	Spinning sensation (vertigo). Isolated vertigo is more likely to be due to a peripheral (labyrinthine) cause	Posterior circulation
Vision	Visual loss in one (amaurosis fugax) or both eyes. Bilateral loss may indicate onset of basilar ischaemia	Retinal artery
	Loss of vision in the left or the right half or quarter of the visual field (hemianopia, quadrantanopia)	Carotid artery territory, posterior circulation
	Double vision (diplopia)	Brainstem
Speech/language	Difficulty comprehending, pronouncing or 'finding' words (aphasia), difficulty reading (alexia) and writing (agraphia)	Left carotid artery territory
	Slurred speech (dysarthria)	Brainstem, carotid artery territory
Higher cortical function	Difficulty dressing or using tools – e.g. combing hair, cleaning teeth (apraxia)	Carotid artery territory
	Visual–spatial–perceptual dysfunction	Carotid artery territory
	Inattention to surrounding environment on one side (hemineglect)	Carotid artery territory (usually right)

- structural intracranial abnormalities (especially with acute haemorrhage).

A TIA is typically rapid in onset, and maximum intensity is usually reached within one minute. Distinguishing a TIA from migraine aura can be difficult. In general, migraine aura tends to have a more gradual onset and resolution and a marching quality. For example, symptoms such as tingling may progress from the finger to the forearm to the face within minutes. Associated headache, nausea and photophobia with a previous history of migraine are more suggestive of migraine than TIA.

In the absence of other focal neurological symptoms, loss of consciousness, fatigue or light-headedness is unlikely to represent TIA. Syncope and seizures should be considered in patients with loss of consciousness.

TIA referral guidelines using the ABCD^2 score

If the clinical features of a TIA have resolved by the time the patient is first seen, the seven-point ABCD^2 score is a validated model to predict the early risk of stroke after this event (Table 2).[3] This can help the front-line physician identify which patients with a suspected TIA should be referred for emergency assessment.

In a cohort of 4799 patients with TIA assessed with the ABCD^2 scoring system, the two-day stroke risk was 1% in patients who had a score of 0 to 3 (low risk), 4.1% in those with a score of 4 or 5 (moderate risk) and 8.1% in those with a score of 6 or 7 (high risk).[3]

Patients with a score of 4 or greater should be sent to an emergency department or to a rapid referral TIA clinic for urgent assessment. Those classified as being at high risk are likely to benefit from urgent inpatient evaluation, treatment and observation, particularly in view of the high social and economic costs of stroke. On the other hand, most patients with a score of less than 4 will not need hospital observation. Ideally, these patients should be assessed at a routine stroke/TIA clinic or by a stroke physician, preferably within a week.

Initial investigations

Initial investigations are often carried out at the emergency department.

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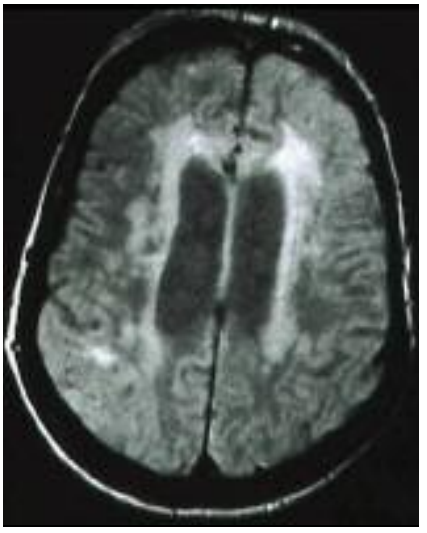

Figure. FLAIR MRI image showing white matter changes with acute cerebral infarction in the right posterior parietal lobe.

Table 2. ABCD[2] score for two-day risk of stroke after a TIA[3]

	Risk	Score
A	Age 60 years or greater	1
B	Blood pressure: systolic greater than 140 mmHg and/or diastolic 90 mmHg or greater	1
C	Clinical features:	
	• Unilateral weakness	2
	• Speech disturbance without weakness	1
	• Other	0
D	Duration of symptoms:	
	• 60 minutes or more	2
	• 10 to 59 minutes	1
	• Less than 10 minutes	0
D	Diabetes mellitus	1

Patients who score 4 or greater should be sent immediately to the emergency department; those scoring less than 4 should be assessed, preferably within a week, at a routine stroke/TIA clinic or by a stroke physician.

Laboratory tests

A complete blood count with platelet count should be obtained to rule out polycythaemia, thrombocytopenia, and thrombocytosis.

It is important to determine the prothrombin time (PT), activated partial thromboplastin time (aPTT) and international normalised ratio (INR) before antiplatelet or anticoagulation therapy is administered. The PT and aPTT can be prolonged and INR elevated in hypercoagulable states.

The glucose level should be determined to rule out hypo- or hyperglycaemia.

Creatinine levels are relevant, because poor renal function may prohibit the use of contrast media in imaging.

An elevated erythrocyte sedimentation rate may indicate rare causes of cerebral ischaemia such as bacterial endocarditis or temporal arteritis.

Electrocardiography and echocardiography

A 12-lead electrocardiogram (ECG) is important for detecting atrial fibrillation as well as concurrent cardiovascular disease. If the ECG is unrevealing, cardiac monitoring may help diagnose paroxysmal atrial fibrillation. Echocardiography may identify a thromboembolic source or left ventricular systolic dysfunction, both of which are predictors of ischaemic stroke.

Transoesophageal echocardiography is superior to transthoracic echocardiography for evaluating dysfunction of the left atrium, intracardiac thrombus, patent foramen ovale and atrial septal defects, and aortic arch plaque. This is usually performed in an elective setting.

Brain imaging

All patients with a suspected TIA should undergo cranial imaging by CT or MRI on the same day as their presentation to rule out structural causes, such as intracerebral haemorrhage, subdural haematoma or brain tumour (Figure). Urgent identification of these conditions is critical because special management or neurosurgical intervention may be required. A head CT scan can also identify signs of early infarction or evidence of previous strokes. MRI is more sensitive than CT for detection of early ischaemia, especially in the brain stem because of increased bony artefact in the posterior fossa affecting CT.

Neurovascular imaging

Imaging of the extracranial circulation is an important component of the diagnostic workup. Patients with anterior circulation ischaemia should have carotid imaging, usually ultrasonography, within 24 hours. The presence or absence of a neck bruit on clinical examination should not alter the decision to image the carotid bifurcation, since this finding is neither sensitive nor specific.

The discovery of severe carotid stenosis (greater than 70%) in patients who present after a TIA is an important finding because there is good evidence that endarterectomy is superior to medical management in this group of patients. Endarterectomy is most beneficial if performed within two weeks of the patient's last symptoms.[5]

Carotid ultrasonography is a good screening tool, but may not provide enough information on its own for surgical decision making.[6]

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Often CT angiography or MR angiography and occasionally conventional catheter angiography are used to confirm the ultrasound findings in patients with estimated stenosis of more than 70% before proceeding to surgery. CT angiography or MR angiography of the vertebral and intracranial arteries may reveal stenosis or dissection. Evaluation of the intracranial cerebral circulation may be important in selected patients, but in general it is unlikely to alter management decisions for the patient presenting with a TIA. Catheter angiography should be reserved for patients being considered for surgery or in whom diagnostic uncertainty remains after noninvasive imaging.

Case continued

Mr L had an ABCD^2 score of 5 and was referred to the emergency department by his GP. He was found have normal sinus rhythm, a blood pressure of 150/85 mmHg, normal routine blood tests and a normal head CT scan. Carotid duplex ultrasonography revealed a 90% stenosis of the left internal carotid artery.

Aspirin therapy was commenced, and an uncomplicated endarterectomy was performed three days later.

At follow-up four weeks later he was found to be hypertensive, and his LDL-cholesterol level was high. He was started on an ACE inhibitor with a diuretic and a cholesterol-lowering agent, and aspirin was changed to combination aspirin/dipyridamole therapy.

Conclusion

The short-term risk of stroke in patients after a TIA is high and relatively predictable. Emergency evaluation and treatment in patients such as the one described in the clinical vignette is justified.

TIA provides an important opportunity for the clinician to intervene and prevent stroke. Effective interventions for secondary stroke prevention exist but need to be implemented rapidly to be effective. The most important step in the prevention of disability from stroke is the early recognition of TIA by patients and their physicians. All patients with an ABCD^2 score of 4 or greater should be sent to an emergency department for urgent evaluation, whereas most patients with a score of less than 4 can complete their workup as outpatients. MT

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DECLARATION OF INTEREST: None.

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