



Early management of acute stroke

Richard I. Lindley, Moran Foundation for Older Australians Professor of Geriatric Medicine, Western Clinical School, University of Sydney; and Peter B. Landau, Senior Staff Specialist, Director, Stroke Unit, Department of Geriatric Medicine, Westmead Hospital, Westmead, New South Wales

Summary

Most patients with a stroke or a transient ischaemic attack require urgent imaging to determine the cause of their symptoms and to guide treatment. Stroke unit care, where available, can facilitate effective use of acute treatments (aspirin and thrombolytic therapy), good multidisciplinary care and early secondary prevention. Implementation of these strategies will have a significant public health impact.

Key words: aspirin, thrombolytic therapy, transient ischaemic attack.

(Aust Prescr 2004;27:120–3)

Introduction

In Australia nearly 50 000 people have a stroke each year. A third will die within a year, and a third are left with significant disability. The cost is considerable, whether measured in dollars (5% of the total health budget), social care or by the impact on the families and carers. In the past decade there has been a major change in how stroke is perceived and managed, especially in the acute phase.

Is it a stroke or TIA?

There is now an interesting problem with the nomenclature of stroke and transient ischaemic attack (TIA). By definition (see box) a stroke either leads to death or is still symptomatic 24 hours later, while a TIA resolves to leave no symptoms at 24 hours. These definitions have been essential for epidemiological studies and they are useful to remind clinicians of the differential diagnoses (Table 1).

Stroke

A clinical syndrome characterised by rapidly developing clinical symptoms and/or signs of focal, and at times global (applied to patients in deep coma and those with subarachnoid haemorrhage), loss of cerebral function, with symptoms lasting more than 24 hours, or leading to death, with no apparent cause other than that of vascular origin.¹

Transient ischaemic attack

A clinical syndrome characterised by an acute loss of focal cerebral or monocular function with symptoms lasting less than 24 hours and which is thought to be due to inadequate cerebral or ocular blood supply as a result of low blood flow, arterial thrombosis or embolism associated with diseases of the arteries, heart or blood.²

Table 1

Important or common mimics of 'brain attack'

Mimics of transient ischaemic attacks (shorter attacks)

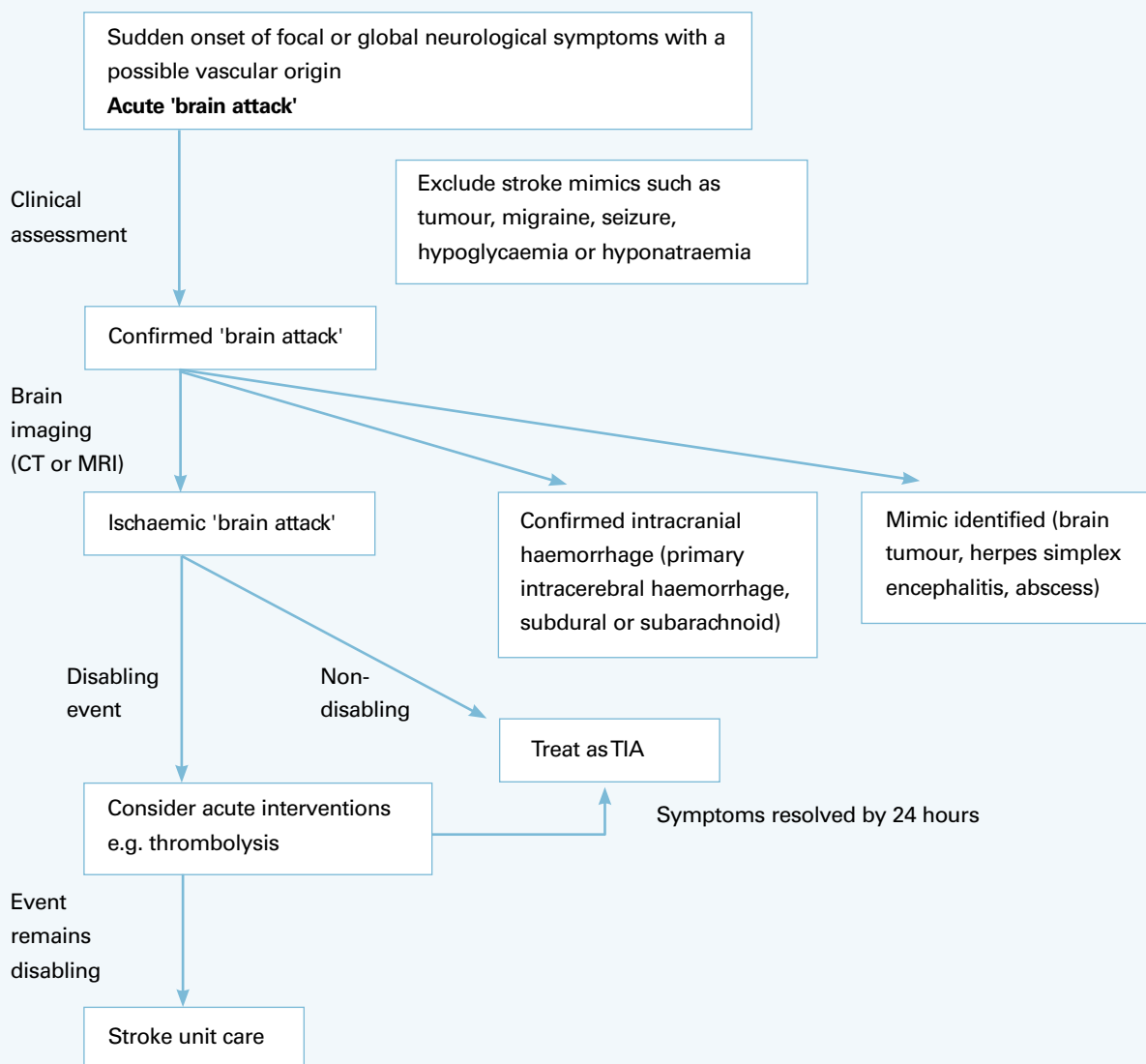
Migraine
Partial seizures
Hypoglycaemia
Brain tumour and other space-occupying lesions
Benign paroxysmal positional vertigo
Hyperventilation
Panic attacks
Transient global amnesia
Medically unexplained (e.g. somatisation)

Mimics of stroke (longer attacks)

Brain tumour and other space-occupying lesions
Todd's paresis
Previous stroke and new non-stroke illness
Bell's palsy or other mononeuropathy
Multiple sclerosis
Vestibular neuronitis
Delirium
Hepatic encephalopathy
Hyponatraemia
Head injury
Medically unexplained (e.g. somatisation)

Fig. 1

A flow diagram for the assessment of patients with suspected stroke or transient ischaemic attack



The definitions obscure the fact that TIA and ischaemic stroke are actually the same disease. New interventions for some strokes will change the natural history, resulting in a TIA (that is, symptoms resolved by 24 hours). In order to increase the priority given to patients with a suspected stroke it has been suggested that all TIAs and strokes should be called 'acute brain attacks' in the first 24 hours. The final diagnosis of stroke or TIA can be made when the situation is clearer 24 hours later (Fig. 1).³

No matter what we call the attack, the immediate medical priority is to distinguish attacks that are vascular from the numerous non-vascular causes. The commonest mimics of stroke are seizure (and the resulting Todd's paresis), migraine (the presence of positive symptoms is often a clue) and brain tumour. A variety of miscellaneous neurological and other conditions can also be misdiagnosed as strokes. Diagnostic uncertainty is common, for example, about a third of patients referred to a TIA/stroke clinic have non-vascular disease.⁴

What is the cause of the 'brain attack'?

When clinicians ask the right question, a logical process of investigations should follow. A young man presenting with a stroke while swimming needs cardiac imaging to exclude a right-to-left shunt (was this a paradoxical embolism from a patent foramen ovale?) and carotid imaging (was this a carotid dissection?). An 80-year-old with extensive vascular disease may require an ECG, carotid duplex scan and transthoracic echocardiogram in addition to computed tomography (CT) and the usual blood tests. A patient with a fever needs blood cultures to exclude bacterial endocarditis.

Imaging and pathology of the 'brain attack'

Computed tomography is the most reliable way to identify intracranial haemorrhage as the cause of the 'brain attack'. Blood shows up immediately as a high attenuation lesion on CT and remains visible for the next four to seven days. After this time

CT can be unreliable as the blood becomes isodense and then the lesion appears as a low attenuation lesion mimicking an ischaemic area.

Magnetic resonance imaging (MRI) can be used if patients present late, as haemosiderin from a haemorrhagic stroke can be identified by special MRI sequences (such as gradient echo T2 or Flash 2D sequences). However, old blood due to early haemorrhagic transformation of a cerebral infarction will also be identified by this technology.

The key point is that a CT scan is needed as soon as possible to identify the pathology. This is also the most cost-effective strategy. Early pathological diagnosis helps determine the investigations required, for example a carotid duplex scan is not required for a stroke due to primary intracerebral haemorrhage.

A CT scan for a straightforward single TIA is not always required. If the 'brain attack' has completely resolved within hours, it is a definite TIA; a haemorrhage is very unlikely (less than 1% chance) so the event can be considered ischaemic. While some TIAs are caused by space-occupying lesions, the patients generally have unusual symptoms or multiple attacks.

Who to admit?

Admission to hospital depends on numerous factors:

- the need for CT or other investigations
- comorbidity and the need for assistance with personal care
- the presence of new disability
- the suspicion of a serious underlying problem such as bacterial endocarditis.

Most experts recommend admission to a stroke unit but there may be circumstances when this would not be in the patient's best interest. For example, a demented patient with a history of a recent gastrointestinal bleed, living in a nursing home, may be better off staying in the nursing home, rather than having the unsettling experience of hospital admission. The patient is ineligible for any antithrombotic treatment so a CT scan would be of little help.

General management

There is now overwhelming evidence that care in a stroke unit gives the patient the greatest chance of independent survival. For every 100 patients treated there will be 40 independent survivors with general non-specialist care and 46 independent survivors in a stroke unit. This gain (six more independent survivors) is two to three times the treatment effect of thrombolysis for acute myocardial infarction.⁵

The benefit is due to the cumulative effect of numerous small interventions performed by an interested specialised multi-disciplinary team of nurses, allied health staff and doctors, who are enthusiastic, well trained and participate in continuing professional development. These interventions include appropriate hydration, very early mobilisation, thorough investigation of the cause of

stroke, rehabilitation, attention to swallowing and oral diet and prevention of deep venous thrombosis.

Stroke unit care is standard in many countries (for example Norway), has been mandated in others (for example England and Wales) but has been slow to be developed in Australia. In the absence of a local stroke unit, a rehabilitation unit would be a suitable alternative, once the patient is medically stable.

Medical treatment

Drug treatments include aspirin, thrombolytics and antihypertensives. Anticoagulation, neuroprotection, steroids, mannitol and glycerol have been shown to be ineffective or harmful.

Aspirin

An immediate dose of aspirin for patients with ischaemic stroke is definitely effective, but the effect is modest with only one extra independent survivor per 100 patients treated.⁶ However, across Australia, this treatment will prevent 400–500 people from dying or becoming dependent each year. Putting it another way, the benefit of aspirin given for two weeks following an ischaemic stroke is the same as the benefit seen in the subsequent 50 weeks, for routine secondary prevention. This is due to the clustering of recurrent ischaemic stroke in the first few weeks after a stroke or TIA.

Thrombolysis

Recombinant tissue plasminogen activator (rt-PA) has a treatment effect of about 10–15 extra independent survivors per 100 patients treated, despite an additional eight patients with symptomatic intracranial haemorrhage.⁷ There is an uncertain effect on overall deaths and treatment is not easy to deliver. In Australia, rt-PA was approved by the Therapeutic Goods Administration in 2003, but only for patients who can be assessed, scanned and treated by experienced stroke teams within three hours of onset. As it can be difficult to meet this deadline, trials are underway to broaden the indications and to evaluate whether treatment given within six hours is safe and effective. The current use of thrombolytic drugs in stroke management is unlikely to have a public health impact, but may help some patients.

Reducing blood pressure

The effect of lowering blood pressure in the acute phase of stroke is uncertain. Some experts prefer to treat patients with sustained very high blood pressure (greater than 180/120), particularly patients with primary intracerebral haemorrhage, but there is no reliable evidence behind this recommendation. Trials are needed to test this potentially widely generalisable treatment. If treatment is considered necessary, oral therapy with usual antihypertensives can be given. For dysphagic patients, oral treatment can be given by nasogastric tube or parenterally.

Reversal of antithrombotic treatment

Antiplatelet therapy should be stopped, probably indefinitely, if the stroke is due to primary intracerebral haemorrhage. If anticoagulated patients have a primary intracerebral haemorrhage they need urgent assessment, and haematological advice should be sought to reverse the anticoagulation. It is probably wise not to re-anticoagulate unless the patient has a major ongoing risk of thromboembolism (such as metal prosthetic heart valves).

Surgical treatment

Occasionally surgery is necessary to relieve acute hydrocephalus caused by a posterior fossa stroke. Hemicraniectomy (removal of a large skull flap) can occasionally save life by reducing intracranial pressure secondary to massive hemispheric swelling and is now the subject of a randomised controlled trial. The results of a trial of early evacuation of the haematoma for those with primary intracerebral haemorrhage (the STICH trial) did not show any benefit of surgical intervention and full publication is awaited.

Support for patients and carers

Stroke can be a devastating problem for patients and carers and they need support and information during this critical time. Assessment of stroke severity and subtypes can help give estimates of prognosis. For example, patients presenting with a hemiparesis, hemianopia and aphasia have a 90–95% chance of being dead or dependent at six months, a prognosis worse than most cancers. Patients with a lacunar syndrome (for example, pure motor stroke) have a low case fatality, but a 30% chance of longer-term disability. Some matters, such as advice about driving, have important medicolegal implications.

Terminal care

About 10% of patients with ischaemic stroke and 50% of those with primary intracerebral haemorrhage die within a month. Appropriate terminal care is therefore an important part of acute management.

Secondary prevention

The best opportunity to commence secondary prevention is in the acute phase. Stopping smoking should halve the future risk of vascular events. Antithrombotic therapy together with cholesterol and blood pressure lowering are effective for patients with ischaemic stroke.^{8,9,10} This evidence-based polypharmacy may be a problem for some patients. Secondary prevention for primary intracerebral haemorrhage depends on effective blood pressure lowering.⁸

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Professor Lindley

The University of Sydney has received donations from several pharmaceutical companies in lieu of payment for lectures and attendance at an advisory committee by Professor Lindley (Servier, Bristol Myers Squibb). He has accepted sponsorship to attend scientific symposia in 2004 (Bristol Myers Squibb, Sanofi). He holds no shares or interests in any company.

He is the co-principal investigator of an academic trial evaluating recombinant tissue plasminogen activator for acute ischaemic stroke.

Dr Landau

Dr Landau is the co-principal investigator in several pharmaceutical company sponsored clinical trials. He holds no shares or interests in any pharmaceutical companies.

Self-test questions

The following statements are either true or false (answers on page 133)

5. If 100 patients with acute strokes are given aspirin there will only be one additional survivor.
6. Most patients with a suspected acute stroke should have urgent computed tomography of the brain.