Stroke in the acute setting

Keith W Muir

Abstract

Acute stroke and transient ischaemic attack (TIA) are focal neurological syndromes of vascular origin and should be treated as medical emergencies. Brain imaging with computed tomography or magnetic resonance imaging is required to distinguish ischaemic stroke from intracerebral haemorrhage, recognize non-stroke pathologies that mimic stroke and guide investigation into the underlying mechanism. Acute interventions of benefit in ischaemic stroke include intravenous thrombolysis with alteplase given within 4.5 hours of onset, endovascular thrombectomy within 6 hours of onset in selected patients, stroke unit care and aspirin. Decompressive hemicraniectomy reduces mortality in ischaemic stroke complicated by severe brain swelling. Intracerebral haemorrhage accounts for 10–15% of strokes, and while specific treatments are lacking at present, patients benefit from general measures, notably stroke unit care. TIA carries a high short-term risk of stroke, and immediate investigation and institution of secondary preventive treatment prevents a high proportion of this. Secondary prevention for ischaemic stroke and TIA should be tailored according to mechanism in individual patients.

Keywords Acute treatment; cerebrovascular disease; intracerebral haemorrhage; stroke; thrombectomy; thrombolysis

Key points

- Acute ischaemic stroke is common and disabling
- Stroke unit care improves outcome, and all patients should be admitted to a specialist stroke service
- Prompt recognition of stroke requires awareness of clinical patterns and radiological features
- Treatment with intravenous thrombolytic drugs within 4.5 hours improves the odds of disability-free recovery; the earlier treatment is initiated, the greater the effect
- Additional endovascular mechanical thrombectomy using stent retrievers is highly effective in selected patients with occlusions of the intracranial internal carotid or middle cerebral artery
- In minor ischaemic stroke and transient ischaemic attack, early institution of secondary prevention reduces the incidence of recurrent stroke
- Intracerebral haemorrhage currently lacks evidence-based treatments beyond stroke unit care, but platelet transfusion is not helpful in antiplatelet-associated intracerebral haemorrhage

Definition

Stroke is a clinical syndrome defined by an acute focal neurological deficit with a vascular basis. Around 85–90% of strokes are ischaemic (caused by arterial occlusion), and 10–15% result from intracerebral haemorrhage (ICH). The 1976 World Health Organization definition also includes subarachnoid haemorrhage, but this is primarily of epidemiological interest. The term 'transient ischaemic attack' (TIA) conventionally denotes complete resolution of all symptoms within 24 hours, but this arbitrary time limit is probably an anachronism in light of modern imaging and reperfusion treatment.

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Epidemiology

Stroke incidence increases with age, although a quarter occur in patients <65 years of age. Causes vary by age group. Ischaemic stroke is the most common, with TIA next and ICH least common. There are approximately 150,000 incident strokes annually in the UK, and globally stroke is the third most common cause of death and most common disabling neurological disease.

Risk factors

Major predisposing factors for ICH and acute ischaemic stroke (AIS) are listed in Table 1. It is important to investigate the underlying mechanism in most individuals, regardless of risk factors.

Diagnosis and natural history

Presentations of AIS and ICH are similar, and the two cannot be distinguished without brain imaging. Symptoms are of sudden onset and usually maximal in severity at, or within minutes of, onset. Evolution of new neurological deficits or reduced level of consciousness is uncommon within the first few hours, but some deficits, notably lacunar strokes ('capsular warning syndrome') and incipient carotid occlusion, can fluctuate dramatically in severity.

Symptoms of TIA are identical to those of ischaemic stroke but can include transient monocular blindness. TIA is

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Major risk factors for stroke

Modifiable	Unmodifiable
Intracerebral haemorrhage	
Hypertension	Age
Alcohol excess	Apolipoprotein E ε2 or ε4 carriage (CAA)
Drug treatments — thrombolytic agents, anticoagulants, antiplatelet agents	Race (probably higher in South-East Asian populations)
Diabetes mellitus	
Cigarette smoking	
Ischaemic stroke	
Hypertension	Age
Diabetes mellitus (or elevated HbA1c)	
Ischaemic heart disease	
Atrial fibrillation	
Valvular heart disease	
Cigarette smoking	

Dietary risks (high sodium, low fruit and vegetables).
Low physical activity.

Table 1

distinguished only by complete resolution, typically within 30–60 minutes: the longer the symptoms last, the higher the probability of brain infarction (30% of TIAs resolving within 24 hours have infarcts on diffusion-weighted magnetic resonance imaging (MRI) — DWI).

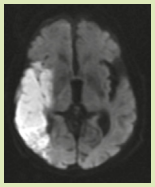
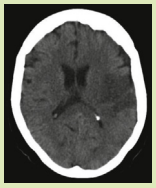
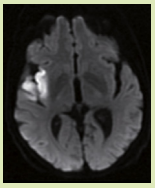
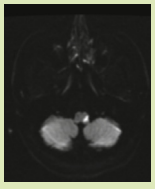
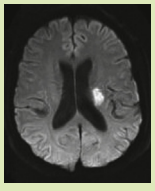
Clinical features

Common clinical patterns are summarized by the Oxfordshire Community Stroke Project (OCSP) classification (Figure 1). A more detailed approach to relevant clinical examination is provided by the National Institutes of Health Stroke Scale (NIHSS; Table 2), for which online training is available.

Contralateral hemiparesis can involve face, arm and leg equally (internal capsule, corticospinal tract) or be more focal (motor cortex), often face- and arm-predominant (typical of the distal middle cerebral artery (MCA) cortical territory) (Figure 2). Hemisensory disturbance is similarly distributed. Severity of weakness is not a reliable indication of stroke severity or prognosis: the presence of other neurological deficits and pattern of weakness are of more relevance.

Conjugate gaze deviation (away from the affected limbs) results from involvement of the frontal eye field, a bilaterally represented centre that directs voluntary gaze. Often incorrectly

OCSP classification: syndromes and imaging examples

OCSP term	Clinical features	Vascular basis	Example CT	Example MRI
Total Anterior Circulation Syndrome (TACS)	• Hemiparesis **AND** • Higher cortical dysfunction (dysphasia or visuospatial neglect) **AND** • Homonymous hemianopia	Usually proximal middle cerebral artery (MCA) or ICA occlusion		
Partial Anterior Circulation Syndrome (PACS)	• Isolated higher cortical dysfunction **OR** • Any two of hemiparesis, higher cortical dysfunction, hemianopia	Usually branch MCA occlusion		
Posterior Circulation Syndrome (POCS)	• Isolated hemianopia (posterior cerebral artery (PCA)), brainstem or cerebellar syndromes	Occlusion of vertebral, basilar, cerebellar or PCA vessels		
Lacunar Syndrome (LACS)	• Pure motor stroke **OR** • Pure sensory stroke **OR** • Sensorimotor stroke **OR** • Ataxic hemiparesis **OR** • Clumsy hand-dysarthria	Small penetrating artery occlusion, usually in lenticulostriate branches of MCA, or supply to brainstem or deep white matter		

CT, computed tomography; ICA, internal carotid artery; MRI, magnetic resonance imaging

Figure 1

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