The management of ischaemic stroke

Robin S Howard

Abstract

Ischaemic stroke often leads devastating long-term neurological sequelae. There are five interventions that improve the outcome after a stroke: management within a stroke unit, intravenous thrombolysis, mechanical clot retrieval, aspirin within 48 hours, and decompressive hemicraniectomy for malignant middle cerebral artery (MCA) stroke. The benefits of intravenous thrombolysis up to 4.5 hours are now well established, but the recent development of clot retrieval has radically altered the acute management of stroke. The development of late complications remains the most important factor determining outcome and it is essential to ensure homoeostasis is maintained during the acute phase of care to reduce the risk of late deterioration.

Keywords Clot retrieval; ischaemic stroke; thrombolysis

Royal College of Anaesthetists CPD Matrix: 2F01

Learning objectives

After reading this article, you should be able to:

- describe the initial assessment and managemant of acute ischaemic stroke
- identify patients eligible for intravenous thrombolysis
- describe the criteria for endovascular intervention in stroke patients

Introduction

Stroke is defined as 'rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer, or leading to death, with no apparent cause other than of vascular origin.'

Stroke is a major public health problem, being the third most common cause of death after myocardial infarction and cancer, and the leading cause of adult disability. Stroke accounts for 9% of deaths in England and Wales. The incidence of stroke has fallen over the last 10 years and prevalence increased, as a result of more effective primary prevention and treatment.

Ischaemic stroke is the result of vessel occlusion from *in situ* thrombosis, embolism or haemodynamic failure. Embolism may be from artery to artery (30–40%) or from the heart (30–40%). In 25% of cases, disease of the walls of small penetrating intracranial blood vessels is responsible for lacunar infarction.

Causes and risk factors

The risk factors for ischaemic stroke are summarized in Box 1.

Clinical syndromes of cerebral ischaemia

Transient ischaemic attacks: the symptoms of transient ischaemia are usually negative, maximal at onset and last typically for a few to 30 minutes. TIAs are associated with a high rate of subsequent stroke: one-third of all untreated patients subsequently have a stroke. The immediate risk is high: 20% of strokes occur within a month and 50% within a year. The differential diagnosis of TIA is summarized in Box 2.

Lacunar stroke: lacunes are small, subcortical or brainstem infarcts ranging from 1 to 15 mm in size. They are caused by occlusion of small penetrating vessels most commonly arising from the MCA and basilar artery.

Large vessel occlusion: the main clinical distinction between large vessel occlusion and lacunar infarction in the carotid circulation is the presence of cortical signs (eye deviation, dysphasia, neglect syndromes, hemianopia). The hemiparesis produced by MCA occlusion affects the arm more than the leg.

Internal carotid artery disease – symptoms are produced either by embolism, thrombus formed on ulcerated plaque, haemodynamic failure from severe stenosis or carotid artery occlusion.

Total MCA – the MCA may be affected by embolism or thrombosis in situ. If the main trunk occludes then the whole territory infarcts with conjugate eye deviation (frontal lobe damage), aphasia (dominant hemisphere), hemiplegia, hemisensory loss and hemianopia (parietal and temporal lobe damage). Patients with complete MCA syndromes occasionally develop fatal brain swelling (malignant MCA oedema) within 48 hours of onset, leading to death from coning. In appropriate cases surgical decompression is required.

MCA branch – upper branch occlusion affecting frontal structures produces hemiparesis, hemisensory loss, ocular deviation and non-fluent motor dysphasia (expressive).

Anterior cerebral artery (ACA) – this territory is rarely affected, ACA occlusion leads to contralateral hemiplegia with the lower limb predominantly affected.

Posterior cerebral artery (PCA) – commonly embolic and causes hemianopia and neglect syndromes. Involvement of the thalami and posterior-medial temporal lobeslead to confusion, dysphasia (thalamic) or memory impairment (thalamic or temporal). If both PCA territories are infracted, as may happen when an embolus lodges at the top of the basilar, cortical blindness and confusion ensues.

Vertebral artery – occlusion of the vertebral arteries causes infarction of the dorsolateral medulla leading to lateral medullary syndrome. This results in a Horner's syndrome, dissociated (temperature and pain) sensory loss on the ipsilateral side of the face and the opposite side of the body, nystagmus, ataxia of the ipsilateral limbs, and ipsilateral palatal and vocal cord paralysis.

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Risk factors for ischaemic stroke

- Age
- Blood pressure
- Smoking
- Diabetes mellitus
- High total cholesterol and low-density lipoprotein (LDL)
- Alcohol consumption
- The presence of asymptomatic carotid disease of greater than 75%
- Illicit drugs
- Sickle cell disease
- Antiphospholipid antibodies and syndrome
- Cardiac disease
    - Atrial fibrillation
    - Sick sinus syndrome through atrial dysfunction may also result in embolism
    - Valvular heart disease
    - Rheumatic
    - Endocarditis on native or prosthetic valves
    - Myocardial infarction
    - Paradoxical embolus due to patent foramen ovale

Box 1

Differential diagnosis of TIA

- Migraine with focal symptoms
- Transient global amnesia
- Epilepsy
- Tumours
- Subdural haematomas
- Multiple sclerosis
- Hypoglycaemia
- Syncope

Box 2

Basilar artery – in the medulla, lower cranial nuclei may be affected giving rise to a bulbar or pseudobulbar palsy but above the medulla, pontine infarction can cause a gaze paresis, internuclear ophthalmoplegia, pinpoint pupils or 'locked-in' syndrome.

Management of acute stroke

In the first few hours after ischaemic stroke, measures are designed to

- restore blood flow (reperfusion)
- preserve the ischaemic penumbra (neuroprotection)
- prevent early recurrence (antiplatelet treatment).

Initial assessment: assessment depends on determining the history and excluding alternative causes. It is important to define the onset of symptoms and the progression of neurological deficit to establish whether the patient has had an ischaemic stroke and, if so, the localization and severity. A focused and detailed neurological examination facilitates anatomical localization, and thereby provides the basis for interpreting the imaging findings and identifying underlying causes.

Emergency treatment: the initial management of acute stroke focuses on stabilization of the airway, breathing and circulation, followed by an assessment of the neurological deficits and comorbidities to identify patients eligible thrombolysis and those at particular risk of the complications of acute stroke.

Admission to a stroke unit and multidisciplinary rehabilitation are vitally important as are complementary to medical treatments. Successful stroke unit care also pays critical attention to the prevention of complications and the prevention of recurrent stroke.

Investigation: emergency investigations including blood glucose, electrolytes and renal function, full blood count, cardiac markers, coagulation profile and ECG are essential.

CT or MRI scan should be performed immediately on admission of the patient to hospital. Early CT scan changes are seen in 60% within 6 hours of stroke onset (Figure 1). They include:

- cortical sulcal effacement
- loss of the insular ribbon
- blurring of the grey–white interface
- obscuration of lentiform nucleus
- hyperdense artery sign (intravascular thrombus).

A cerebral and/or neck CT angiogram/MR angiogram should be undertaken to exclude arterial deception and carotid or vertebral occlusive disease. Brain haemodynamic measurements with CT or MRI perfusion allow the identification of viable tissue at risk of infarction (penumbra). Diffusion-weighted imaging is the most reliable technique for detecting acute ischaemic stroke as early as 30 minutes from symptom onset. Conventional MRI sequences are most useful for assessing the extent and age of subacute and chronic infarcts.

Maintenance of homoeostasis

Initial supportive treatment is essential to improve functional outcome. This includes:

- Stabilization of airway, breathing and circulation.
- Oxygen saturation should be monitored routinely and hypoxaemia treated appropriately ($SatO_2$ >95%).
- Blood sugar levels are elevated in about one-quarter of all stroke admissions and elevated blood glucose on admission is a risk factor for haemorrhagic transformation of the acute infarct. The optimal level is 6–9 mmol/L and insulin may be necessary to achieve this.
- Blood pressure – elevated blood pressure should not be lowered acutely unless the patient is a candidate for thrombolysis, has hypertensive encephalopathy, malignant hypertension or the blood pressure readings are persistently above an arbitrary threshold of 220/120.
- Hypotension should be corrected promptly by raising the foot of the bed, fluid replacement and stopping hypotensive medication.
- Pyrexia should be treated aggressively.

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