Stroke and transient ischaemic attack

Stroke and transient ischaemic attack - I60-I64, G45 (Clinical term: Stroke and cerebrovascular accident unspecified G66)

Presenting complaints

- Usually sudden on-set focal symptoms and signs, eg left or right hemiparesis/hemisensory deficit, ataxia, dysphasia or hemianopia. The evolution may be stuttering over a few days or, rarely, longer.
- Dysarthria is very common but not of localizing value because it may occur with hemisphere or brainstem lesions.
- Some patients complain of headache at the time of stroke or leading up to it.

Transient ischaemic attacks (TIAs) have been arbitrarily defined as manifesting symptoms lasting less than 24 hours, and stroke more than 24 hours. However, it is the pathophysiology and not the timing that is important. The symptoms of a TIA should mimic a stroke. Isolated vertigo or amnesia is unlikely to be due to transient ischaemia or stroke. Transient monocular blindness is common in TIA associated with ipsilateral carotid atheroma, but retinal infarction (i.e stroke in the eye) is rare.

Diagnostic features

Stroke is a clinical syndrome usually taken to mean 'sudden-onset focal symptoms or signs secondary to vascular disease'. Physical signs usually reflect a lesion within a single vascular territory - hence the majority present with hemiparesis. It is not possible to distinguish infarction (90% of cases) from haemorrhage (10%) clinically, so neuroimaging is essential.

With ischaemic stroke within the carotid or basilar circulation, occlusion of a small penetrating vessel (lacunar infarction) will usually result in one of the following:

- pure motor hemiparesis
- pure hemisensory deficit
- motor/sensory deficit
- ataxic hemiparesis
- dysarthria - clumsy hand syndrome.

Cortical signs imply larger vessel disease (eg middle cerebral artery occlusion):

- dysphasia in the dominant hemisphere
- neglect syndrome in either the dominant or more frequently non-dominant hemisphere
- hemianopia in either.

In large-vessel occlusion these cortical signs are usually seen in conjunction with hemiplegia, but may be isolated, eg sudden onset isolated acute hemianopia or dysphasia are very likely to be due to stroke.

Haemorrhage mimics any of the ischaemic stroke syndromes and there are no definite discerning features.

The vast majority of patients have underlying vascular risk factors. Hypertension is the single most important of these; others include ageing, diabetes and smoking. Hypercholesterolaemia may be present. Atrial fibrillation and carotid stenosis are also very important and need specific identification.

Differential diagnosis

Stroke is a syndrome and not a diagnosis; within the umbrella of 'stroke' the following distinctions are usually made:

- infarct or haemorrhage
- small-vessel disease, large-vessel disease or embolism from the heart
- profile of risk factors leading to the above.

Although most strokes are the result of atherosclerosis or heart disease, rare causes of stroke need to be considered if the history is appropriate, eg:

- bacterial endocarditis
- meningitis
- dissection of carotid or vertebral artery
- venous sinus thrombosis.

In addition, 'mimics' of stroke that may also present with sudden-onset focal signs and need to be excluded by neuroimaging, eg:

- subdural haematoma
- intracranial tumour
- inflammatory lesions of the brain.

Essential information for patient and family

- Stroke is the result of a localized area of brain damage due to occlusion or rupture of a blood vessel.
- Predicting prognosis at the outset is difficult.
- Hospital treatment focuses first on preventing secondary brain damage and then planning a goal-orientated rehabilitation programme.
- Rehabilitation units focus on the needs of the individual patient.
- Attention will need to be paid to the environment of a person with complex impairments.
- Secondary prevention lowers the risk of future stroke greatly but does not eliminate it; treating and monitoring hypertension is paramount in this regard.
- Depression is common following stroke.

General management and advice to patient and family

- Immediate diagnosis, treatment and rehabilitation are fundamental for improved outcomes.
- Vascular risk factors need aggressive management. Although treatment of hypertension should include lifestyle measures, this is rarely enough for secondary prevention. A similar approach is needed for lipids with diet, but the evidence is for drug treatments.
- Smoking and excessive alcohol are serious risk factors that patients need careful detoxification from with the help of formal programmes and drug treatments.

- Risk factors need long-term control - it does not matter if the blood pressure is a bit high one day; it is the long-term reduction that is beneficial.
- With risk factor control the incidence of further stroke is approximately halved but remains at 2-10% per year.
- Driving should cease for at least 1 month following stroke or TIA. Other than for LGV/PCV licences, there is no need to inform the DVLA unless there is residual neurological deficit (ref 3). Hemianopia contraindicates driving, and patients will need a formal ophthalmic assessment if in doubt - this can become a cause of much doctor/patient disharmony.

References

3 Driver and Vehicle Licensing Agency. At a Glance Guide to Medical Aspects of Fitness to Drive. URL http://www.dvla.gov.uk. Further information is available from The Senior Medical Adviser, DVLA, Driver Medical Unit, Longview Road, Morriston, Swansea SA99 ITU, Wales.

Medication

(ref 229, 230)

Prompt treatment improves prognosis. Virtually all patients with stroke will need vascular risk factor control using medication.

Antihypertensives: Hypertension remains the most important risk factor for haemorrhage and infarction.

- It should not generally be treated immediately after stroke. Only malignant hypertension is treated acutely.
- Secondary prevention usually begins at about 2 weeks following stroke, aiming for a blood pressure <140/80 mmHg at all times.
- After transient ischaemic attack, if there is no immediate access to specialist assessment, antihypertensive medication can be started at once.
- The optimal first-line medication is a long-acting ACE inhibitor combined with a diuretic. However, it is likely that the most important action is to lower the blood pressure, with no definite evidence of a class-specific effect. (ref 231)
- Lowering so-called 'normal' blood pressure after stroke may also be beneficial. (ref 231)

Antiplatelet treatment: Usually given to all those with ischaemic stroke both acutely and for secondary prevention. There is no definite evidence that giving aspirin (300 mg stat) acutely is detrimental to those with intracranial haemorrhage; (ref 232) however, most defer until the results of neuroimaging. There are three main strategies for antiplatelet use in secondary prevention:

- aspirin alone (75 mg od)
- aspirin combined with slow-release persantin (Asasantin Retard 200 mg bd)
- clopidogrel (75 mg od)

Aspirin alone is first line, with clopidogrel usually reserved for those with aspirin intolerance and increasingly in those who have a further vascular event while on aspirin (so called aspirin failure). Combination aspirin with persantin may be of greater benefit than aspirin alone,(ref 233) but use of this combination first line is variable, some preferring to add persantin in the event of 'aspirin failure'. Persantin alone is rarely used. Clopidogrel as monotherapy or combined with aspirin is being used increasingly in high-risk patients and those with concurrent ischaemic heart disease (ref 234) (BNF section 2.9)

Statins: Beneficial in secondary prevention whatever the level of blood cholesterol. Current practice is to start a statin in almost everyone following an ischaemic stroke or TIA (ref 235)

Others: Warfarin is reserved for patients with atrial fibrillation and some other cardiac conditions.

Antiplatelet treatment and statins are not monitored beyond ensuring normalization of cholesterol levels, if raised. Treatment of hypertension needs long-term and sometimes intensive short-term monitoring and titration. Patients after stroke need medication in addition to lifestyle measures. Patients need to understand that this triple approach is additive and that the reduction in risk afforded is long term and not from day to day.

References

229 Gubitz G, Sandercock P. Stroke management. Clin Evidence 2002; 8: 169-183. (AI) This systematic review in people with ischaemic stroke found that giving aspirin (compared with placebo) within 48 hours of stroke onset significantly reduces death or dependency at six months and significantly increases the numbers making a complete recovery. Specialist stroke rehabilitation units significantly reduce death or dependency after a median follow-up of one year compared with usual non-specialist care.

230 Clinical Evidence Writers on Stroke Prevention. Stroke prevention. Clin Evidence 2002; 8: 184-208. (AI) Antiplatelet treatment reduces the risk of serious vascular events in people with previous stroke or transient ischaemic attack (TIA) compared with placebo or no antiplatelet treatment. Antihypertensive treatment reduced stroke among people with a previous stroke or TIA, whether they were hypertensive or not. Low-dose aspirin (75-100 mg) daily is as effective as higher doses in the prevention of serious vascular events.

231 PROGRESS Collaborative Group. Randomised trial of a perindopril-based blood-pressure-lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. Lancet 2001; 358: 1033-1041. (BII) This blood-pressure-lowering regimen reduced the risk of stroke among both hypertensive and nonhypertensive individuals with a history of stroke or transient ischaemic attack.

232 Sandercock P, Gubitz G, Foley P, Counsell C. Antiplatelet therapy for acute ischaemic stroke (Cochrane Review). In: The Cochrane Library, Issue 2, 2003. Oxford: Update Software. (AI) Nine studies were analysed. Antiplatelet therapy with aspirin at 160-300 mg daily, started within 48 hours of onset of presumed ischaemic stroke, reduces the risk of early recurrent ischaemic stroke without a major risk of early haemorrhagic complications and improves long-term outcome.

233 Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high-risk patients. Br Med J 2002; 324: 71-86. (Erratum appears in Br Med J 2002; 324: 141.) (AII) Aspirin (or another oral antiplatelet drug) is protective in most types of patient at increased risk of occlusive vascular events, including those with ischaemic stroke or previous stroke. Low-dose aspirin (75-100 mg) is an effective antiplatelet regimen for long-term use, but in acute settings an initial loading dose of at least 150 mg may be required. Adding a second antiplatelet drug to aspirin may produce additional benefits in some clinical circumstances, but more research into this strategy is needed.

234 Hankey GJ, Sudlow CLM, Dunbabin DW. Thienopyridine derivatives (ticlopidine, clopidogrel) versus aspirin for preventing stroke and other serious vascular events in high vascular risk patients (Cochrane Review). In: The Cochrane Library, Issue 2, 2003. Oxford: Update Software. (AI) Four studies were analysed. Thienopyridine derivatives are modestly but significantly more effective than aspirin in preventing serious vascular events in patients at high risk (and

specifically in transient ischaemic attack/ischaemic stroke patients), but there is uncertainty about the size of the additional benefit.

235 Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet 2002, 360: 7-22. (CII) Among the high-risk individuals studied, these antioxidant vitamins appeared to be safe. Although this regimen increased blood vitamin concentrations substantially, however, it did not produce any significant reductions in the five-year mortality from, or incidence of, any type of vascular disease, cancer or other major outcome.

Referral

- Ideally, all patients with stroke or TIA should be referred for specialist investigation and opinion.

- Most patients with acute stroke should be admitted without delay to a hospital with a stroke unit because:

  - intravenous thrombolytic therapy may be effective when given within 3 hours for selected patients.
  - correction and control of abnormal physiological parameters (hyperglycaemia, hyperpyrexia, hypoxia, hypotension, dehydration) may also improve outcome.
  - admission to a Stroke Unit reduces mortality and poor outcome, and
  - multidisciplinary rehabilitation improves outcome

- Patient with TIAs and mild strokes should have access to a rapid diagnostic and management service aimed at identifying the pathophysiology of the attack and correcting risk factors.
- Patients with multiple TIAs should be admitted urgently to hospital for further investigation.
- After investigation, the vast majority of vascular risk factor control can take place in a primary care setting, and the patient can be referred for further advice if deterioration occurs.
- Severe/uncontrolled hypertension should be managed by an appropriate specialist (eg stroke physician, clinical pharmacologist, renal physician).
- A thorough knowledge of local community and inpatient rehabilitation services is necessary to optimize patient care and ensure appropriate placement and ongoing care.

Resources for patients and families

Stroke Association 0845 3003 3100 (helpline)
Email: stroke@stroke.org.uk; website: http://www.stroke.org.uk
Provides a comprehensive series of information leaflets, including Stroke – Questions and Answers, Sex After Stroke, Cognitive Problems After Stroke, Stroke: a Carers Guide, and many others.

Different Strokes http://www.differentstrokes.co.uk
Provides free services to younger stroke survivors throughout the UK.

My Year Off by Robert McCrum. A personal account of recovery after stroke. Broadway Books, New York 1999.