

The patient presenting with acute hemiparesis

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Abstract

Acute hemiparesis is a common cause of presentation to hospital. In the majority of cases the cause is acute stroke, which is ischaemic in 80% of cases. This article aims to provide the reader with a practical approach to the initial management of suspected stroke. The problem-based format highlights some of the specific questions raised in the 2009 curriculum for training in Acute Internal Medicine, with reference to recent guidance from the National Institute for Health and Clinical Excellence (NICE).

Introduction

Hemiparesis can result from a lesion at any point along the motor system from the cerebral cortex, through the centrum semiovale, internal capsule, brainstem and pyramids and along the cervical spinal cord. Similarly sensory disturbance may arise from a lesion in peripheral nerves, spinal cord, brainstem, thalamus or primary sensory cortex. Parietal lesions in either hemisphere may give rise to visual and/or sensory inattention – the failure to attend to stimuli on either side of the body.¹

Acute stroke is the commonest cause of hemiparesis and is ischaemic in 80% of cases, with haemorrhagic stroke accounting for the remaining 20%. Ischaemic strokes are most commonly the result of cardioembolism, large vessel atherosclerotic disease (resulting in artery-to-artery embolism) and small vessel disease. The two commonest causes of spontaneous intracerebral haemorrhage include hypertensive vasculopathy, with bleeds typically arising within the basal ganglia; and lobar haemorrhages secondary to cerebral amyloid angiopathy, occurring mainly in the elderly. There is no reliable clinical method to distinguish between these two main stroke subtypes.

In the UK, the Oxfordshire Community Stroke Project (OCSP) has provided the most commonly used classification system for ischaemic stroke² (Table 1). It is simple to use and has been shown to have good inter-observer reliability. Further, it predicts the site and size of cerebral infarction shown on CT with reasonable accuracy³ and allows prediction of case fatality and functional recovery.

A Typical Case History

A 64 year old man is brought to the Emergency Department by ambulance having collapsed at a wedding ceremony one

Stroke type	Clinical features
Lacunar Infarct (LACI)	Pure motor/pure sensory/sensorimotor/ataxic hemiparesis ≥ 2 affected out of face/arm/leg No cortical dysfunction
Total anterior circulation infarct (TACI)	Hemianopia Cortical dysfunction e.g. dysphasia, inattention Limb motor and/or sensory abnormalities
Partial anterior circulation infarct (PACI)	Two of the features of TACI or Limb weakness/numbness more restrictive than LACI
Posterior circulation infarct (POCI)	Brainstem/cerebellar dysfunction or Isolated hemianopia

Table 1. The Oxford Community Stroke Project Classification system for stroke.

hour earlier. Witnesses reported that he slumped to the floor after attempting to stand and he appeared to be unable to move the right side of his body, although he did not lose consciousness. His face is asymmetrical with a droop to the right side of his mouth, and he is unable to speak or move his right arm or leg.

What key aspects of the history should be sought?

In patients with suspected stroke, the history is a key part of the clinical evaluation. Onset tends to be sudden, with loss of function the predominant feature rather than involuntary movements or visual/sensory phenomena. Timing of onset is crucial since this will determine the treatment options. Obtaining a witness account of the events leading up to the presentation is also important, particularly if the patient is aphasic, as in this case.

Improving symptoms may suggest a transient ischaemic attack (TIA). This is defined as an acute episode of focal loss of cerebral or visual function lasting less than 24 hours and attributed to inadequate blood supply. It may be unclear at the time of presentation to hospital whether the symptoms are going to resolve within 24 hours;

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Age ≥ 60 years	1 point
Age < 60 years	0 points
Blood pressure >140/90 mmHg at acute presentation	1 point
Clinical features, score 1 of: Speech disturbance without focal weakness	1 point
Unilateral weakness	2 points
Other	0 points
Duration of symptoms, score 1 of: < 10 minutes	0 points
10–59 minutes	1 point
≥60 minutes	2 points
Diabetes	1 point

Table 2. ABCD2 Score.

however in reality, the duration of TIA is typically much shorter, with the majority of episodes resolving within an hour. Approximately half of TIAs extending beyond one hour demonstrate ischaemic changes on neuroimaging, which has led to some authorities adopting a pathophysiological classification of TIA, rather than the traditional chronological approach; however this has not been adopted in the UK.⁴ The ABCD2 score (Table 2) is useful for risk stratification of TIA. Patients with a score ≥ 4 have a high risk of progression to stroke, and may require admission to hospital for early investigation and treatment (Figure 1).⁵

Symptoms of vertigo, dysarthria, ataxia or dysphagia in isolation without other focal neurological signs rarely point to a diagnosis of stroke. Furthermore, transient loss of consciousness or syncope is rarely due to stroke or TIA unless it is the result of a severely disabling stroke, a brainstem stroke (when associated with additional brainstem signs), or seizure complicating a stroke.

Defining the speed of onset is may also be helpful in determining the cause of hemiparesis. A subacute process developing over hours or days may suggest an infectious, inflammatory, malignant or metabolic pathology.

What is the main differential diagnosis?

Important differential diagnoses include seizure, hemiplegic migraine, reversible posterior leukoencephalopathy, malignancy and functional weakness. Hypoglycaemia may also present with focal neurological deficit, including hemiparesis.

Features which may be helpful in distinguishing some of these conditions are listed below:

- Seizures may be followed by post-ictal confusion or hemiparesis (Todd's paresis) lasting for several hours. An eye witness description of tonic-clonic movements at the time of onset, or a past history of seizure, alcohol excess, malignancy or other known structural brain disease should arouse suspicions. It should also be remembered that seizure may also complicate acute stroke.
- Migraine may present with a progressive hemiparesis over several minutes, usually in association with unilateral headache, nausea and vomiting; however the motor signs can occur in isolation. Patients are usually younger and may describe a series of symptoms occurring sequentially including positive visual phenomenon, word finding difficulty and limb paraesthesia. A history of previous events should be sought.
- Reversible posterior leukoencephalopathy syndrome manifests with posterior fossa symptoms including visual disturbances (e.g. hemianopia, cortical blindness), headaches, vomiting and seizures. Cerebral autoregulatory failure and endothelial dysfunction is thought to be responsible. Magnetic resonance imaging demonstrates white matter oedema in the posterior cerebral hemispheres.
- Functional symptoms represent transient motor or sensory symptoms not attributable to an identifiable neurological abnormality. In one case series, functional symptoms accounted for a third of 'stroke mimics' in patients under 50, which overall

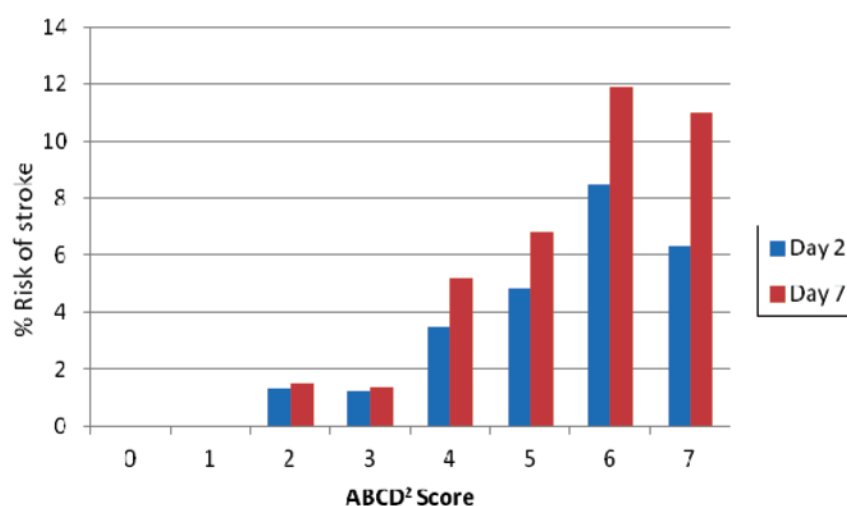


Figure 1. Short term risk of stroke following TIA as predicted by ABCD2 Score.

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accounted for 21% of stroke department admissions in this age group.⁶ Functional symptoms were very uncommon in those aged over 50 years.

What key physical examination findings could help in localisation and differential diagnosis?

The purpose of the physical examination is to localise the lesion and attempt to identify aetiological factors such as atrial fibrillation. However this should not delay neuro-imaging for patients presenting acutely, as early diagnosis and treatment is crucial. In some cases detailed examination may need to be deferred until after the imaging has been undertaken.

Neurological and general examination should include assessment of speech and cognitive function. In the acute phase, affected limbs become hypotonic and deep tendon reflexes may be hypo- or hyper-reflexic. Limb weakness typically follows a pyramidal pattern, with extensors affected to a greater degree than flexors in the upper limb and vice versa in the lower limb. Mild weakness may result in a pronator drift (a downward drift of the arm with pronation when held extended with the palm facing upwards). Later, affected limbs become hypertonic with brisk reflexes. Limb weakness, ataxia or sensory loss in association with cranial nerve involvement may help localise the lesion to the posterior circulation.

What features may suggest 'functional' hemiparesis?

Inconsistent examination findings may help to distinguish functional disorders from stroke; features of functional disturbance may include:

- Equal proximal/ distal and flexion/ extension weakness in the limbs.
- Ipsilateral sternocleidomastoid weakness.
- Intermittent voluntary effort with simultaneous contraction of flexion and extension muscles groups.
- Reflexes and muscle tone tend to be normal.
- Prominent verbal grunts during examination – 'Wrestler's sign'.
- Raising the contralateral leg may elicit a downward movement of the 'paretic' leg – 'Hoover's sign'.
- Finally, the patient may seem remarkably unconcerned and calm in the presence of marked weakness – 'la belle indifference'.

With encouragement, it is often possible to elicit full power. No single clinical sign is definitive and clinical judgement should always take precedence. Ultimately, a diagnosis of functional weakness should only be made when other possibilities have been excluded. Further, it should be remembered that exaggerated signs may be elicited following stroke, mimicking the feature of functional disorders in some cases.

Case Progression

The patient had been well immediately prior to this episode, although two weeks earlier his wife reported that his speech had

sounded 'jumbled' for a short period; this had recovered after around 30 minutes and he had declined her suggestion of visiting his GP. He usually took ramipril 5mg and bendroflumethiazide 2.5mg once daily for high blood pressure. His father died of a myocardial infarct, aged 75, and he had smoked 20/day since the age of 18.

Examination revealed right hemiplegia with brisk reflexes on the right and up-going plantar. There was an upper motor neurone VII nerve palsy and he was looking to the left with homonymous hemianopia on the right. He had not spoken since arrival in hospital but was able to follow simple commands.

Cardiovascular examination revealed a regular pulse of 80/min with blood pressure 150/90; there were no carotid bruits or heart murmurs. ECG confirmed sinus rhythm.

What immediate imaging should be undertaken?

Computed tomography (CT) remains the first line investigation for the early differentiation of haemorrhage from infarct, and to exclude non-stroke pathology, such as cerebral tumours. In this case the patient has a past history of hypertension, raising the possibility of intracerebral haemorrhage as a cause for his symptoms; however he also has risk factors for ischaemic stroke (smoking, hypertension and family history) and a possible recent left hemisphere TIA causing transient dysphasia. Given the importance of early intervention following acute ischaemic stroke the patient should undergo CTscan as soon as possible after arrival in hospital. An unenhanced brain scan takes approximately 10 seconds to obtain.

Following intracerebral haemorrhage, CT appearances evolve with time. After 5 days, small lesions may become isodense with the surrounding brain, particularly following a small bleed. By the end of the first week, lesions may become hypo-dense. Indeed CT appearances of previous intracerebral haemorrhages will often mimic the appearances of old infarction, such as the *ex-vacuo* effect of ventricular and sulcal enlargement near sites of previous haemorrhage.

Following ischaemic stroke, 60 % will have changes visible on CT scan within first 24 hours rising to 70% of those scanned within 72 hours. Early CT changes found in acute ischaemic stroke include cortical sulcal effacement and loss of the insular ribbon, a hyperdense middle cerebral artery due to thrombus within the artery, and loss of grey-white matter differentiation within the basal ganglia. Such findings are associated with larger infarcts and worse functional outcomes.⁷ As with haemorrhagic stroke, CT appearances evolve with time with lesions becoming more demarcated and hypodense during the first few days. Cerebral oedema is maximal around day 3 – 5, gradually subsiding during the 2nd – 3rd week, such that by the 2nd week infarct may increase in density and become isodense. Later, the infarct lesion forms sharp demarcated hypodense lesions.

Magnetic resonance imaging may provide a more detailed view of anatomical and pathological changes, as well as enabling non-invasive imaging of intracranial vessels. Diffusion weighted MR sequences are able to detect ischaemia within 30 minutes of onset. Patient related factors such as claustrophobia and the high level of co-operation

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needed for the longer period needed to obtain images may limit the use of this test in the acute setting, along with resource factors such as expense and accessibility.

Case Progression

The patient undergoes urgent CT scan within 30 minutes of arrival in the Emergency Department, which reveals evidence of acute ischaemic change in the left middle cerebral artery territory. There is no evidence of intracerebral haemorrhage. On return from the scanner, his condition remains unchanged with evidence of persistent right hemiplegia.

What immediate treatments should be considered for patients with acute ischaemic stroke?

Antiplatelet therapy should be commenced as soon as CT has excluded intracranial haemorrhage; aspirin 300mg is usually administered initially and can be given rectally if the patient is unable to swallow. NICE now recommends clopidogrel 75mg daily for secondary prevention of ischaemic stroke. A combination of aspirin with dipyridamole 200mg twice daily is recommended where clopidogrel is contraindicated or not tolerated.⁹

In 1995 the National Institute of Neurological Disorders & Stroke study¹⁰ (NINDS) demonstrated the efficacy of rt-PA intravenous thrombolysis when given within three hours of stroke onset. Patients in the treatment group were 30% more likely to have minimal or no disability at 3 months, at the expense of a significant rise in the incidence of symptomatic intracranial haemorrhage (6.4%). There was no mortality difference observed at three months. The European Co-operative Acute Stroke Study (ECASS III)¹¹ demonstrated the efficacy of thrombolysis up to 4.5 hours, with less benefit, but similar mortality and intracranial haemorrhage rates to the original NINDS trial. European Union approval of alteplase permits its use up to 3 hours post stroke onset, however some units in the UK have extended treatment time to 4.5 hours following ECASS III data. Improved outcomes are strongly associated with shortened door-to-needle times, and early thrombolysis is critical.

In this case the patient has presented to hospital early and CT findings confirm that he would be candidate for thrombolysis.

Intravenous thrombolysis results in restoration of vessel patency (i.e. recanalisation) in 46.2% of patients treated.¹² Should thrombus be demonstrated on angiography within an intracranial artery, intra-arterial thrombolysis may be attempted (recanalisation rates of 63.2% – 67.5%) or mechanical clot disruption techniques (83.6%). There are no national guidelines to guide therapy and patients should be discussed with the affiliated tertiary centre on a case by case basis. Recanalisation is associated with favourable functional outcomes and reduced mortality compared with failure to recanalise, with similar rates of haemorrhagic transformation.

What are the risks of thrombolysis?

The likelihood of improved outcome following thrombolysis needs to be balanced against the increased

risk of symptomatic intracerebral haemorrhage in around 1 in 14 patients. Should intracranial haemorrhage occur, treatment options are limited and it may be fatal. Treatment also carries a risk of anaphylaxis of < 1%. The patient and their next of kin should be counselled carefully regarding the risks and informed consent should be obtained where possible.

What are the other key management issues in acute hemiparesis?

Patients should be kept nil-by-mouth until a formal swallow assessment has been performed. If this is delayed, or if the assessment demonstrates that the patient is at risk of aspiration a nasogastric tube should be sited to enable initiation of enteral feeding and the administration of medication.

Additional management measures are aimed at maintaining homeostasis in an attempt to rescue ischaemic tissue at risk of infarction (i.e. penumbral salvage). Such measures are illustrated in Table 3.

Admission to a stroke unit is strongly associated with better outcomes, due to improved monitoring of physiological parameters, prevention of complications and

Hyperglycaemia	Associated with worse outcome Use insulin to maintain glucose $\leq$ 11mmol/l
Hypoxia	Use supplementary oxygen only to maintain saturations $>95\%$ (use clinical judgement in those with significant airways disease)
Pyrexia	Associated with worse outcomes Identify potential sources then treat with antipyretics
Hypertension	No evidence in favour of treating in the acute phase Treat if evidence of end organ damage (NICE) ⁸

Table 3. Maintaining homeostasis following acute stroke.

early mobilisation. National guidelines in England now recommend that all patients are managed throughout their hospital stay in a specialist stroke unit.⁸

What other complications may arise following middle cerebral artery infarction?

Malignant middle cerebral artery syndrome occurs in 10% of MCA infarcts. Findings include dense hemiplegia and forced eye deviation, while CT will demonstrate a large area of hypodensity and midline shift. Left untreated, it carries a mortality of 78% as a consequence of herniation of the temporal lobe onto the brainstem.¹³ Decompressive hemicraniectomy and durotomy is the surgical technique used to relieve the increased intracranial pressure, thus improving cerebral perfusion pressure. A pooled analysis of outcome following the procedure demonstrated greater survival (numbers needed to treat 2) and favourable outcome at one year mRS 0–4 (75 Vs 24 percent in the control group).¹⁴ Referral for decompressive hemicraniectomy is recommended for patients fulfilling the following criteria:⁸

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1. Age < 60 years
2. Clinical deficits suggestive of MCA territory infarction, with a score on the National Institutes of Health Stroke Scale (NIHSS) of > 15.
3. Decrease in the level of consciousness to give a score of 1 or more on item 1a of the NIHSS.
4. Signs on CT of an infarct of at least 50% of the middle cerebral artery territory, with or without additional infarction in the territory of the anterior or posterior cerebral artery on the same side, or infarct volume greater than 145 cm³ as shown on diffusion-weighted MRI.

What other investigations should be considered?

In anterior circulation ischaemic stroke, carotid doppler assessment should be considered to enable imaging of extracranial carotid vessels, particularly where there has been significant functional improvement. In ischaemic stroke in younger patients, tests for vasculitis and lupus should be performed and CT or MR angiography if intracranial carotid or vertebral artery dissection is considered a possible cause. Trans-thoracic echocardiography should also be considered to enable identification of a cardiac source of embolic stroke, which may require treatment with long-term anticoagulant drugs.

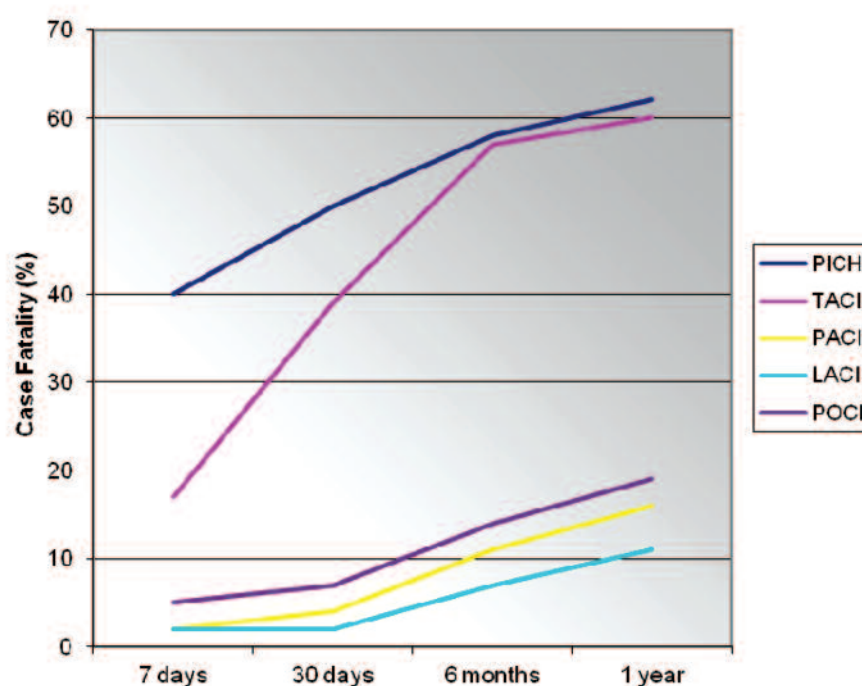
What is the prognosis for patients presenting with acute hemiparesis?

Mortality and functional recovery following stroke is dependent on the stroke subtype and may be predicted

by using the OCSF classification (Figure 2). Recovery from stroke occurs through a variety of mechanisms. Penumbra salvage results in the restoration of functioning neurones. Neuroplasticity refers to the ability of neighbouring cells to take over the lost function. Adaptive behaviour is learned with the support of various members of the multi-disciplinary team, including physiotherapists, occupational therapists, speech therapists and neuropsychologists. The rate of recovery is highest in first few weeks after stroke; functional improvement continues at a slower rate for many months (occasionally up to 2 years). It remains difficult to predict the rate and completeness of recovery.

Case progression

The patient was transferred to the acute stroke unit and treated with rt-PA within 1 hour of arrival in hospital (2 hours after onset of the weakness). Following this treatment an improvement in his weakness was noted with power improving to 3/5 in his limbs and some recovery of speech. He was initially fed via a nasogastric tube after failing a swallow assessment, but this improved within 72 hours enabling oral nutrition to be initiated. Carotid doppler showed minor plaque only, and an echocardiogram was normal. He was commenced on clopidogrel 75mg daily and simvastatin 40mg daily and his blood pressure control was maintained without alteration of his usual medication. The improvement continued over subsequent weeks with the support of the multiprofessional team on the stroke unit and he was transferred to a community rehabilitation unit after an inpatient stay of 2 weeks.



N.B PICH: primary intracerebral haemorrhage

Figure 2. Stroke prognosis based on OCSF subtype¹ & primary intracerebral haemorrhage.

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Additional on-line training resources:

1. NIHSS certification (used in stroke thrombolysis assessment): <http://nihss-english.trainingcampus.net/uas/modules/trees/windex.aspx>
2. Stroke thrombolysis training: http://www.strokeadvancingmodules.org/labyrinth_thrombo/
3. Resource on functional symptoms for professionals and patients: <http://www.neurosymbols.org/>

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