Acute Stroke – Diagnosis and Management

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ABSTRACT

Stroke (noun): a sudden disabling attack... caused by an interruption in the flow of blood to the brain, especially through thrombosis.

Stroke is a considerable cause of mortality and morbidity in the UK. The field of stroke medicine has changed considerably in recent years with the development of hyper-acute treatments such as thrombolysis, specialist stroke units and a better understanding of secondary prevention. Mortality rates may have decreased but diagnostics have become more sensitive and it is not clear whether incidence of stroke is falling overall. It is predominantly a problem of advancing age and many of those suffering a stroke will be from the older age bracket. This often raises interesting challenges in the diagnosis and management process due to the complex needs of the patient in the bed.

This article will review the tools available to assist in the systematic assessment and treatment of people with a suspected stroke.

Key Words: stroke; imaging; thrombolysis

Background

Stroke is a considerable cause of mortality and morbidity in the UK. The field of stroke medicine has changed considerably in recent years with the development of hyper-acute treatments such as thrombolysis, specialist stroke units and a better understanding of secondary prevention. Mortality rates may have decreased but diagnostics have become more sensitive and it is not clear whether incidence of stroke is falling overall. It is predominantly a problem of advancing age and many of those suffering a stroke will be from the older age bracket. This often raises interesting challenges in the diagnosis and management process due to the complex needs of the patient in the bed.

This article will review the tools available to assist in the systematic assessment and treatment of people with a suspected stroke. The change in stroke services has meant that acute events are now often admitted directly to acute stroke units as these are proven to provide optimal care in the acute and sub-acute phases. In some cases this means that they are omitted from the experience of the acute general medical take. They remain however, an important general medical emergency.

Recognition and Diagnosis

Rapid diagnosis of a stroke is the first step to instigating appropriate treatment. In the case of thrombolysis, where potent fibrinolytic drugs are given to restore cerebral blood supply, the faster a stroke is recognised and treated, the better the outcome. Strokes are not “black
and white” clinical entities however and many “mimics” have similar symptoms that can lead to diagnostic uncertainties.

The symptoms of a stroke are due to the acute interruption of the blood supply to an area of the brain. This can be through blockage of a blood vessel (infarcts) or haemorrhage. Infarcts can be caused by emboli, usually from thrombus in the carotid arteries or left atrium, or from in-situ clot formation. In both haemorrhagic strokes and infarcts the onset is sudden. The vascular territory involved and the presence of associated symptoms may assist in the diagnosis.

- Anterior circulation – weakness, sensory deficits, dysphasia (expressive and receptive), visual field defects, dyspraxia and higher cortical dysfunction, contralateral signs.
- Posterior circulation – visual field defects, ataxia and vertigo, inco-ordination, cranial nerve deficits, ipsilateral signs.

To allow rapid identification of these stroke patterns and to assist in the exclusion of other possible diagnoses, the following screening tools have been developed.

**F.A.S.T.**

This stands for **Face**, **Arm**, **Speech**, **Time** and has been the subject of a national television awareness campaign. It is used by paramedics and emergency department triage staff to screen for stroke symptoms and can be up to 81% sensitive. It prompts assessment for facial asymmetry, arm weakness, slurred or disordered speech and then rapid transfer to the appropriate acute care setting for further assessment. FAST is not infallible and is particularly prone to missing posterior circulation events.

**R.O.S.I.E.R.**

This tool is for **Recognition of Stroke in the Emergency Room**. It was developed to help emergency department staff assess possible stroke patients and provides some tools for screening out mimics such as hypoglycaemia, seizures and syncope. It has a sensitivity of 93% and specificity of 83%. See Table 1.

### Table 1 – ROSIER score

<table>
<thead>
<tr>
<th>Is the blood glucose above 3.5mmol? If not, treat and re-assess.</th>
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<tbody>
<tr>
<td>1. Has there been loss of consciousness or syncope? Yes (-1) No (0)</td>
</tr>
<tr>
<td>2. Has there been seizure activity? Yes (-1) No (0)</td>
</tr>
<tr>
<td>3. Is there NEW ACUTE onset (or on awakening from sleep) of:</td>
</tr>
<tr>
<td>• Asymmetrical face weakness Yes (+1) No (0)</td>
</tr>
<tr>
<td>• Asymmetrical arm weakness Yes (+1) No (0)</td>
</tr>
<tr>
<td>• Asymmetrical leg weakness Yes (+1) No (0)</td>
</tr>
<tr>
<td>• Speech disturbance Yes (+1) No (0)</td>
</tr>
<tr>
<td>• Visual field defect Yes (+1) No (0)</td>
</tr>
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Paramedic teams trained in the use of FAST screening will often refer patients directly to acute stroke units. Use of the ROSIER scale in emergency departments and medical admissions units allows self-presenting patients or those admitted via GPs to be assessed.
rapidly so that appropriate admission pathways and investigations can be instigated promptly. Following assessment with screening tools, patients with suspected strokes should have a thorough history taken and a comprehensive neurological examination. This may need to be simultaneous with the ordering of investigations such as CT imaging if the patient may be a candidate for thrombolysis as will be discussed later.

**History and Examination**

Timing is a crucial point when taking a patient’s history of a suspected stroke. The onset of symptoms is usually sudden, with all deficits occurring together, as opposed to a “marching” or progressive deficit. The time of onset is vital, especially when considering patients for thrombolysis that must be initiated within 4.5 hours of symptom onset\(^9\). It is necessary to clearly determine when the patient was last well. Often patients wake up with symptoms however they may have got up in the night or spoken to their partner in the early hours of the morning and the time they were last well can still be identified.

It is useful when reviewing neurological symptoms to consider them as positive or negative. Positive symptoms involve gaining a quality such as extra movements or shaking, added sensations such as prickling or burning and extra visual signs such as flashing lights. Negative symptoms describe the loss of a normal function such a weakness, loss of sensation, loss of comprehensible and useful speech, loss of vision (full or partial) or incoordination. Strokes generally produce negative symptoms. The presence of positive symptoms can sway the assessing physician towards a diagnosis of a mimic. The negative symptom of loss consciousness is rarely a feature of stroke (Table 2).

### Table 2- Positive and negative features on examination and stroke mimics

<table>
<thead>
<tr>
<th>Positive symptom</th>
<th>Stroke Mimic</th>
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<tbody>
<tr>
<td>Flashing lights and colours in vision.</td>
<td>Migraine</td>
</tr>
<tr>
<td>Shaking or jerking limbs.</td>
<td>Seizures (focal or generalized).</td>
</tr>
<tr>
<td>Tingling / prickling sensation.</td>
<td>Radiculopathy, herpes zoster infection.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Negative symptom</th>
<th>Stroke Mimic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of consciousness</td>
<td>Syncope, seizures.</td>
</tr>
</tbody>
</table>

Associated symptoms mainly have a role in identifying mimics or increasing the suspicion of an intra-cerebral haemorrhage. Haemorrhagic strokes may present with headache, nausea and vomiting, which are otherwise uncommon in strokes but may also be present in migraine. Confusion and agitation can occur with intra-cranial haemorrhages or non-stroke diagnoses such as infection or hypoglycaemia. A medication history is also important, particularly if patients are taking antiplatelet or anticoagulant therapy.

When considering the diagnosis of a stroke it is useful to look at the vascular risk factors of the patient. This provides a guide to the probability of a stroke but also as a target for future secondary prevention. Hypertension, diabetes, smoking, hyperlipidaemia, family history, male gender, age and other vasculopathies are potent indicators of stroke risk however strokes are still possible in those without these factors.

Each patient with a suspected stroke should have a thorough neurological examination including assessment of motor and sensory modalities and cerebellar function. Pronator
Drift is an excellent sign of subtle motor weakness. Higher cortical function such as speech and praxis should be assessed. Speech quality, fluency and word finding skills can be assessed throughout taking the history. Providing simultaneous stimuli to bilateral visual fields or sensory pathways can identify inattention. The inability to identify when both sides are stimulated suggests cortical dysfunction. Cranial nerve examinations should differentiate between upper and lower motor neuron facial weakness and should include an assessment of visual fields. Neurological examination findings can be applied to the National Institute for Health Stroke Scale (NIHSS) to give a way of communicating and monitoring stroke severity. The application of this scale in a reproducible way requires training. The examination findings will also allow the stroke to be classified as per the Bamford Classification. This gives some guide of mortality and morbidity with a total anterior circulation stroke carrying a higher chance of both.

**Total anterior circulation stroke (TACS)** – All three of the following: 1. Unilateral weakness (and/or sensory deficit) of face, arm and leg. 2. Homonymous hemianopia. 3. Higher cerebral dysfunction (dysphasia, visuospatial disorder).

**Partial anterior circulation stroke (PACS)** – Two of the following: 1. Unilateral weakness (and/or sensory deficit) of face, arm and leg. 2. Homonymous hemianopia. 3. Higher cerebral dysfunction (dysphasia, visuospatial disorder).

**Lacunar stroke (LACS)** – One of the following: 1. Unilateral weakness (and/or sensory deficit) of face and arm, arm and leg or all three. 2. Pure sensory stroke. 3. Ataxic hemiparesis.

**Posterior circulation stroke (PoCS)** – One of the following: 1. Cerebellar or brainstem syndromes. 2. Loss of consciousness. 3. Isolated homonymous hemianopia

Please note that both TACS and PACS involve the anterior and middle cerebral arteries (ACA and MCA), whilst the PoCS involves the posterior circulation.

**Transient Ischaemic Attack**

TIAs can present with symptoms in either of the vascular territories described above but resolve entirely within 24 hours and usually in less than one hour. Amaurosis fugax, described as a curtain coming over the vision in one eye and resolving rapidly is also a form of TIA. TIAs can generally be managed as an outpatient and therefore should rarely be seen on medical admission units having been assessed and referred from GPs or emergency departments.

Following a TIA, the risk of progression to a full stroke within the next 7 days can be predicted by the use of the ABCD2 score (Table 3).

<table>
<thead>
<tr>
<th><strong>Table 3 – ABCD2 score</strong></th>
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<tbody>
<tr>
<td>A – age &gt; 60</td>
</tr>
<tr>
<td>B – blood pressure &gt;140/90</td>
</tr>
<tr>
<td>C – clinical picture:</td>
</tr>
<tr>
<td>Unilateral weakness</td>
</tr>
<tr>
<td>Speech disturbance</td>
</tr>
<tr>
<td>D – Duration</td>
</tr>
<tr>
<td>&gt;60 minutes</td>
</tr>
<tr>
<td>1-59 minutes</td>
</tr>
<tr>
<td>D – Diabetes</td>
</tr>
</tbody>
</table>

Total out of max. 7
Once diagnosed with a TIA, with particular emphasis that all symptoms should have resolved entirely, patients can be commenced on an antiplatelet agent. An ABCD2 score of 4 or less can be referred for assessment in a TIA clinic. The aim of this service is to identify modifiable risk factors, in particular carotid stenosis that may be amenable to surgical intervention, and atrial fibrillation for which oral anticoagulants may be appropriate. TIA clinics are often run five or even seven days a week to allow rapid access. A score greater than 4 carries a very significant risk of progressing to a stroke and these cases should be assessed within 24 hours with carotid imaging, as should patients presenting with crescendo TIAs (more than one episode in a week). In centres without seven-day TIA clinics this may require admission to hospital.

Investigations

Once a stroke is suspected clinically, the appropriate investigation should be pursued urgently to allow optimal treatment.

Laboratory tests.
A blood glucose level is necessary to exclude hypoglycaemia as a stroke mimic and this can usual be done as a bedside finger prick test. Blood tests should be sent for full blood count and biochemistry. A coagulation screen should be sent particularly if a bleed is suspected, if the patient is anti-coagulated or if thrombolysis is being considered. In the days after a stroke, thyroid function tests, lipid profile and ESR are also useful investigations.

Imaging and their common appearances.
CT remains the mainstay in acute radiological investigation in suspected stroke. This should happen urgently, within 1 hour, in cases being considered for thrombolysis and those cases with a low Glasgow come score (GCS), signs of meningism or a high suspicion of intracerebral bleed\(^\text{1,12}\). All strokes should have imaging within 24 hours of presentation\(^\text{1,12}\).

The role of CT in the immediate phase is mainly to exclude the presence of intracranial haemorrhage. Intra-cerebral, subarachnoid, subdural and extradural bleeds have a characteristic appearance on CT. Intra-cerebral haemorrhages are generally rounded, well-circumscribed lesions within the brain parenchyma (Figure 1). They may have surrounding oedema or extend into the ventricles.

Infarcted cerebral tissue appears unchanged on CT scans in the first few hours. The changes seen later develop at a range of 2-3 Hounsfield units per hour. The human eye can only detect a difference in contrast of over 6 Hounsfield units so a clinically apparent stroke will only become radiological apparent after a few hours.

There are patterns on CT that can indicate cerebral infarction, particularly when applied with the suspected vascular territory. There may be a loss of differentiation between grey and white matter. When this occurs in the insular region it is referred to as the insular ribbon sign. Loss of differentiation may also occur between structures such as the basal ganglia and internal capsule. There may be sulcal effacement due to underlying oedema. Clot may be visible in the proximal middle cerebral artery, “dense MCA” sign (Figure 2) or as a “dot sign” if a more distal branch has been occluded. In the posterior circulation, a hyper-dense basilar artery may be visible (Figure 3) and a high suspicion of impending occlusion is necessary in patients presenting with nausea and vertigo and posterior circulation signs. Cerebellar and brainstem strokes are poorly imaged on CT and may require MRI if the diagnosis is in doubt.
Figure 1: CT of an intra-cranial bleed

Figure 2: Dense MCA Sign

Figure 3: Hyper-dense basilar artery
MRI has become a useful tool in stroke diagnosis in recent years. This is primarily due to the use of diffusion weighted imaging (DWI). Acute infarcts will appear bright on DWI for up to fourteen days. The detailed images produced with MRI, as well as the superior imaging of the posterior intra-cranial structures makes this modality useful when a PoCS is suspected or if the diagnosis or vascular territory is in doubt. Other MRI modalities such as gradient echo can identify microhaemorrhages.

The availability of MRI, particularly out of hours, means that it does not have a place in the hyper-acute phases of stroke management and is unlikely to replace CT in the near future however it remains a useful adjunct.

Imaging of the cerebral blood supply, particularly at a carotid level, can assist in identifying the pathophysiology of embolic strokes. Plaque formation causing stenosis in the internal carotid arteries leads to thrombus formation that then embolises to more distal cerebral vessels. This imaging can be done by Doppler measurements in the acute phase after stroke so decisions can be made about carotid endarterectomy. The use of CT angiography is becoming increasingly popular as new hyper-acute treatments including intra-arterial thrombolysis and mechanical clot retrieval are investigated. These interventions rely on early and detailed imaging of the cerebral blood supply. It is also useful in cases of suspected carotid or vertebral dissection. CT angiography is often not routinely available out of normal working hours and its use is at the discretion of stroke physicians and radiologists.

Cardiac investigations.
The aim of cardiac investigations in stroke patients is to identify atrial fibrillation as a source of cardiac emboli. In the acute phase, this can be done with a bedside 12 lead ECG. In time, patients with embolic strokes will need further cardiac investigations to guide secondary prevention measures.

Treatment and Management

In the acute phase, there are several important areas of stroke care to consider including treatment of the acute event and prevention of complications. Haemorrhagic strokes may need discussion with the neurosurgeons.

Apart from thrombolysis or antiplatelet measures, the management of these cases has many similarities to cerebral infarcts and is discussed below. For cerebral infarcts, specific treatment options need to be considered.

Acute Treatment Options

Thrombolysis.
All stroke patients presenting with a suspected cerebral infarction and a time of onset within 4.5 hours should be considered for thrombolysis. This requires the urgent exclusion of haemorrhage and discussion with a stroke consultant. Thrombolysis has been shown to improve functional outcome though it has no effect on mortality. It involves the giving of an intravenous fibrinolytic agent (recombinant tissue plasminogen activator).

Thrombolysis is licensed for adults of all ages and benefit has been confirmed in the elderly beyond the age of 80 years\(^9\). Treatment is most effective when the “door to needle” time is
shortest. The primary risk of treatment is haemorrhage, either intra- or extra-cranial. Asking about contraindications can minimise this risk.

Contraindications include a time of onset greater than 4.5 hours prior to treatment, blood pressure >180mmHg systolic or > 110mmHg diastolic, blood glucose >22 or <2.8mmol, abnormal clotting or known anti-coagulation, thrombocytopenia, established infarct on CT, history of haemorrhage, recent surgery, previous strokes in patients with diabetes, seizure activity at any time in the presentation and pre-existing significant disability. Some contraindications are relative where others absolute. Some may be modifiable, such as blood pressure. The issue of pre-existing disability relates to the aim of thrombolysis to improve the chances of good functional recovery, this requires a reasonable premorbid level of function.

Stroke physicians will review each case on an individual basis and balance the risks and benefits. The counselling undertaken before thrombolysis can be complex and emotional for patients and relatives. After thrombolysis patients require intensive monitoring in a specialist unit and repeat scan after 24 hours to exclude haemorrhagic transformation. Following this they can be started on high dose aspirin.

**Antiplatelet and anticoagulant therapy.**
For those patients not having thrombolysis but in whom a haemorrhage has been excluded, the mainstay of treatment is high dose aspirin (300mg daily). This is continued for fourteen days after the initial event with the aim of reducing the risk of a further embolic event. Long-term antiplatelet therapy is with clopidogrel.

Anticoagulant therapy is usually omitted for fourteen days post-stroke. In those on anticoagulants at the time of infarction the decision to continue or stop may be taken by the stroke consultant and depends on the infarct size and risks involved.

**Blood pressure control.**
Blood pressure levels often rise around the time of an acute stroke. The risk of allowing blood pressure to rise is that intra-cerebral haemorrhage may expand and cerebral infarcts may develop haemorrhagic transformation. Lowering blood pressure in the acute phase after a stroke is contentious as it may be that the rise seen is a physiological response to maintain cerebral perfusion. Ideally blood pressures should be kept below 180mmHg but advice should be taken from a stroke specialist.

**Nutrition and hydration.**
Many stroke patients will have an unsafe swallow in the early phases after the event. A ward based swallow assessment should be completed as soon as possible. In those with an unsafe swallow, medication should be reviewed and either suspended or given via an alternative route (rectal, intravenous or topically). Intravenous fluids can be given and artificial feeding should be considered within the first 24 hours by a nasogastric tube. Aspiration pneumonia is a common complication of stroke and a low threshold for treatment should be maintained.

**Thrombo-embolism prophylaxis.**
Thrombo-embolic events are an important cause of death following stroke. Routine prophylaxis with low molecular weight heparin carries risks. It is contra-indicated in acute haemorrhagic stroke and can increase the risk of haemorrhagic transformation in cerebral infarction. Graduated compression stockings have been shown to cause more harm than
benefit including pressure damage in stroke patients and are now contraindicated\textsuperscript{13}. Recent research suggests the use of intermittent pneumatic compression stockings can be used to reduce thrombo-embolic risk\textsuperscript{14} and the case for using low molecular weight heparin can be discussed on a case-by-case basis dependent on the individual risk.

**Review of patients with acute strokes.**
All stroke patients should be transferred to the acute stroke unit as soon as possible after admission. They should be seen within the next 24 hours by a stroke consultant or associate specialist and within 72 hours by the multidisciplinary team therapists.

**The Next Step**

The aim of all management in stroke is two fold, to restore as much function as possible to the individual and to reduce the chance of future similar events. The former relies on rehabilitation and the skills of the multidisciplinary team. The latter has grown from an evidence base that can then be tailored to each individual patient’s needs. These issues will be reviewed in the next article on stroke care.

**References**

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