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PART 2: Care Bundle

Emergency Department Stroke and Transient Ischaemic Attack Care Bundle:

Information and implementation package

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Part 2: Care Bundle

All people presenting to emergency departments with stroke-like symptoms should receive:

✓ Rapid initial stroke screen

✓ ABCD[2] assessment when TIA suspected

A Age: ≥ 60 years (1 point)

B Blood pressure: ≥ 140/90mmHg (1 point)

C Clinical features: unilateral weakness (2 points), speech impairment without weakness (1 point)

D Duration: > 60 mins (2 points), 10-59 mins (1 point)

D Diabetes (1 point)

Tool interpretation[1]

>4 = HIGH risk; ≤4 = LOW risk Maximum score = 7

✓ Urgent* CT or MRI

✓ Nil by mouth until bedside swallow screen (within 24 hours) for stroke

✓ Aspirin as soon as possible**, if haemorrhage excluded

150-300mg one-time loading unless contraindicated

✓ Physiological monitoring and management:

- **Neurological status**
  Regular monitoring to establish baseline and identify change
- **Blood glucose**
  Cautious treatment of markedly elevated blood glucose levels; early, intensive maintenance of euglycaemia is not recommended. Avoid hypoglycaemia
- **Blood pressure**
  Cautious lowering by no more than 10-20% if extremely high ≥ 220/120; monitor for neurological deterioration. Avoid hypotension
- **Hydration status**
  Maintain euvolemia

* 'Urgent' is considered as soon as possible, but certainly less than 24 hrs[1]
** 'As soon as possible' is considered within 48 hrs[1]

Please note: This care bundle represents key components of stroke and TIA care that are essential to evidence-based stroke care. This is not a complete list of all care components that will be required. Other interventions will be necessary within the continuum of care.

Rapid initial stroke screen

NSF[1] recommendation: ED staff should use a validated stroke screen tool to assist in rapid accurate assessment for all people with suspected stroke (Grade C; Level II)

Rationale

The diagnostic accuracy of ED staff is increased by the use of a validated stroke recognition tool and training in that tool.[9,18] Rapid and accurate diagnosis leads to earlier and more appropriate referrals to available stroke expertise[18], i.e. a stroke unit or a physician experienced in stroke care. This in turn should lead to timely treatment and better outcomes.[19]

Results from the 2009 NSF *National stroke audit acute services organisational survey report*[2] also found that half of hospitals surveyed did not have emergency department triage protocols for stroke.

This audit also found that even in hospitals surveyed with stroke units, a third of stroke patients were receiving care on other wards.

Resource Implications

- Available tools should be assessed and the preferred tool selected for use in the initial assessment in ED. The recommended tool is the Recognition of Stroke in the Emergency Room scale (ROSIER).[18] See Appendix G for a copy of the ROSIER scale.
- The selected tool should be implemented into standard practice as part of the initial assessment in ED of all suspected stroke patients.
- ED staff responsible for initial assessment should be trained in the use of the selected tool as evidence indicates that training improves the accuracy of diagnosis.[9,14,20]
- Any locally used tool should prompt rapid referral to available stroke expertise (i.e. acute stroke response team).

Audit Measure

☑ Indication that agreed stroke screen tool was used at agreed time (i.e. triage or initial assessment).

Guideline Summary

Along with the NSF[1], the AHA/ASA[11], NICE[9] and SIGN[15] also recommend the use of a validated stroke screen tool for rapid initial assessment of suspected stroke and TIA patients. In addition, the ESO[12], ICSI[14] and RNAO[13] guidelines recommend that these patients be rapidly assessed, but do not specifically recommend the use of a validated stroke screen tool.

Assessment and initial treatment for stroke should be performed as a priority in the ED. The clinical assessment is the cornerstone of this process.[11]

An effective and efficient medical assessment is essential to the early identification of stroke as well as the exclusion of stroke mimics.[1,11] A validated stroke screen tool has been shown to increase diagnostic accuracy and immediate diagnosis improves speed of access to treatment.[9,15] Such a tool should be used in conjunction with the standard clinical examination for all stroke patients.

There are a number of validated stroke screening tools currently in use in Australia and internationally, including the:

- Cincinnati Prehospital Stroke Scale (CPSS)[21]
- Face Arm Speech Test (FAST)[22]
- Los Angeles Prehospital Stroke Screen (LAPSS)[23]
- Melbourne Ambulance Stroke Screen (MASS)[24]
- National Institutes of Health Stroke Scale (NIHSS)[25]
- Recognition of Stroke in the Emergency Room (ROSIER)[18]

NICE[9] suggests that whilst a simple assessment, such as FAST, is necessary for pre-hospital assessment, a more detailed assessment tool, such as ROSIER, is required in the ED to exclude stroke mimics.

The ROSIER scale (Appendix G) is the only tool that has been validated specifically for use in the ED following triage.[18] When used by medical or ED staff, it has been shown to identify stroke and stroke mimics more accurately than CPSS, FAST or LAPSS.[9,15] NSF[1], NICE[9] and SIGN[15] recommend using the ROSIER scale for initial assessment in the ED, as it is the only scale that has been adequately studied in the ED.

It should be noted that CPSS, FAST, LAPSS and MASS have been developed and validated only in the pre-hospital setting, i.e. for paramedics. However these tools may be useful in triage if the patient did not present via ambulance. NIHSS was originally designed to assess differences in interventions in clinical trials, although it has increasingly been used in patient care as an initial assessment tool.[26]

A small number of studies have found that emergency medical staff have a high diagnostic accuracy (approximately 90% sensitivity). However 20-30% of patients are incorrectly diagnosed with stroke or TIA, indicating a high sensitivity, but lower specificity.[1]

Studies have shown that education and training of ED staff results in improvements in the accuracy of diagnosis[9] and processes of care.[1] ED clinical staff should be educated in the importance of stroke symptom recognition[14], stroke mimics[13] and the appropriate triage measures to take if stroke or TIA is suspected.[14]

ABCD^2 assessment when TIA suspected

NSF[1] recommendation: All patients with suspected TIA should have a full assessment that includes assessment of stroke risk using the ABCD^2 tool at initial point of health care contact (Grade B; Level II)

Rationale

TIA and minor stroke patients are at high risk of subsequent stroke[15], with up to 10% suffering a stroke within the following 48 hours (2.5-5% at 2 days; 5-10% at 30 days; 10-20% at 90 days).[12] Efficiency and accuracy of TIA diagnosis and management in the ED is important in reducing the incidence of subsequent stroke.[10,27] The ABCD^2 assessment tool can provide stratification information to guide management decisions.

The 2009 NSF *National stroke audit acute services organisational survey report*[2] found that less than half of hospitals surveyed had a defined pathway for assessing TIA patients and only 39% were using a risk stratification tool.

Resource Implications

- ABCD^2 should be implemented into standard practice in the selected area of ED (e.g. triage or treatment area).
- ED staff responsible for assessment should be trained in the use of ABCD^2.

Audit Measure

☑ Indication that ABCD^2 assessment was undertaken at the initial assessment (for all TIA patients only).

Guideline Summary

The NSF[1], SIGN[15], NICE[9] and ICSI[14] guidelines all recommend the use of the ABCD^2 tool for suspected TIA patients. The CSN guideline[10] recommends the use of a standardised stratification tool but does not recommend the ABCD^2 tool.

As there are strong similarities between minor ischaemic stroke and TIA, it follows that initial assessment and management should be the same.[1]

Recent data has shown a higher and earlier risk of subsequent stroke for TIA patients than previously thought (2.5-5% at 2 days; 5-10% at 30 days; 10-20% at 90 days[12]). Approximately half of the early risk is seen within the first 48 hours, necessitating early initial assessment and management in order to prevent further events.[1] Streamlined systems that definitively diagnose TIA and initiate secondary treatment within 24-48 hours are associated with reduced rates of early death.[14]

Simple risk stratification tools for TIA have been shown to be accurate in identifying patients at high risk of early subsequent stroke who require immediate assessment and management in the ED.[9,14,15] The ABCD^2 tool (Figure 2) has been found to be accurate in identifying TIA patients at high risk of subsequent stroke[1,9,14,15] and is the best, validated tool currently available.[28] ABCD^2 is a simple, efficient way of predicting stroke in TIA patients and is appropriate for use in emergency care.[29]

Included in the tool are the five risk factors that have been identified for early stroke after TIA:

- age (≥ 60 years)
- high blood pressure (≥ 140 systolic or ≥ 90 diastolic)
- motor or speech symptoms
- longer symptom duration (> 10 min)
- diabetes mellitus.

Recommendations for hospital admission following TIA have historically been vague and practice varied.[28] The ABCD^2 score can be used as a decision tool to determine the course of treatment of TIA patients.

NSF[1] recommends that TIA patients with an ABCD^2 score of 5 or greater be designated as high risk, be admitted (or, where available, referred to a TIA clinic for urgent assessment) to facilitate rapid assessment, including urgent head imaging (as soon as possible, but certainly within 24 hours), and be treated as for acute stroke.

All TIA patients with an ABCD^2 score of 4 or less are designated as low risk and should have a CT brain scan and carotid ultrasound (where indicated) as soon as possible, that is, within 48 to 72 hours. Low risk TIA patients should be referred to a general practitioner, private specialist or TIA clinic for ongoing management.[1]

NICE[9] and ICSI[14] designate high risk patients as having an ABCD^2 score of 4 or greater, as derived from Johnston et al[28], and recommend they be immediately identified, assessed and secondary prevention be initiated. NICE[9] considers secondary prevention to include antiplatelet agents, blood pressure management, anticoagulation in selected patients and management of dyslipidaemia including statins.

In strict accordance with Johnston et al[28], SIGN has designated ABCD^2 scores of 0-3 as low risk; 4-5 as moderate risk; and 6-7 as high risk.

ICSI recommends that hospitalisation, or expedited outpatient assessment, be considered for recent (within 24-48 hours) and crescendo TIAs. ICSI[14] does not recommend that patients be selected for hospitalisation solely on their ABCD^2 score, although accepts that this may happen in practice.

ABCD^2 Tool[28]

A Age: ≥ 60 years (1 point)
B Blood pressure: ≥ 140/90mmHg (1 point)
C Clinical features: unilateral weakness (2 points),
speech impairment without weakness (1 point)
D Duration: > 60 mins (2 points), 10-59 mins (1 point)
D Diabetes (1 point)

Tool interpretation[1]

>4 = HIGH risk; ≤4 = LOW risk Maximum score = 7

Urgent CT or MRI

NSF[1] recommendations: All patients with suspected stroke should have an urgent* brain CT or MRI (Grade A; Level I)

TIA patients classified as high risk ($ABCD^2 > 4$) should have an urgent* CT brain. Patients classified as low risk ($ABCD^2 < 5$) should have a CT brain and carotid ultrasound (where indicated) as soon as possible** (Grade B; Level I & III-3)

* 'urgent' is considered as soon as possible, but certainly less than 24 hours
** 'as soon as possible' is considered within 48-72 hours

Rationale

Clinicians disagree on the clinical diagnosis of stroke (versus stroke mimic) in about 20% of patients. Brain imaging is required to distinguish ischaemic stroke from intracranial haemorrhage and stroke mimics and should be performed immediately so that treatment can start promptly.[12,15]

One systematic review reported that the most cost effective strategy in acute stroke is for all patients to undergo 'immediate' imaging, as opposed to 'within 48 hours'.[9,10,12,15]

The 2009 NSF *National stroke audit acute services organisational survey report*[2] found that one third of rural hospitals surveyed that managed acute stroke patients had no access to CT.

Resource Implications

- Initial assessment should be performed using agreed tools by the most appropriate ED staff member, i.e. the clinician most experienced in stroke, to determine diagnostic needs and urgency.
- Local protocols should be developed for prioritising stroke and high risk TIA for rapid access to brain imaging services (imaging and reporting).
- An organised system of stroke care should be developed to ensure timely access to brain imaging services (imaging and reporting) if not available at the presenting hospital.

Audit Measure

☑ CT or MRI conducted within 24 hours of presentation (time of registration or triage, whichever comes first chronologically)

Guideline Summary

In addition to the NSF[1], the following guidelines also have a similar recommendation for an urgent initial brain CT or MRI for all suspected stroke: ESO[12], NICE[9], CSN[10], AHA/ASA[11], ICSI[14] and SIGN.[15]

Imaging modalities

The primary purpose of initial brain imaging is to exclude intracranial haemorrhage (ICH)[12,14,15] and non-vascular stroke mimics[12], although it may also provide information on

the ischaemic penumbra[12] and on early ischaemic changes in the brain such as mass effect from oedema, middle cerebral artery embolic material, other vascular lesions and prior cerebral infarctions.[10]

According to the AHA/ASA[11], ESO[12], CSN[10], SIGN[15] and ICSI[14], CT (without contrast enhancement) is the modality of choice for the initial brain scan. The AHA/ASA[11] states that, in most instances, a CT is the most practical initial brain imaging test and will provide enough information to make decisions about emergency management.

The CSN[10] states that although an MRI may provide more information in some cases, it is generally not recommended for the initial scan. It also states that emergency treatment of stroke should not be delayed in order to obtain multimodal imaging studies, even though they may provide additional information. If MRI is used for the initial scan, it should include diffusion-weighted sequences to detect ischaemia and gradient echo and FLAIR sequences for haemorrhage.[11]

Studies have shown that MRI is more sensitive than CT for early ischaemic changes[1,12,15], and is as sensitive for acute haemorrhagic changes.[1,11] It has also been shown to have a potential diagnostic advantage over CT in non-thrombolysis situations due to improved ability to identify acute, small cortical, small deep, and posterior fossa infarcts[15]; to distinguish acute from chronic ischaemia; and to identify subclinical satellite ischaemic lesions that provide information on stroke mechanism.[11] CT is sensitive to ICH in the acute phase, but not after 8-10 days.[1] Thus, to confirm diagnosis and differentiate ICH from haemorrhagic stroke, MRI may be preferred over CT in some presentations.[1,12,15]

Despite this, limited availability, contraindications and longer imaging time currently limits the routine application of MRI. For these reasons, CT is predicted to remain the first choice for imaging in the foreseeable future.[1,15]

Physicians' ability to reliably and reproducibly recognise early CT changes has been shown to be variable.[15] It is recommended that the greatest possible level of radiological expertise is employed to interpret images. Protocols should also be in place so that this occurs without delay.[11,14]

Time

NSF[1] recommends an 'urgent' scan, where 'urgent' is considered as soon as possible but certainly less than 24 hours, for acute stroke and high risk TIA. NICE[9] recommends immediate brain imaging within a maximum of 24 hours after symptom onset for all acute stroke patients without indications. This is in accordance with the understanding that immediate scanning in some patients will result in immediate changes in clinical management.[9] The CSN[10], ESO[12] and SIGN[15] state that initial brain imaging should be conducted immediately, but do not specify a maximum timeframe.[10,12] The CSN does, however, recommend that those TIA patients 'classified at *highest* risk of recurrent stroke [emphasis added]' undergo brain imaging within 24 hours.[10]

NICE[9] recommends that imaging is conducted within one hour of presentation if any of the following apply: indications for thrombolysis or early anticoagulation treatment; on anticoagulant; known bleeding tendency; depressed level of consciousness; progressive/fluctuating symptoms; papilloedema, neck stiffness or fever; or severe headache at symptom onset.

Wardlaw et al[30] found that out of 13 strategies assessed, the least costly and most effective strategy was for all patients to undergo immediate imaging.[1,9,10,15] NICE[9] recognises that while this approach is the most cost effective, it may be difficult to implement in all cases because of scanning availability.

Nil by mouth until bedside swallow screen (within 24 hours) for stroke

NSF[1] recommendations: All patients should be screened for swallowing deficits before being given food, drink or oral medications. Screening should be undertaken by personnel specifically trained in swallow screening (Grade C, Level I)

Patients should be screened within 24 hours of admission (Grade ✓)

Patients who fail the swallowing screen should be referred to a speech pathologist for a comprehensive assessment (Grade ✓)

Rationale

Dysphagia occurs in 27-55% of people with new onset strokes. Only about 50% of those affected recover normal swallowing ability by six months after onset.[10]

Dysphagia is associated with an increased risk of complications, such as aspiration, aspiration pneumonia, dehydration and malnutrition.[15,16] Early bedside screening is required to prevent these complications.[15] A failed bedside screen should always be followed by a complete assessment by a speech pathologist prior to any oral ingestion.[10]

In the 2007 NSF *National stroke audit clinical report acute services*[31], only half of the stroke patients included had a documented swallow screen before being given food or drink.

Resource Implications

- Bedside screening tools should be assessed and the preferred tool selected or developed for use in ED.
- Appropriate ED staff are trained to perform the selected bedside swallow screen.
- Initial bedside screening to be performed by a trained health practitioner for all newly admitted stroke patients.

Audit Measure

☑ Maintained nil by mouth prior to bedside swallow screen.

☑ Bedside swallow screen conducted within 24 hours of presentation (time of registration or triage, whichever is earliest).

Guideline Summary

Along with the NSF[1], the ESO[12], CSN[10], NICE[9], AHA/ASA[11], ICSI[14], RNAO[13], SIGN[15] and SIGN-D[16] guidelines all recommend an early bedside swallow screen for stroke patients, with the patient maintained as nil by mouth until screened.

TIA patients are not specifically mentioned in the guidelines in relation to bedside swallow screening, although standard practice does not usually require screening for TIA patients.

Reported incidence of dysphagia varies between 27-55% of people with new onset stroke (depending on the definition, and timing and method of evaluation)[1,10,13,16], but is commonly quoted at around 40%.[9] Of this percentage, approximately half do not recover a normal swallow at six months after onset.[10,13]

Patients with brain stem infarcts, multiple strokes, major hemispheric lesions and depressed consciousness are at higher risk of aspiration.[11,12]

Patients with dysphagia have an increased risk of the following complications: aspiration, aspiration pneumonia, dehydration and malnutrition.[1,10,11,13,16] Bacterial pneumonia, which is mainly caused by aspiration, is one of the most important complications in stroke patients.[12] Dysphagia is also associated with poorer outcomes, specifically higher incidence of death, disability, chest infection, and longer length of stay.[9,11,13,16]

Studies have found that implementation of and adherence to a formal dysphagia screening, referral and assessment protocol reduces the incidence of pneumonia, improves the process of care and patient outcomes.[1,16] Evidence suggests that a protocol for screening, diagnosis and treatment may yield dramatic reductions in pneumonia rates, feeding tube dependency and length of hospital stay.[10]

Screening tools

A simple bedside swallow screen, using a simple, valid, reliable tool, should be conducted on admission, or as soon as possible following admission (within 24 hours), for all acute stroke patients. This screening will identify possible dysphagic patients who should then be referred for a complete examination by a speech pathologist.[1,10,12,13,16]

Although a bedside swallow screen is useful for determining early feeding management, it may result in a false positive and/or false negative due to the variable sensitivity and specificity of the screening tool used.[9] *It is therefore essential that all patients who fail an initial bedside screen be kept nil by mouth and be referred for a complete examination by a speech pathologist. All patients, regardless of whether they pass or fail the initial bedside screen, should be monitored during their stay in the ED for symptoms of swallowing difficulties.*[10]

Numerous variations of a number of screening tools are available, although currently available data, including three systematic reviews, are not able to conclusively recommend one tool over another.[1,10]

Bedside screening generally involves observation of the patient's level of alertness to participate in the screening process, and an oromotor evaluation of the patient's oral motor function, oral sensation, and presence of a cough. This may be followed by a water swallow test, administered using a preset protocol along with monitoring for signs of impaired swallowing. Coughing during and up to one minute following test completion and/or 'wet' or hoarse voice are suggestive of an abnormal swallow.[10]

Studies have shown that a 50ml water swallow test (administered in 10ml aliquots) followed directly by an oxygen saturation test has high sensitivity (87-100%).[1]

It should be noted that a bedside swallow screen will not pick up 'silent' aspiration, thought to comprise up to half of all aspirations.[13] This necessitates careful clinical observation even after a patient has 'passed' a swallow screen.[9] An assessment of gag reflex is not a valid screen for dysphagia and should only be used as part of a more detailed assessment.[1,11,13,16]

Bedside swallow screens have been designed for use by non-specialist staff who should be trained by a specialist in the tool prior to use.[9,13,16] Any ED clinician (nurse or physician) can be trained in the screening tool. Staff should be selected according to resources, in order to maximize coverage in the ED of trained personnel. As studies have demonstrated inter-rater variability with these tools, consistent application and interpretation of the chosen tool should be ensured via a set protocol.[10,13,16]

Aspects to consider for the nil by mouth patient:

- Non-oral feeding does not prevent the aspiration of saliva.[9]
- Removal of food and drink requires immediate replacement of fluids to avoid dehydration, either intravenously, subcutaneously or via an enteral route (nasogastric tube or percutaneous endoscopic gastronomy).[1,9]
- Nil by mouth has an adverse psychological effect on patients.[9]

Aspirin as soon as possible if haemorrhage excluded

NSF[1] recommendation: Aspirin (150-300mg) should be given as soon as possible after the onset of stroke symptoms (i.e. within 48 hours) if CT/MRI excludes haemorrhage (Grade A; Level I)

Rationale

Acute phase (< 48 hours) aspirin therapy improves outcomes and reduces the risk of early recurrent ischaemic stroke.[15] Long-term aspirin therapy reduces the risk of ischaemic stroke, myocardial infarction and vascular death. There are no data from randomised controlled trials to support the use of other antiplatelet regimes in acute stroke patients.[10,15]

Resource Implications

- Protocols in place for timely access to diagnostic services (neuroimaging).
- Protocols in place for prompt post-imaging assessment by the most experienced clinician to determine appropriateness for aspirin therapy.

Audit Measure

☑ Aspirin administered within 48 hours of presentation (time of registration or triage, whichever is earliest), unless contraindicated, for all ischaemic stroke patients

Guideline Summary

The NSF[1] recommends 150-300mg of aspirin be given as soon as possible after the onset of stroke symptoms (i.e. within 48 hours) if CT/MRI scan excludes haemorrhage. The ESO[12], CSN[10], NICE[9], AHA/ASA[11], ICSI[14] and SIGN[15] similarly recommend an early initial dose of aspirin unless contraindicated (e.g. ICH, allergy or genuine intolerance, thrombolysis candidate).

As for stroke, antiplatelet therapy should be commenced in TIA patients as soon as haemorrhage has been excluded.[1]

All included guidelines recommend a similar dose of aspirin within a similar timeframe:

- NICE[9] recommends 300mg within 24 hours
- ESO[12] recommends a one-time loading dose of 160-325mg within 48 hours
- CSN[10] recommends at least 160mg immediately as a one-time loading dose
- AHA/ASA[11] recommends 325mg within 24 to 48 hours
- ICSI[14] recommends 160-325mg promptly
- SIGN[15] recommends 300mg within 48 hours.

Aspirin is the only oral antiplatelet agent that has been evaluated for treatment of acute ischaemic stroke (AIS)[9,11,15], and only doses of 160-300mg have been evaluated for treatment at the acute stage.[9,10]

Two large trials, which contribute 98% of data for the most recent Cochrane review of antiplatelet therapy in acute stroke (n=41,399)[12,32], found that 160-300mg of aspirin daily commenced within 48 hours of symptom onset was associated with improved outcomes in AIS patients.[15] With treatment, there was a significant decrease in death or dependency at the end of follow up. Treatment also increased the odds of patients making a full recovery.[1,9,10,15] It is not clear whether aspirin limits the neurological consequences of the AIS itself.[11]

Administration of aspirin within 24 hours of use of a thrombolytic agent is not recommended.[10,11,14,33]

Little evidence exists comparing the different methods of aspirin delivery.[9] The most clinically appropriate route should be selected from those available. Dysphagic patients may receive aspirin via enteral tube.[9,10,14,15]

Again, little evidence also exists for the management of aspirin-intolerant patients. Other antiplatelet agents, such as clopidogrel, may be considered for patients who are truly allergic, although they have not been evaluated in AIS.[10-12,14] Consensus from the NICE[9] guideline developers is that patients who are not truly allergic to aspirin should be administered aspirin along with a proton pump inhibitor, e.g. omeprazole, where appropriate.

Physiological monitoring and management

- neurological status
- blood glucose
- blood pressure
- hydration status

NSF[1] recommendations: Patients should have their neurological status (including Glasgow Coma Scale) and vital signs including pulse, blood pressure, temperature, oxygen saturation, glucose, and respiratory pattern monitored and documented regularly during the acute phase, the frequency of such observations being determined by the patient's status (Grade C; Level II & III-2)

Patients with hyperglycaemia should have their blood glucose level monitored and appropriate glycaemic therapy instituted to ensure euglycaemia, especially if the patient is diabetic. Hypoglycaemia should be avoided (Grade ✓)

Intensive, early maintenance of euglycaemia is currently not recommended (Grade B; Level II)

If extremely high blood pressure (BP > 220/120) exists, instituting or increasing antihypertensive therapy may be started, but blood pressure should be cautiously reduced (by no more than 10-20%) and the patient observed for neurological deterioration (Grade ✓)

Close monitoring of hydration status and appropriate fluid supplementation should be used to treat or prevent dehydration (Grade B; Level I)

Rationale

Monitoring and management of vital signs is routinely conducted for all ED patients in order to identify adverse physiological events that may require early intervention.[15]

These particular four elements have been included because they require special attention in acute stroke patients.

Neurological status: The severity of the initial neurological defect has been found to be the single most important variable in determining the rate and degree of recovery.[13] Monitoring of neurological status during the acute phase also helps to identify deterioration which can lead to earlier intervention.[13]

Blood glucose: Hyperglycaemia at the time of acute stroke is associated with poorer clinical outcomes[1], infarct progression, greater mortality and reduced functional recovery.[9-15] Hypoglycaemia may cause focal neurological deficits[11] that can be reversed by treatment.[10-12]

Blood pressure: Both hyper and hypotension in the first 24 hours of acute stroke have been found to negatively affect outcomes, although evidence regarding specific therapies is lacking.[15]

Hydration status: Suboptimal fluid intake leads to negative outcomes.[15] This is particularly problematic in patients with dysphagia. Dehydration is linked to cerebral hypoperfusion[34] and increased ischaemic penumbra* size.[35]

Resource Implications

- Clinicians to be trained in assessment and monitoring requirements of neurological status, blood pressure, blood glucose and hydration status in acute stroke.
- Definition and dissemination of information on best practices for stroke patients in the ED for blood pressure, blood glucose and hydration monitoring and management.
- Protocols developed for routine monitoring of stroke patients within ED.

Audit Measure

☑ Evidence that neurological status, blood glucose, blood pressure and fluid status were measured during initial assessment and indication of ongoing monitoring and management as required

Guideline Summary – Neurological Status

Along with the NSF[1], ESO[12], ICSI[14] and RNAO[13] also recommend for an initial neurological assessment, followed by regular monitoring.

Studies have found that neurological monitoring in the first two days following stroke enhances the benefits of conventional stroke unit care.[1]

An initial exam must be performed to assess whether the presentation is consistent with stroke, estimate the severity of the deficit and establish baseline data.[14] The single most important variable that influences the rate and degree of recovery following stroke is the severity of the initial deficit.[13] Prompt early assessment can also influence patient outcomes.[13]

Regular monitoring through the acute phase provides a standardised method of detecting neurological change.[13] This should result in early intervention in the event of a change in neurological status, which can influence patient outcomes.[13] There is little direct evidence to indicate how intensively monitoring should be carried out for non-thrombolysis patients, but it is common practice to have a minimum of four-hourly observations for the first 72 hours after stroke.[12]

The frequency of subsequent neurological assessments should be determined by the patient's status and whether a problem is identified.[1]

* Ischaemic penumbra is the cerebral area peripheral to the area of ischaemia where metabolism is active but blood flow is diminished.

Neurological scales

ESO[12] and RNAO[13] recommend using a validated neurological assessment tool, such as the Glasgow Coma Scale (GCS), the Canadian Neurological Scale (CNS) or the National Institutes of Health Stroke Scale (NIHSS), to ensure reliable assessment and documentation of a patient's status. The selection of a tool will be dependent on patient needs, organisational resources, and educational support available.[13]

NSF[1] recommends the use of the GCS as a minimum.

ICSI[14] recommends using the NIHSS for the initial assessment by physician or nursing staff in order to establish a baseline evaluation and then after resuscitation or treatment to assess change. NIHSS is recommended as it is rapid (5-8 minutes), covers all key aspects, is validated and has both inter-rater and intra-rater reliability. ICSI[14] does not recommend the full NIHSS be conducted for subsequent, regular neurological checks as this is often not feasible and not a good use of time.

RNAO[13] states that at a minimum, the assessment tool used should include:

- level of consciousness
- orientation
- motor
- pupils
- speech/language
- vital signs (TPR, BP, SpO2)
- blood glucose.

Examples of the three validated tools mentioned (GCS, CNS and NIHSS) are available in Appendix F.

Guideline Summary – Blood glucose

The NSF[1] recommends monitoring of stroke patients' blood glucose and appropriate management of hyperglycaemia during the acute phase. The ESO[12], CSN[10], NICE[9], AHA/ASA[11], ICSI[14], AHRQ[17], RNAO[13] and SIGN[15] documents also have similar recommendations.

Hyperglycaemia

Hyperglycaemia after stroke is commonly found in one third of patients, although reported prevalence varies between 8-83%, depending on cohort and definition.[1,11]

Several large clinical studies have shown hyperglycaemia directly after stroke to be associated with poorer clinical outcomes[1], infarct progression, greater mortality and reduced functional recovery.[9-15]

It is unclear as to what extent post-stroke hyperglycaemia is a 'normal' physiological response, or whether hyperglycaemia per se increases cerebral damage in the acute phase and is an independent predictor of poor outcome.[10,14] It is speculated that hyperglycaemia may be a result of physiological stress, especially in non-diabetic patients.[11,13,14] Hyperglycaemia is also a marker of more severe stroke; therefore poorer outcomes in these patients may be a result of stroke severity, and not a direct result of hyperglycaemia only.[11]

There is evidence to suggest that hyperglycaemia following stroke is associated with impaired glucose metabolism.[10,15] Glucose intolerance following stroke is found in approximately 25% of patients and is linked to higher stroke recurrence[1], while previously unrecognised diabetes mellitus and glucose intolerance preceding stroke is thought to exist in up to 42% of stroke patients.[10]

Observational data indicates that hyperglycaemia fluctuates in the first 72 hours in non-diabetic and diabetic patients, even with current best practice.[1] Gray et al (2004)[36], in AHA/ASA[11] and AHRQ[17], found that plasma glucose levels also spontaneously decline in many patients.

Early identification of hyperglycaemia in AIS is recommended.[1,13,14]

While the need for close monitoring is clear, current evidence does not point towards a specific management strategy for treating hyperglycaemia in acute stroke.[12] There is little evidence to support early, aggressive control of blood glucose in patients with mild to moderately elevated glucose levels[15], however general consensus across the included guidelines suggests that cautious treatment of patients with markedly elevated blood glucose is reasonable.[1,10,11,14]

The NSF[1] does not recommend a specific level at which to initiate therapy, but suggests monitoring and therapy as appropriate to maintain euglycaemia, although does not recommend early, intensive maintenance of euglycaemia. Similarly, the CSN[10] recommends that blood glucose be monitored regularly and treatment with glucose-lowering agents be instigated if patient has markedly elevated glucose levels.

NICE[9] and AHA/ASA[11] consider mild to moderately elevated blood glucose to be between a median of 7-9 mmol/L, and cautious treatment in patients with glucose levels above 11 mmol/L to be reasonable. ESO[12] recommends treatment of patients with glucose levels above 10 mmol/L with insulin titration. RNAO[13] recommends that glucose levels above 8.3 mmol/L be referred to a physician for further management.

The NICE guideline development group reached consensus that where possible, patients should be treated to maintain blood glucose between 4-11 mmol/L following stroke.[9]

ICSI[14] states that until there is evidence regarding the appropriateness of more aggressive treatment, usual management of hyperglycaemia (blood glucose > 8 mmol/L) with gentle dosing of subcutaneous insulin, avoiding hypoglycaemia, should be followed in a timely manner. SIGN[15] does not recommend the routine use of insulin regimens aimed at lowering blood glucose levels in patients with moderate hyperglycaemia.

ESO[12] states that the use of intravenous saline and avoidance of glucose solutions in the first 24 hours following stroke appears to reduce blood glucose levels.

Close monitoring of blood glucose with adjustment to insulin dose is required to maintain euglycaemia and avoid hypoglycaemia[1,11,14], although SIGN[15] does not recommend the routine use of insulin regimens in patients with moderate hyperglycaemia. Simultaneous administration of glucose and potassium may also be appropriate.[11]

Hypoglycaemia

Hypoglycaemia can cause focal neurological deficits that mimic AIS. Hypoglycaemia may also lead to brain injury.[11] For these reasons, prompt initial measurement and correction are important for patients diagnosed with stroke. Symptoms of hypoglycaemia can be reversed by administration of glucose.[10-12] The goal of this treatment is euglycaemia; hyperglycaemia should be avoided.[11]

Guideline Summary – Blood pressure

As well as the NSF[1], the following guidelines have a similar recommendation for regular monitoring and cautious management of high blood pressure after stroke: ESO[12]; NICE[9]; AHA/ASA[11]; ICSI[14]; and RNAO[13]. SIGN[15] recommends an active monitoring protocol should include frequent observation of blood pressure, although routine active management is not recommended.

Blood pressure (BP) abnormalities, especially hypertension, are common after stroke; in the 1997 International Stroke Trial, as reported in NICE[9], 54% of patients had systolic blood pressure (SBP) greater than 160 mmHg. BP changes may occur as a result of disturbed cardiovascular autonomic regulation, with changes in absolute BP levels and BP variability both possible. Hypertension may also indicate hypertensive encephalopathy or an increase in the risk of primary ICH.[13] Many hypertensive patients also have pre-existing hypertension that may or may not have been treated prior to the stroke.[9,11]

Both hyper and hypotension in the first 24 hours after stroke are associated with poor outcome[1,9,12,14], and poor short and long term prognosis.[11] Hypertension may also be associated with oedema and haemorrhage.[9] According to AHA/ASA[11], for every 10 mmHg increase above 180 mmHg, the risk of neurological deterioration increases by 40% and the risk of poor outcome increases by 23%. A study in AHA/ASA[11] found that an elevated baseline mean arterial BP was not independently associated with poor outcomes, but elevations in mean BP over the first days after stroke were. The same study found that an elevated pulse pressure was also associated with poor outcomes after three months.

In most hypertensive stroke patients, BP spontaneously reduces over the first 4-10 days after stroke.[9,11] This can be hastened by moving the patient to a quieter room, controlling their pain, allowing the patient to rest, or allowing them to empty their bladder.[11]

Hypotension is not as common in acute stroke and may result in extension of an ischaemic stroke and increased likelihood of a poor outcome. The underlying cause of hypotension should be sought and treated as it may be the result of a large cerebral infarct, cardiac failure, ischaemia, hypovolaemia or sepsis.[11,12,14] Patients with stroke may have depleted blood volume, in which case, correction of hypovolemia and optimisation of cardiac output are important priorities during the first hours after stroke.[11]

BP should be taken as part of the initial assessment[13] and general measures introduced to monitor and manage changes in the acute phase.[1,14]

Although strong evidence exists for lowering of BP for secondary prevention, acute BP therapy (during the first 48 hours) for both hypo and hypertension remains controversial[12] with treatment in both situations found to negatively affect outcomes.[1] There are concerns that lowering BP acutely in those patients with hypertension may have a deleterious effect by reducing cerebral flow and impairing penumbral viability, thus affecting outcome.[9] It may also be the case that the effects of lowering or elevating BP may have different effects in different stroke subtypes.[9]

Due to the limited number of studies on acute BP therapy for stroke, it remains unclear which agent should be used and whether lowering or increasing BP improves patient outcomes.[1,9,11,12,15] No specific recommendations can be made until more evidence becomes available.[9]

Close monitoring of BP, with or without therapy, is recommended.[1,15,33]

In the absence of clear data, consensus decisions were reached by a number of the included guidelines that cautious BP lowering therapy should be initiated or increased in response to severe hypertension. The point at which BP lowering therapy should be initiated varied between guidelines:

- NSF[1], AHA/ASA[11], ESO[12] and ICSI[14] recommend SBP > 220 mmHg, or diastolic blood pressure (DBP) > 120 mmHg or mean arterial pressure (MAP) > 130 mmHg (ICSI only)
- NICE[9] recommends SBP > 200 mmHg

Outside of organ dysfunction, BP should not be lowered rapidly as evidence indicates that this may be harmful.[11,12] NSF[1] recommends a decrease of no more than 10-20%. AHA/ASA[11] recommends a decrease of 15-25% with the first 24 hours. ICSI[14] recommends that, where BP lowering treatment is required in the acute phase, hypertensive agents with a short duration of action and minimal effect on cerebral blood flow are preferred. Agents that tend to cause a precipitous drop in BP should be avoided.[14]

When deciding on management, it is important to take into account the patient's acute presentation and whether or not there is a previous history of hypertension. Young patients without a previous history of hypertension may be less tolerant of elevated BP, while specific comorbidities may require more aggressive antihypertensive therapy.[11,14]

NSF[1] and ICSI[14] recommend that existing antihypertensive drugs be continued, unless the patient has symptomatic postural hypotension or other reason to withhold treatment.

Guideline Summary – Hydration level

Along with NSF[1], the ESO[12], NICE[9], SIGN[15], AHA/ASA[11], ICSI[14], and RNAO[13] guidelines have similar recommendations or statements for close monitoring of hydration levels and use of fluid supplementation to treat or prevent dehydration.

Dehydration is common in stroke patients on admission[12,15] due not only to swallowing impairments[11] but also loss of appetite, motor and sensory or visual impairment, reduced awareness, communication difficulties, depression and cognitive impairment.[1,9,13]

Suboptimal fluid intake and early dehydration are associated with slower recovery and poor outcomes following stroke.[12] These include increased complications, including deep vein thrombosis, and increased mortality.[1,11,12] Dysphagic patients are particularly at risk.[1]

RNAO[13] recommends that a nutrition and hydration screen be conducted within 48 hours of admission and then repeated after any changes in neurological or medical status. A fluid chart should be started to monitor fluid levels and help manage dehydration.

In ischaemic stroke, haemorrheological[†] disturbances may be a factor in limiting cerebral blood flow. Dehydration associated with haemoconcentration may also impair cerebral blood flow, as well as increasing thrombus formation and recurrent embolisation in cardiogenic stroke.[14] It should be noted that specialist fluid replacement therapy with haemodilution has not been shown to improve stroke outcomes.[12]

For non-dysphagic patients simple strategies have been shown to increase fluid intake including offering preferred fluids and providing supervision during meals.[1]

For those patients with dysphagia, initial fluid intake should be increased via IV or enteral routes.[1,9,12] Currently there is no clear evidence to indicate that one option is more beneficial than the other.[1,12] ESO[12] and ICSI[14] recommend treatment with isotonic fluids for maintenance of euvolemia and avoidance of dehydration, i.e. 0.9% normal saline at a rate of 75-125 ml/hr or 2-3 L/day, adjusted for febrile patients.[14] Hypotonic fluids should not be used as they promote brain swelling.[14]

[†] Haemorrheology is the study of the deformation and flow behaviours of blood and its elements, i.e. plasma, erythrocytes, white blood cells, and blood platelets.