The Newcastle upon Tyne Hospitals NHS Foundation Trust

Management of Aneurysmal Subarachnoid Haemorrhage

Version No.:	V2
Effective From:	19th April 2016
Expiry Date:	19th April 2019
Date Ratified:	19th April 2016
Ratified By:	Critical Care Guidelines Committee

1 Introduction

Trauma is the commonest cause for blood in the subarachnoid space. Non traumatic causes include vascular malformations, hypertension or coagulation disorders, but aneurysms (aSAH) are the most common cause, accounting for approximately 85% of cases. Subarachnoid haemorrhage, resulting from the rupture of a cerebral aneurysm (aSAH), accounts for about 5% of all cerebrovascular events in the UK. At least half of the remainder of atraumatic SAH cases are caused by non-aneurysmal bleeding from a ‘‘perimesencephalic’’ SAH.

This guideline is aimed at the management of adults with suspected or confirmed aneurysmal subarachnoid haemorrhage.

2 Guideline scope

Adults, spontaneous, non-traumatic SA

SAH confirmed either by:

- CT or
- Lumbar puncture: xanthrochromia on LP > 12hours after ictus.

The scope of this SOP covers initial patient care, timeliness of theatre scheduling, post procedural care, management of complications, discharge and follow up.

3 Main Body of Guideline

Patients with aneurysmal subarachnoid haemorrhage may deteriorate rapidly due to a number of potentially reversible causes, including:

Rebleeding, which is usually only a problem in unsecured aneurysms. This occurs in 5-10% in the first 72 hours.

Hydrocephalus occurs in 30% within the first 3 days, which can be treated by placement of an external ventricular drain, and is more common with heavier blood loads and intraventricular haemorrhage.

Vasospasm, and delayed cerebral ischaemia This may present as neurological deficit in the vascular territory of the treated aneurysm's parent vessel, or a completely different and distant vascular territory. It is unusual for this to be the cause of deterioration before day three following the ictus unless there has been a haemorrhage prior to the presenting bleed.

Intra-arterial thrombosis or embolism, which we think is more commonly seen after coiling, especially if a coil has prolapsed into the parent vessel. This may require Reopro, aspirin, heparin or clopidogrel, either singly or in combination. It is an important cause of deterioration early after coiling (see section 11.3).

Neurogenic pulmonary oedema and cardiac stunning may cause low cardiac output, hypoxia and further compromise oxygen delivery to a brain under pressure.

Seizures may also occur, more commonly with middle cerebral artery aneurysms.

Electrolyte disturbances may occur due to cerebral salt wasting syndrome, or, much less commonly, SIADH, or a mixture of both.

3.1 Patient Referral

- All patients with SAH suitable for treatment should be accepted for admission to the Regional Neurosciences Centre, RVI. Patients with deteriorating neurology due to ICH or hydrocephalus should be admitted as an emergency. All other patients should be transferred as soon as possible. Medical staff contacting the neurosurgical team about patients not suitable for treatment should be advised to re-refer the patient should their clinical state improve.
- CT Angiography (CTA) may be performed at the base hospital, but this should not delay transfer.
- All patients with a SAH should be started on a 21 day course of Nimodipine as soon as possible. Advice should be given to the referring medical team about starting this.
- All relevant imaging should be transferred to the RVI PACS at the time of referral.
- If patient referred from another Neurosciences centre (e.g. JCUH), it is **essential** that the vascular imaging is reviewed by Interventional Neuroradiologist (INR) **before** the patient is accepted to prevent unnecessary transfer

3.2 On admission to RVI

Good grade patients newly admitted with SAH are usually admitted to one of the neurosurgical wards and nursed in a quiet room avoiding the stress associated with invasive monitoring on ITU. Poorer grade patients will often be admitted to HDU or ITU and use of an EVD should be considered.

1. Clerk the patient noting:

• smoking, alcohol, hyptertension history

• timing of ictus

• any sentinel haemorrhages?

• family history of SAH

• any blood thinning medication

2. Perform clinical examination, but don't test for full power in all muscle groups, as straining can provoke a rebleed. Don't forget endocarditis and mycotic aneurysms as a cause of SAH. Document any motor or sensory deficit and pupillary reactions.

3. Document presenting / admission WFNS grade and later, pre-definitive treatment grade, see table 11.1.

4. Bloods, FBC, U&E, clotting, group and save, ECG (all)

5. CTA should be performed as soon as practical to facilitate early intervention. If SAH is diagnosed on CT then CTA should be performed in the same sitting.

6. Document blood load using Claassen (or modified Fisher) Grade, see table 11.2.

7. Ensure prescription with Nimodipine 60mg enterally 4 hourly for 21 days.

8. Prescribe analgesia, antiemetics and laxatives, TEDs, **no** tinzaparin

9. Consider need for EVD or lumbar drain

10. Blood pressure management: SBP up to 160mmHg is not thought to increase risk of rebleeding. Treatment of extreme hypertension should be considered in an HDU setting. Moderate elevations of blood pressure (MAP<110 mmHg) do not require treatment.

11. Neurological observations should be four hourly on a MEWS chart if MEWS = 0.

12. Prepare for angio ± proceed in the morning. Fast from 2 am.

13. There should be early liaison with treating teams, and decisions should be multidisciplinary. If the patient arrives during normal hours, the endovascular team should be informed immediately. All overnight admissions should be notified next morning.

WFNS Grade	GCS	Focal neurological deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	<7	Present or absent

Table 11.1: **WFNS Grade.**

Grade	Criteria	Incidence of DCI
0	No SAH or IVH	0%
1	Minimal/thin SAH, no IVH in both lateral ventricles	12%
2	Minimal/thin SAH, *with* in both lateral ventricles	21%
3	Thick SAH,* no IVH in both lateral ventricles	19%
4	Thick SAH,* with IVH in both lateral ventricles	40%

Table 11.2: **Claassen (2001) grade to quantify blood load on CT.** Thick SAH is defined as completely filling one or more cistern or fissure

3.3 Treatment of Ruptured Aneurysm

- **Monday to Friday** – The patient is discussed with the on-call consultant neurosurgeon at the 8am handover meeting and should be referred to the endovascular team if deemed suitable for treatment. All requests for angiography and embolisation must be made on erecord by neurosurgical staff.
- **Saturday and Sunday** – The neurosurgical on-call team should represent the interventional team at the 8am theatre scheduling meeting, prioritise the good grade SAH cases and ensure availability of HDU bed. Poor grade cases should be discussed with INR before scheduling. If the interventional neuroradiologist has agreed to treat a patient, the on-call neurosurgery team should obtain patient consent and take responsibility for preparing the patient for treatment. The consultant interventional neuroradiologist should see the patient and confirm consent prior to being anaesthetised. The interventional neuroradiologist will inform the endovascular team of the programmed time for the procedure.
- If the aneurysm is suitable for coiling and can be occluded, the INR will treat the patient within 48 hours of diagnosis. In complex cases and where complete endovascular occlusion is not possible or carries high risk, the case should be discussed with vascular neurosurgeon and patient should be offered the safest treatment.
- Some patients may require urgent clipping during evacuation of haematoma. Some may require urgent coiling prior to evacuation of haematoma. For these patients there should be a discussion between the on-call interventional neuroradiologist and vascular neurosurgeon.
- Availability of HDU bed should be confirmed prior to treatment.
- Post treatment the patient is cared for in recovery and HDU/ICU before transfer to the neuro wards.

3.4 Following coiling or clipping

3.4.1 Place of care

1. All patients should be managed on ward 18 critical care immediately post procedure.

2. Clerk the patient (noting the success or otherwise of the procedure, complications, previously noted neurological deficit, time of SAH).

3. Full examination, including groin site and distal pulses if coiled.

4. Routine post-operative bloods, ECG, ± CXR, ± ABGs.

3.4.2 Monitoring

• Pulse oximetry, ECG.

• NIBP or IABP.

• Urinary catheter.

• Monitor groin/lower extremity pulses following groin puncture for coiling.

• Daily U&E.

• Transcranial Doppler (Mon/Wed/Fri)—liaise with neuroradiology (DECT)

3.4.3 Fluid management

1. Approximately 35ml/kg day enteral intake/ 0.9% saline ± KCl, targeting euvolaemia.

2. Maintain normotension in the first instance unless vasospasm is present.

3. Establish early enteral feeding (NG feeding should stop at 06.00 if extubation is planned).

3.4.4 Nimodipine

1. Continue 60mg 4-hourly PO/NG (for a total of 3 weeks).

2. If MAP falls, try 30 mg 2-hourly.

3. Do not routinely use IV (as it is associated with hypotension).

Avoid

• Prolonged hypotension (MAP < 60 mmHg).

• Prolonged hypoxia (SaO2 < 97%, PaO2 < 10 kPa).

• Prolonged hypocapnia (PaCO2 < 4 kPa).

• Hypo and hyperglycaemia (aim for glucose of 6–10 mmol/l).

• Fluid overload.

• Hyponatraemia (Na < 135)

• Fever.

3.5 DVT prophylaxis

All patients should be given mechanical methods of DVT prophylaxis, either TED stockings or sequential compression devices, unless contraindicated. Low molecular weight heparin can be started once the aneurysm is coiled, or 24 hours after completion of surgery. It should be omitted for 12 hours before and after manipulation of an EVD, lumbar puncture or lumbar drain.

Delayed Neurological Deterioration Following Subarachnoid Haemorrhage

Neurological Deterioration after SAH

Aneurysm "secure"

Neurological Deterioration

Any of:

- New/evolving focal sign(s) (hemiparesis, dysphasia,, hemianopia, neglect)
- Fall in GCS ≥ 2 points or 1 in motor component

Identify likely cause

Common causes

(may occur in combination)

Vasospasm/DCI

Hydrocephalus

Cerebral Oedema

Seizures

Electrolyte derangement/hyponatraemia

Fever

Infection

Drugs

(Thromboembolic complication/Re-bleed)

Consider likelihood of vasospasm/DCI

High Risk Factors

Poor Grade (WFNS 4-5)

High Fisher Grade

Vasospasm previously noted

TCD velocity > 120cm/s or rapidly rising

Smoker

MAP >110 unsupported

CT (& CTA/CTP*) Brain

*Discuss with specialist.

CTA+/- CTP should be considered in high risk patients and those deteriorating ≥ 72 hours after ictus.

Aim to scan within ≤ 1 hour

Obvious cause for deterioration

Vasospasm/DCI likely

Treat

Improves

Vasospasm/DCI treatment guideline

3.6 Early Neurological deterioration after Intervention

- This requires urgent assessment & management.
- General ABC approach, ensure airway protected (if GCS < 8 call the anaesthetist and discuss the need for intubation), give 100% oxygen.
- In addition to well recognised causes of delayed neurological deterioration other procedure specific aetiologies should be considered:

3.7 Deterioration following surgical clipping

Deterioration in the early period following surgery is most likely to be due to seizures, post-op haematoma, retraction effects or ischaemia / infarction due to clip placement.

- Early post-surgery discuss with the operating neurosurgeon the need for CTA to assess vessel patency.

3.8 Deterioration following coiling

Deterioration early post coiling may be due to thromboembolic complications (3.6%) or intraprocedural rupture (2.9%.) In the NCEPOD review 14% patients failed to regain their pre-intervention neurological state within 4 hours.

- Urgently discuss with neurosurgical SpR the need for CT (to exclude a further bleed, hydrocephalus, vasospasm etc.).
- The neurosurgical SpR should then contact the interventional neuroradiologist who performed the procedure or the On-call Neuroradiologist
- Consider need for antiplatelet agents (Reopro/Aspirin) or heparinisation. If the aneurysm is large or giant (> 12mm) consider treatment with intravenous Dexamethasone to reduce possible effects of peri-aneurysmal oedema related to thrombosis.

3.9 Vasospasm and Delayed Cerebral Ischaemia

3.9.1 Introduction

Vasospasm is the clinical syndrome of delayed cerebral ischaemia in the context of subarachnoid haemorrhage and is a major cause of disability and death following SAH. It rarely presents before three days following the first haemorrhage and has usually presented by 14 days. See figure 11.1

3.9.2 Risk factors for vasospasm

Patients with risk factors for clinical vasospasm should be identified on admission so that screening for vasospasm can be instigated before overt ischaemia evolves. Risk factors include:

- Heavy blood load.
- Recent smoking.
- Poor grade (Hunt and Hess or WFNS).
- Pre-existing hypertension (unsupported MAP > 110 mmHg).
- Repeated haemorrhages.
- Angiographic vasospasm.

3.9.3 Monitoring

Serial neurological examinations

Patients at risk of clinical vasospasm should have serial neurological examinations and vasospasm suspected by the onset of a new neurological deficit or a drop in GCS of two points. Anterior cerebral ischaemia may be manifested by abulia and lack of spontaneity, middle cerebral ischaemia by a speech disturbance or limb deficit and posterior cerebral ischaemia by a hemianopia.

3.9.4 Transcranial doppler

Transcranial doppler is performed on Monday, Wednesday and Friday and should be performed as soon as practical following admission for all subarachnoid haemorrhage patients as a baseline measurement. An increase in velocity compared to baseline has a higher specificity for vasospasm that a single high value. A rapid rise in velocities of 50 cm/s in 24 hours is also indicative of vasospasm.

3.9.5 Confirmation of vasospasm

If there is clinical deterioration that may be attributable to vasospasm then a fluid bolus should be given and the double H protocol, see section 11.4.5, followed, whilst arrangements are made to confirm the diagnosis by CTA. Realistically it will take about an hour to confirm the diagnosis by CTA and the response to initial double H therapy will known by then. If the response is poor then mechanical angioplasty or intra-arterial infusion should be discussed at that time with the interventional neuroradiology team.

3.9.6 Management of Vasospasm / DCI

See algorithm on page 10

3.10 Double H protocol

The aim is to maximise oxygen delivery to the brain by ensuring that the patient is not hypovolaemic, by inducing hypertension and by ensuring there is optimum oxygen carrying capacity.

1. Make a presumptive diagnosis of vasospasm from the clinical picture, CT and TCD, following the algorithm in figure 11.1.

2. Ensure patient has arterial and CVP lines in situ.

3. Assess volume status

• Target euvolaemia, ensure not hypovolaemic.

• Fluid challenge—500-1000 ml of 0.9% NaCl over 30 minutes.

4. Hypertension.

• Start noradrenaline infusion if central access, phenylephrine if only peripheral access until central access obtained.

• Rapidly increase systolic blood pressure in 20% increments.

• Review response every 15–20 minutes. If no improvement raise to max SBP of 180–200 mmHg.

• Consider use of CO monitor to aid titration of inotrope and if high vasopressor requirement, cardiac disease, age > 50 yrs, or pulmonary oedema.

5. Hb 8–10 g/dl.

Double H therapy is generally continued for a minimum of 5 days and should be weaned off in a stepwise manner.

6. Endovascular Treatment of Vasospasm / DCI

Discuss the need for mechanical angioplasty or intra-arterial therapy with the interventional neuroradiology team if there is poor response to double H therapy for 1 hour.

Guideline for the Management of Vasospasm/Delayed Cerebral Ischaemia Following Subarachnoid Haemorrhage

Neurological Deterioration after SAH due to vasospasm/DCI

Aneurysm "secure"

Time (h)

0

Neurological Deterioration ≥1hour

Any of:

- New/evolving focal sign(s) (hemiparesis, dysphasia,, hemianopia, neglect)
- Fall in GCS ≥ 2 points or 1 point in motor component.

1

Assess volume status

Target = EUvolemia

Ensure NOT hypovolemic

Consider fluid challenge.

- Fluid challenge (eg 1000ml 0.9% NaCl or 500ml Gelofusine over 30mins)
- Aim Hb ≥ 8-10g/dL
- Keep Mg > 0.7-1 mmol/L
- Aim for normothermia

2

I M P R O V E S

Ascertain **Usual** BP

Induced **HYPERTENSION**

Avoid hypotension

HH Therapy

- Phenylephrine (peripherally) or noradrenaline (via CVC)
- Rapidly increase SBP in 20% increment(s) from usual BP.
- Review response every 15-20minutes, if no improvement raise in increments to max SBP 200 – 200 mmHg
- Consider reducing nimodipine to 30mg 2 hourly or change to iv infusion to prevent BP drops.
- Consider use of CO monitor to titrate inotrope against.

3

Discuss with NXR*

Trial of inotrope to be considered .

4

Emergency Neuroradiology opinion: trial of Endovascular Treatment

5

- ***Emergency intervention most likely to be effective if undertaken within 6 hours of deterioration so early liason advocated.***
- Inform neuroradiologist if first 20% increment in SBP does not result in improvement.

Maintain current level of treatment

Consider other rescue therapies to reduce CMRO2

3.11 Hyponatraemia & Cerebral Salt Wasting Syndrome in aSAH.

Acute hyponatremia (Na < 135mmol/L) is the most common electrolyte imbalance seen in patients with aneurysmal subarachnoid haemorrhage, occurring in one-third to one-half of patients.

- It can cause or exacerbate neurological deterioration.
- It may be caused by cerebral salt wasting and by the syndrome of inappropriate secretion of antidiuretic hormone or a combination of both. These two processes are difficult to discriminate in clinical practice.
- Both CSWS and SIADH are associated with hypotonic hyponatremia (sOsm < 275mosmol/L) and inappropriately high urinary sodium content (Na > 30mmol/L); however, they are fundamentally different in their pathogenesis.
- 90% of cases will have a degree of CSWS.

1. Patients should have U&E checked daily for the first week or whilst on iv fluids.
2. If Na < 135 patients should be assessed as per hyponatraemia guideline and treatment commenced to prevent a further fall in sodium.
3. Assess fluid/volume status to ensure euvolemia, frequently this involves a trial of 0.9% NaCl infusion with close monitoring of U&Es.
    - Fluid restriction, as usually recommended in cases of pure SIADH, is dangerous in SAH and should be avoided.
4. Supplement enteral salt intake (NaCl 1mmol/ml enterally) and reduce natriuresis with fludrocortisone 100mg tds for 7 days.
5. Continuing decline (<131mmol/L) or symptomatic patients should receive 2.7 - 3% NaCl infusion with close monitoring of serum sodium 4 hourly with the aim of limiting any rise to the normal range or maximum 10mmol/L per 24 hours.

3.12 Ward Care

- During hospital stay, the patients will remain under the joint care of intensivists, neurosurgeons and neuroradiologists. The junior neurosurgical team will be responsible for day to day management and should contact the relevant INR for advice if there is clinical deterioration.
- Most patients should stay in hospital for at least 8 days. If earlier discharge is contemplated, it should only be considered after agreement with consultant neurosurgeon and INR.
- Prior to discharge the patients receiving endovascular treatment and their relatives will be given relevant clinical information, access to the SAH support group and details of follow up by endovascular team.
- Patients requiring rehabilitation are referred to the appropriate medical team after discussion at the rehab round.

3.13 Follow Up

- Patients should be assessed for treatable risk factors (ie hypertension and smoking), and have these treated.
- All patients with endovascular treatment are followed up by endovascular team. Routinely this will be with MRA at five months and Endovascular Clinic review at

six months although exceptions might apply. Further follow up is then agreed usually with an MRA at two years and then the patient may be discharged.

- A Neuroradiology treatment plan is sent to the Consultant Neurosurgeon outlining the plan for follow up and for management of co-existing aneurysms. A copy is placed in the patient notes.
- Patients treated by surgical clipping will be followed by neurosurgical team. Some patients may need joint review.
- All SAH patients are invited to the monthly support group meeting.
- Every patient with a strong family history of two or more affected first-degree relatives and/or a history of polycystic renal disease should:
    i. be advised that their family may be at increased risk of subarachnoid haemorrhage
    ii. be considered for a referral to a neurovascular and/or neurogenetic specialist for up-to-date information and advice.

4 Training, Implementation, Resource Implications

Ongoing education will be the responsibility of consultants, senior nursing staff and nurse educators working within the neurosciences directorate and neurocritical care.

5 Monitoring Section

Audit of management and outcomes of subarachnoid haemorrhage continually occurs at the RVI and is discussed in monthly meetings involving neurosurgery, neuroradiology and critical care staff.

6 Evidence Review and Evaluation

This guideline is based on recommendations outlined in the NCEPOD report published in 2013.

7 References

Subarachnoid Haemorrhage: Managing the flow. NCEPOD 2013