

Management of Sub-Arachnoid Haemorrhage(SAH)

Aneurysmal subarachnoid haemorrhage may lead to multi-system dysfunction.

Grading of SAH- WFNS (World Federation of Neurosurgical Societies)

SAH Grade	GCS
1	15
2	13-14 and no focal deficit
3	13-14 and focal neurological deficit
4	7-12 with or without focal neurological deficit
5	3-6

Initial Management

Respiratory System

- Intubation may be required for patients with the following indications:
 - Compromised airway/breathing
 - Hypoxaemia (with high FiO₂)
 - GCS ≤ 8
 - Hypoventilation and PaCO₂ > 6 kPa
 - Hyperventilation and PaCO₂ < 3.5 kPa
- Aim PaO₂ > 13 kPa
Aim PaCO₂ 5 kPa

NPO (Neurogenic Pulmonary Oedema) can occur in the initial period after SAH ictus resulting in high oxygen requirements

Cardiovascular System

Pre definitive securing of aneurysm (SBP=Systolic Blood Pressure)

- SBP 120 -160 (Hypotension should be avoided, pre-morbid BP should be used to refine targets¹)
Agents: 1st Line [Labetalol](#)
2nd Line Hydralazine

Post definitive securing of aneurysm

- Hypotension should be avoided
Initially aim SBP ≥ 140 mmHg
- Blood pressure should be guided by neurological status (GCS, vasospasm) for blood pressure target augmentation, up to SBP~220mmHg.

Neurological system

- Nimodipine
 - For all patients with **aneurysmal** SAH (Not for traumatic SAH)
 - 60 mg **NG** 4 hourly (**all** patients should have NG/OG tube)
 - If patient on vasopressors
 - 20ml 8mg% Noradrenaline → 30mg Nimodipine 2 hrly
 - 40ml 8mg% Noradrenaline → Stop Nimodipine
- Anti-Epileptic Drugs (AEDs)
 - Seizures commonly occur at aneurysm rupture but only if seizures occur after this period then phenytoin should be commenced
 - Prophylactic phenytoin is not recommended
 - If AEDs are commenced then continuation for 3-6 months is recommended.
 - Non-convulsive status epilepticus (NCSE) in poor-grade SAH patients is present in 10-20% of patients. EEG is mandatory to exclude NCSE as a cause of decreased GCS

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Management of Sub-Arachnoid Haemorrhage

Critical Care Guidelines FOR CRITICAL CARE USE ONLY

Thromboprophylaxis

- TEDS and mechanical calf compression should be applied to all patients unless contraindicated
- Prophylactic heparin is **not given** until the aneurysm is secured (D/W Interventional radiologist or surgeon)

Fluid Management

- Plasmalyte 148 is used as initial fluid therapy aiming for euvolaemia

Initial Investigations

Routine admission bloods & Group and save

CXR

ECG

Pupillometer (NPIs)

Complications and sequelae of SAH

Hydrocephalus

- Consider hydrocephalus if neurological deterioration
Diagnosis requires urgent CT scanning and Neurosurgical review for consideration of CSF drainage

Intracranial Hypertension (raised ICP)

- Raised ICP not caused by hydrocephalus may be caused by cerebral oedema, intraparenchymal haematomas and/or infarction.
- Diagnosis requires urgent CT scanning and Neurosurgical review for consideration of ICP monitoring.
Management of raised ICP aiming for an ICP of ≤ 20 mmHg with use of hyperosmolar therapy

Rebleeding

- Rebleeding is highest in the first 72 hours after aneurysm rupture (5-10%)¹ and is prevented by BP control and securing of the aneurysm

Delayed Cerebral Ischaemia (DCI)

- DCI can occur up to 21 days after the SAH and is commonest from day 3 to day 14
- Diagnosis is initially clinical and should be confirmed by radiological imaging.

DCI Monitoring

- Regular clinical assessment of neurology should be used to identify signs of DCI (decreased GCS, focal neurological deficits) This may require regular sedation holds if patient is intubated.
- Transcranial Doppler Ultrasound (TCD) should be considered

DCI Treatment

- Maintenance of euvolaemia.
- Augmentation of BP titrated to clinical effect.
- Discuss with interventional radiology for consideration of intra-arterial therapy .

Fever

- Infection should be sought in patients with pyrexia
- Non-infective fever occurs in 60% of patients with SAH.
- Control fever above 39 degrees Celsius (antipyretics)

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Critical Care Guidelines FOR CRITICAL CARE USE ONLY

Electrolyte Abnormalities

- Hyponatraemia
 - Due to SIADH or Cerebral Salt Wasting
 - Plasma/serum osmolalities and urinary sodium should be measured to aid the diagnosis and treatment.
- Hypernatraemia
 - May be caused by neurogenic diabetes insipidus (DI) and also by hyperosmolar treatment
 - Plasma/serum osmolalities should be measured to aid the diagnosis and treatment.
 - DDAVP may be required when DI confirmed

Anaemia

- Current evidence suggests a haemoglobin target of 90g/L

Myocardial Impairment

- Myocardial impairment should be considered in patients with significant vasopressor requirement and may require cardiac output monitoring and use of inotropic agents (Dobutamine)

Neurogenic Pulmonary Oedema (NPO)

- Treatment should focus on:
 - Avoidance of fluid overload
 - Lung protective ventilation
 - Inotropic therapy if required

Glucose Control

- Blood glucose should be controlled in the range 5-10 mmol/l.

References

1. Diringer MN, Bleck TP, Hemphill JC et al. Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. *Neurocrit Care* 2011;15: 211-240