Critical Care 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 FiO_2)
  GCS ≤ 8
  Hypoventilation and $PaCO_2$ > 6 kPa
  Hyperventilation and $PaCO_2$ <3.5 kPa
- Aim PaO_2 > 10 kPa

Aim $PaCO_2$ 5-6 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 targets[1])
  Agents: 1st Line Labetalol
  2nd Line Hydralazine
  3rd Line Nicardipine

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.

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Critical Care Guidelines
FOR CRITICAL CARE USE ONLY

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)
  - IV Nimodipine if enteral route not established
  - 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

Thromboprophylaxis

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

Fluid Management

- 0.45% NaCl/5% Glucose 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

- If CSF drainage device in-situ (EVD-External Ventricular Drain/VAD-Ventricular Access Device), level of drainage will be set by Neurosurgical team and then altered to challenge CSF drainage after aneurysm secured

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 ≤ 30 mmHg with use of hyperosmolar therapy (ICP targets reviewed for individual patients with consideration of pupil size/reactivity)

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 performed if available

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.
- If EVD/VAD in-situ consider ventriculitis as cause of fever
- Control fever above 39 degrees Celsius (antipyretics/external cooling device)

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 6-10 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