
ICH SOP

1.Purpose of the Document

To guide the management of patients with an acute intracranial haemorrhage (ICH) admitted to the Department of Critical Care Medicine (DCCM)

2.Responsibility

All medical and nursing staff providing care and treatment for patients admitted to the DCCM with ICH.

3.Document Principles and Goals

Patients with an intracranial haemorrhage have not been shown to benefit from ICU care. Therefore, they should only be admitted if there is a specific indication e.g. for airway protection, procedural support, blood pressure management. The mortality and morbidity for ICH patients is high, 60% are dead within 1 year and only 20% are living independently at a year.

The specific aims of intensive care in this group of patients are:
Reversal of anticoagulation – this should be commenced in the Emergency Department

Control of blood pressure – this should be commenced in the emergency department

Desedation and neurological assessment for prognostication

Detection of deterioration

Facilitation of multi-disciplinary planning for on-going support with the neurosurgical and stroke teams.

Support of whanāu – high mortality and morbidity event

4. Inclusion Criteria

All patients admitted to the DCCM with ICH.

5. Exclusion Criteria

Patients may not receive this treatment if:
The duty intensivist (in consensus with the stroke and neurosurgical teams) determines a clinical or other indication for deviation from this document.
The ICH is unsurvivable and the patient has been admitted as a potential organ donor or for palliative care.

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6. Process of treatment

6.1 Initial Assessment

The majority of these patients are admitted through the Emergency Department or are transferred from other units after discussion with the duty intensivist, stroke service and neurosurgical team. Please record the names of the stroke and neurosurgical consultants that the patient will be admitted under.

Initial assessment should include:

History. Risk factors – those of hypertension and anti-coagulation are particularly important. It is obviously important to gain other history of events, co-morbidities and functional status, drugs and allergies.

Clinical evaluation – GCS and airway assessment, blood pressure alongside usual clinical examination

Clinical neurological examination – record the GCS, pupil examination, any focal neurological signs.

Blood tests: FBC, U+C, LFT, Coagulation (ensure a TCT is requested if on dabigatran), G+S

ECG

A priority is urgent imaging of the head. See below: Neuroimaging.

6.2 Initial stabilisation - All patients on arrival.

Airway Control + Post-intubation initial cares

Intubate and ventilate if indicated. It should be considered that stroke patients, in general, do not have improved morbidity and mortality for being admitted to intensive care and therefore should only be intubated if required for airway protection or procedural support. Induction of anaesthesia should be neuro-protective aiming to maintain an adequate blood pressure. A nasogastric tube should be inserted at the time of intubation, and a bridle placed. A urinary catheter must also be placed. A chest X-Ray should be taken to confirm position of tubes.

Following intubation, the patient should be established on an appropriate sedation regimen (e.g. propofol 100- 200mg/hour + opiate for tube tolerance) until they are transferred to the DCCM and the effects of muscle relaxants have worn off to allow for clinical assessment.

Monitoring and access

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An arterial line and adequate IV access should be inserted. It may be appropriate to wait to place the CVL until the patient has arrived in the DCCM.

Reversal of anticoagulation – this is a priority in ICH patient management.

Warfarin – FFP 15ml/kg, 10mg Vit K, consider ProthrombineX

Dabigatran – Idarucizumab 5g (Praxbind) with immediate recheck of coagulation including TCT. Coagulation screen should be repeated at 12hours. The guidance from the haematology department should be followed. Do not mix with other medications. If using a pre-existing line, flush with sodium chloride before and after Praxbind administration. Do not run other infusions using the same IV access whilst giving Praxbind. There may be a role for renal replacement therapy in these patients if they also have impaired renal function and this should be discussed with the SMO intensivist.

Rivaroxaban – there is no specific antidote available in NZ at the time of writing this SOP. Discussion between the SMO intensivist and transfusion medicine specialist regarding the use of recombinant Factor VII (NovoSeven) should be considered.

Anti-platelet agents – there is no evidence to support the use of platelet transfusion in this group (PATCH)

TXA there is no evidence that TXA administration improves mortality, there is some evidence it reduces haematoma expansion. Our current practice is not to give TXA routinely in this group of patients (TICH 2)

Blood Pressure control

A MAP of 80 – 100 mmHg should be targeted, with a systolic blood pressure under 160mmHg, whilst this hasn't been shown to improve mortality there is a tendency to improved functional outcome with aggressive and early control of blood pressure (INTERACT, INTERACT2. ATTACH). It is also important to avoid secondary brain injury associated with hypotension.

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Immediate blood pressure control				
	Drug	Dose	Renal/Hepatic	Notes
1 st	Labetalol	10-50mg IV, repeat after 5 mins PRN Max: 200mg / 24 hours	Dose adjustments generally not required	Give undiluted over at least 1 min Max effect seen by 5 mins CI: asthma, heart block, heart failure, bradycardia
2 nd	Hydralazine	5-10mg IV, repeat after 20-30mins PRN. Review ongoing use if cumulative dose reaches 50mg / 24 hours	Give half dose in renal or hepatic impairment	Give over >1 min Response unpredictable – max effect within 30mins
	Nicardipine	Infusion 3-5mg/hr. Increase by 0.5-2.5mg/hr every 15 minutes as required. Maximum 15mg/hr. Once target SBP reached – adjust by 1-2.5mg/hour to maintain BP (max 15mg/hr). If SBP falls below target, reduce by 2.5mg/hr every 15 mins until discontinued.	Start at half-dose and titrate more gradually for elderly, severe liver or renal impairment or in congestive heart failure.	If unacceptable hypotension/tachycardia occurs, stop infusion. Once resolved, restart infusion at half rate if needed Continue to monitor BP for 12 hours post infusion
<i>Intravenous nitrates should generally be avoided as they may cause vasodilation and increase the ICP</i>				
Longer term blood pressure management				
	Drug	Dose	Renal / Hepatic	Notes
	Metoprolol	Max: 400mg / 24 hours	Dose adjustments not required	Crush or give liquid for NG
	Amlodipine	Max: 10mg / 24 hours	Consider dose reduction in severe hepatic failure.	Long half-life – Give once daily. Crush for NG.

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6.3 Neuroimaging

CT head is the best initial imaging modality to confirm the diagnosis. A CTA will be required and at the discretion of the radiologist a CTV may also be required if there is a suspicion of an AVM.

6.4 Neurosurgical discussion

All patients should be discussed with the neurosurgical team. However, their role is generally limited to those patients requiring an extra ventricular drain (EVD). There may be an increased role for surgical intervention in patients with a posterior fossa bleed.

In general:

No role for routine ICP monitoring (ERICH study: mortality and intervention worse)

No role for routine surgical evacuation

No role for routine decompression craniectomy

Hydrocephalus is a poor prognostic sign. The placement of an EVD should be discussed with neurosurgery and DCCM.

7. ICU Management in DCCM

There are few evidence based, outcome changing interventions for this group of patients. We may be able to reduce the haematoma expansion by early blood pressure control and ensuring that anticoagulation has been reversed. These interventions, alongside the safe supportive cares for the patient, should be the top priority.

Standard Monitoring and Parameters

MAP 80 – 100mmHg

Systolic blood pressure <160mmHg

Temperature: 36 – 38.5

PaCO₂ – 4.6– 6.0kPa

Sats: >92%

Positioning: Standard turns

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Blood pressure control + circulatory system

See section 6 on blood pressure management. In the longer term either follow the table above or restart the patient's usual anti-hypertensive regimen.

Pain may be contributing to the hypertension so ensure the patient has had appropriate analgesia (see below).

This group of patients are at high risk of a Takotsubo's cardiomyopathy. Cardiovascular shock should be investigated with a high priority for Echo and avoidance of catecholamines.

Sedation and Analgesia

Aim to desedate and assess the patient's neurology as soon as possible. Routine sedation should be used as required for comfort, but the aim should be to make a desedated neurological assessment as soon as possible. Also consider that these patients may have a considerable headache – appropriate analgesia includes paracetamol and IV opiates.

Seizure prophylaxis

Treat clinical seizures, there is no role for seizure prophylaxis. Drug of choice is levetiracetam (20mg/kg loading dose.)

Gut

Standard indications for stress ulcer prophylaxis.

A bridled NGT with early enteral feeding is recommended. The Speech and Language Therapists (SLT) should be involved in the assessment of swallow if oral intake is appropriate.

Glucose control – if required. Target 7 – 14.1mmol as per nursing protocol

Renal and Electrolytes

Increased risk of AKI with rapid BP lowering – monitor U+C

Sodium target: 135 – 145 mmol/L

DVT prophylaxis

Initially mechanical only. Liaison with neurosurgical and stroke teams around routine prophylaxis and the re-instigation of usual anti-coagulation (e.g. for valve replacements, AF etc.) at 24-48 hours with UFH or LMWH.

Whanau

Whanau should be updated as appropriate.

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8. Prognostication

ICH is a disease process with high morbidity and mortality with 60% of patients dead at 1 year and only 20% able to live independently. It is important to prognosticate carefully to prevent a self-fulfilling prophecy.

CT: haemorrhage volume >30ml has a significantly increase mortality
Rebleed = significant increased mortality

An ongoing assessment of the patient's neurology is key to prognostication. In the intubated patient the GCS is the most important component of this, in the unintubated patient the NIHSS. (appendix) Other important prognosticating factors: age >80, GCS <8, >30ml ICH volume, NIHSS, posterior fossa

Deterioration in the neurological examination should be carefully assessed. Further CT is likely to be required as the two most common causes are re-bleed (carrying a poor prognosis) and hydrocephalus (which should be discussed with the SMO on duty and neurosurgery as to the utility of an EVD placement)

Aim to desedate and examine – clinical examination is the single most important prognostic sign.

Other tools in prognostication (repeated CT, EEG, SSEP, MRI) will be decided upon in light of clinical examination.

Appendix:

[https://adhb.hanz.health.nz/Pharmacy/Medicines-Information/Documents1/MAGs/Idarucizumab-\(Praxbind%C2%AE\)-Adult-MAG.pdf](https://adhb.hanz.health.nz/Pharmacy/Medicines-Information/Documents1/MAGs/Idarucizumab-(Praxbind%C2%AE)-Adult-MAG.pdf)

NIH Stroke scale: https://www.stroke.nih.gov/documents/NIH_Stroke_Scale_508C.pdf

References:

Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-810.
doi:10.1001/jama.2016.0287

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