Killer Headache: Intracranial Hemorrhage

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Introduction

Intracranial hemorrhage is the second most common cause of all strokes, with ischemic stroke being first. This review will cover the practical diagnosis and management of ICH in the ED.

Risk factors

There are lots of underlying pathologies causing an intracranial bleed. The most common underlying pathology overall is hypertensive vasculopathy. Others that come up include ruptured saccular aneurysm, AV malformations, sympathomimetic abuse, tumors, Moyamoya disease, cerebral venous thrombosis, TPA, and even amyloid.[1] For the record, AV malformations are the most common cause in children.[2]

Risk factors: hypertension, anticoagulant usage (especially warfarin)
Lower risk with the DOACs (direct oral anticoagulants)
Antiplatelets: small absolute increased risk with aspirin or others, but this has been debated.[3]
NSAIDs have not been found to increase risk.[4]

Pathophysiology of brain injury

Intracranial hemorrhages are so bad because of the limited, fixed volume of the skull. As the clot expands, there is increasing cytotoxic damage and edema, worsening the mass effect and increased intracranial pressure, furthering the risk of herniation.[5]
In the majority of patients, the bulk of hemorrhage expansion occurs in the first 3 hours after onset.[6]
Expansion into the intraventricular space occurs in nearly 50% of patients and is associated with worse outcomes.[7]

The greatest predictor of hemorrhage growth is anticoagulant use, contrast extravasation (Spot Sign), and ICH volume on baseline imaging.[8]

Obviously, hemorrhage enlargement is bad and clearly is associated with neurologic deterioration and worse outcomes.
So, improvements in patient outcomes from ICH may be achieved by minimizing both secondary brain ischemia and hemorrhage enlargement in the early hours following the onset of bleeding. We will cover this in the management section.

Presentation

Most cases occur during routine activity. Neurologic symptoms may develop rapidly in minutes or hours.
Common associated symptoms include headache and vomiting, together seen in over 50% of patients. Decreased level of consciousness can occur if the hemorrhage is large enough.
Small hemorrhages rarely cause headaches. They are more likely to cause progressive stroke symptoms.

Coma/stupor is an ominous sign, with the one exception being thalamic hemorrhage. Those patients may recover after blood is reabsorbed from the reticular activating system.

LOCATION	PRESENTATION
General Features	Progressive (min-hrs) and non-fluctuating encephalopathy, HTN, HA, vomiting is suggestive. Varies by site of ICH, size, direction [illegible]read, and rate of hematoma development.
Putaminal (Most Common)	Contralateral: hemiparesis/plegia and he is sensory loss Ipsilateral: gaze preference Left: Possible aphasia Right: Constructional apraxia, left visual field extinction, alloesthesia (delayed feeling of noxious stim on unaffected side)
Lobar (up to 32% of spontaneous ICH)	Presentation varies by location. In acute phase, can develop focal seizures given cortical location. Large ICH can lead to hydrocephalus.
Thalamic	Associated with HTN. Contralateral: Pansensory loss Vertical gaze impairment. Posterior hemorrhage can lead to saccadic hypometria away from hemorrhage, difficulty with smooth pursuit. Extension into ARAS can lead to somnolence.
Cerebellum (Can be a neurosurgical emergency due to space occupying nature and potential progression to herniation!)	Associated with HTN. Presents with dizziness, vertigo, N/V, truncated/limb ataxia, imbalance, skew deviation, nystagmus, ocular bobbing.
Pontine	Can be d/t HTN and ruptured AVM. Severity depends on the size. Given location to 4th ventricle, obstructive hydrocephalus is possible —> coma, miotic but reactive pupils, absent oculocephalic reflex, ocular bobbing, absent corneal reflexes, decerebrate posturing.

Neurologic symptoms and signs greatly depend on location, much like an ischemic stroke. You can expect varying exam findings given the region of the brain affected by the hemorrhage. Here's a nice table detailing these changes on the left.

Diagnosis

It goes without saying, ICH is a medical emergency. Head CT is diagnostic as it tells us the size and location and can also tell us about extension into the ventricular system, associated edema, and herniation risk. MRI is equal to CT for detection of acute ICH, but MRI is better for chronic ICH and takes more time to acquire the images,

and its availability has limitations.

Predicting hemorrhage expansion: Spot and Swirl Sign are regularly tested, high yield markers that suggest increased risk for hemorrhage expansion.

- Spot sign: small focal areas of contrast enhancement within a hemorrhage on CTA. It is linked to poorer outcomes and higher mortality. It looks like a bright white dot inside the hemorrhage.[9]
- Swirl Sign: rounded, linear, or irregular regions that are hypodense inside the hemorrhage.[10]

Labs: All patients should have a PT with INR, activated partial thromboplastin time (aPTT), and complete blood count (CBC) with platelet count. If taking LMWH, add on a factor Xa level if your hospital does it in-house. If on warfarin, an INR> 1.4 indicates they are anticoagulated. If they are on a factor Xa inhibitor, it is important to note the last time they took it. Anticoagulation can be based on a ingestion within a period of five half-lives and/or laboratory evidence of anticoagulant effect (eg, INR> 1.4 or an increased anti-factor Xa activity).

- Clinical Pearl: ICH while on warfarin usually occurs with a therapeutic level of anticoagulation (INR 2.0 to 3.5)

Management
See our handout on our website regarding the management of increased ICP. The two cornerstones of rapid therapy that must occur ASAP in the ED include reversal of anticoagulation and BP management.

Anticoagulation reversal: all anticoagulative agents should be discontinued, and their effects should be reversed.[11]

Warfarin reversal: 10 mg of IV vitamin K at 0.5-1 mg/min (takes 12-24 hrs to work). Given the delayed onset, you will also need a 4-factor prothrombin complex concentrate (PCC) at 50 units/kg or a fixed 2000 units. If 4-factor PCC is not available, you can use 3-factor PCC combined with rFVIIa or fresh frozen plasma (FFP).

> *PCC has been found to have a faster onset, less volume administered, and fewer side effects as it is manufactured and not requiring type and screen. It is the go-to agent, preferred in most institutions. Thrombosis risk is low at 14-days, around 3%.

Heparin reversal: protamine sulfate is recommended. It can be given as a slow IV infusion. Its dose is dependent on the dose of heparin given.

Any patients with severe coagulation factor deficiency or thrombocytopenia should have this corrected immediately with an appropriate factor or platelet replacement.

What about the DOACs? These are difficult, and only a few have direct reversal agents.

Dabigatran reversal: Idarucizumab. If unavailable or there if continued bleeding, use 4 factor PCC.

Rivaroxaban, apixaban, edoxaban, fondaparinux, betrixaban reversal: For these Factor Xa inhibitors, Andexanet alfa or PCC are used. There is no definitive study to support any advantage of Andexanet alfa over PCC in favorable outcomes. The former is much more expensive and not available at many facilities.

BP management- this is a debated area, with the guidelines continually shifting. Let's summarize the most basic, general recommendations.[11,12]

- For patients with SBP 160-220, lower to a target of ~140 SBP.
- Reducing below 140 SBP is not beneficial and should be avoided.
- For patients with SBP >220, lower to ~140-160 SBP.
- Preferred agents: clevidipine drip > nicardipine drip, labetalol, esmolol, or enalaprilat.

Prognosis
30-day mortality is not good, ranging from 30-50%. Perhaps more sobering, one-half of these patients die in the first 48 hours. Recurrence is 1-7%, higher in those with lobar ICH. Recurrence is most common within the first year of hemorrhage.[13,14]

References: check out our website under this topic's heading for a complete reference list.

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