

# Intracranial Hemorrhage

- Author: Zoe Oliver, MD, CCFP(EM), Assistant Professor, Department of Emergency MedicineUniversity of Manitoba, Winnipeg, Canada
- Editor: Rahul Patwari, Rush University, Chicago Illinois
- Last Update: 2015

# Introduction

Bleeding within the fixed vault of the cranium is a life-threatening emergency. Accumulated blood can cause increased intracranial pressure, which in turn damages the brain parenchyma and can lead to permanent neurologic deficit or death. Timely diagnosis and intervention by the emergency physician can be a major determinant of patient outcome.

#### Objectives

Upon completion of this self-study module, the student should be able to:

- Cite classic history and physical exam findings in intracranial hemorrhage (ICH)
- Identify different types of hemorrhage seen on computed tomography (CT) images of the brain
- Interpret lumbar puncture results in the assessment of possible subarachnoid hemorrhage (SAH)
- Outline the first steps in managing a patient with a deteriorating level of consciousness

• Outline the first steps in managing a patient with an intracranial hemorrhage

# Four categories of ICH

Intracranial hemorrhages (ICH) fall into four broad categories:

- 1. Epidural hematoma
- 2. Subdural hematoma
- 3. Subarachnoid hemorrhage
- 4. Intracerebral hemorrhage

Be sure to understand the difference between the terms intracranial hemorrhage and intracerebral hemorrhage. The former refers to all bleeding occurring within the skull, while the latter indicates bleeding within the brain parenchyma.

All intracranial hemorrhages (ICH) share some classic clinical features. Common presenting symptoms include headache, nausea, vomiting, confusion, somnolence, or seizure. There is a wide clinical spectrum: patients can be alert and conversant, or moribund. In elderly, alcoholic, and anticoagulated patients, even minor head trauma can result in devastating intracranial bleeding.

Despite these commonalities, there can be differences in the presentation of the four types of ICH:

#### Subarachnoid Hemorrhage

The classic presenting symptom of SAH is an acute onset "thunderclap" headache that may be accompanied by loss of consciousness, vomiting, neck stiffness, or seizure. Thunderclap headaches reach maximum intensity within seconds. The headache is often occipital in location. A significant proportion of patients (30-50%) will also have a warning (sentinel) headache – this is a small bleed which heralds a much larger, potentially catastrophic event.

The Hunt and Hess Grading System is one method used to describe patients with SAH.



https://saem.org/cdem/education/online-education/m4-curriculum/group-m4-neurology/intracranial-hemorrhage and the same statement of the same statement o

Asymptomatic, mild headache, slight nuchal rigidity	1
Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy	2
Drowsiness / confusion, mild focal neurologic deficit	3
Stupor, moderate-severe hemiparesis	4
Coma, decerebrate posturing	5

Recent exertion, hypertension, excessive alcohol consumption, sympathomimetic use, and cigarette smoking are risk factors for both SAH and intracerebral bleeds. However, the strongest risk factor for SAH is family history, which carries a 3-5 fold risk.

Most SAH is due to the rupture of saccular aneurysms. It is important to note that most aneurysms do not rupture.

#### Epidural Hematomas (EDH)

Epidural hematomas are accumulations of blood between the skull and the dura, and typically occur after significant blunt head trauma. Fractures of the temporal bone can disrupt the middle meningeal artery, leading to high-pressure bleeding within the cranial vault. Herniation can occur within hours if the hematoma is not evacuated, so early recognition is key.

The classic description of an EDH is brief loss of consciousness after a blow to the head, followed by a lucid period. Soon after, level of consciousness deteriorates again, possibly progressing into herniation and death. You might hear this described as the 'talk and die' phenomenon. In reality, most EDH patients either do not lose consciousness, or do not regain it.

#### Subdural Hematomas (SDH)

Subdural hematomas are extra axial blood collections between the dura and the arachnoid mater. Subdural hematomas form when bridging veins are sheared during acceleration-deceleration of the head.

Since the bleeding is venous and low-pressure, the hematoma can grow fairly slowly and the presentation can be delayed by days to weeks. This is particularly true in patients with brain atrophy, whose bridging vessels are more susceptible to shear and who can more readily accommodate the additional intracranial blood volume.

Subdural hematomas have a wide clinical spectrum. Rapid accumulation of extra-axial blood, the absence of pre-existing atrophy, and the presence of other traumatic brain injuries correspond to a worse neurologic status at presentation. As the younger brain is less atrophic, even small volumes of extra-axial blood can increase ICP and result in severe deficits.

In the pediatric population, presence of acute or chronic SDH should raise suspicion of for child abuse, although SDH can also occur as a result of birth trauma. The physician should search for other signs of 'Shaken Baby Syndrome', including retinal hemorrhages and long bone fractures. Infants with increased ICP might present with a bulging fontanelle, enlarged head circumference, emesis, failure to thrive, and seizure.

Chronic SDH is a grand imitator. It occurs more often in elderly and alcoholic patients as they are most prone to atrophy and/or coagulopathy. The most common presentation is altered mental state. Hemiparesis, headache, and falls are other possible features. As the symptoms can be subtle, the differential diagnosis broadly encompasses any potential cause of weakness or confusion in the elderly. This highlights the need have a low threshold for CT scanning any elderly patient with a change in mentation that is not convincingly explained by other pathologies.

# Initial Actions and Primary Survey

A patient with any type of intracranial hemorrhage may present with coma, rapidly declining level of consciousness, or seizure. In such cases, the priority is the ABCD's.

- Secure the Airway if there are concerns about oxygenation, ventilation, airway protection, prolonged seizure, or rapidly deteriorating clinical status. A neuroprotective rapid-sequence intubation protocol is preferred.
- Proceed with a brief assessment of Breathing, Circulation, and Disability while the patient is being pre-oxygenated, and intubation equipment and drugs are prepared. Neurosurgeons often find the documentation of a pre-intubation neurological exam to be helpful in determining prognosis. At minimum, such an assessment should include documentation of the Glasgow Coma Score, the pupillary size and reactivity, and motor strength in the four limbs. Sensation and reflexes can be included if time permits.
- Make sure to check a fingerstick glucose before intubating.

Cushing's triad describes the physiologic response to rapidly increasing intracranial pressure and imminent brain herniation. Its features are:

- 1. Hypertension
- 2. Bradycardia
- 3. Abnormal respiratory patterns

Other signs of imminent herniation are lack of pupillary reaction and/or pupillary asymmetry. Signs of imminent herniation necessitate emergent intervention (see 'Treatment').

# **Diagnostic Testing**

CT scan

Head CT is the mainstay of diagnosis in ICH. On CT, acute bleeding appears hyperdense (whiter) relative to the surrounding tissues. The subacute phase occurs between days 3 and 14, when blood becomes isodense to the brain parenchyma. It is particularly easy to miss intracranial bleeding during this phase as the blood and the brain may appear the same shade of grey. After about two weeks, blood appears hypodense (darker) relative to the brain.

The standard diagnostic pathway for SAH used to include CT followed by LP. This was because the sensitivity of CT, while reasonable, was still in the low 90's and missing a diagnosis of SAH is potentially lethal. So many sources used to advocate that an LP was necessary in all cases of suspected SAH in order to conclusively exclude the presence of blood in the CSF.

However, improvements in CT scanning technology have lead to improved sensitivities for SAH, particularly within the first 6 hours (99-100%).1 It is important to note that sensitivity can be affected by both the generation of the scanner and the experience of the reader. As such, many clinicians are now moving away from the 'mandatory LP' if the patient's CT is normal in the first 6 hours after onset of symptoms.

Large volumes of blood in the cranium can cause radiologic signs of increased intracranial pressure. These include:

- Midline shift
- Ipsilateral compression of the ventricles with or without contralateral ventricular enlargement
- Obliteration of the sulci
- Blurring of the grey-white junction













#### Lumbar Puncture

In cases of suspected SAH with a negative CT, lumbar puncture is often the second diagnostic step, particularly if the CT is delayed more than 6 hours after the onset of symptoms. An excellent (but password protected) tutorial video on lumbar puncture is available through the New England Journal of Medicine website (try accessing through your university library). Ultrasound is now being used more frequently to landmark for LP; for a video on the technique follow this link.

Two CSF features are most important:

- 1. Absence or clearing of blood.
- 2. Xanthochromia

Normal CSF does not contain red blood cells. Blood in the CSF may be a result of SAH, infection, or atraumatic tap. Many sources state that if the number of red blood cells decreases by 50% from tube 1 to tube 4, the blood can be attributed to tap trauma. However, this decrease can occur in SAH as well, so the tap should only be labeled 'traumatic' if the fourth tube is almost completely free of blood (less than 5 rbc's per high powered field).

Xanthochromia refers to a yellow or pink discoloration of the supernatant once the CSF is centrifuged. It results from the breakdown of blood cells within the CSF. Xanthochromia is determined with either visual inspection or spectrophotometry; the latter being less commonly available. The presence of xanthochromia is highly sensitive for SAH.

If the CT or LP results are consistent with SAH, some form of angiography is necessary. While conventional digital subtraction angiography (DSA) is the gold standard, it may be less readily available than CT or MR angiography. Since CT angiography is rapid and non-invasive, it is commonly used to identify saccular aneurysms once the diagnosis of SAH is confirmed.

# How do I make the diagnosis?

- 1. Suspect the illness. Complete a good history and physical exam.
- 2. Order a CT head when appropriate. For trauma cases, you might consider using the Canadian CT Head Rules.
- 3. Know the limitations of CT: small SAH's or those with delayed presentation might be hard to spot on CT. Isodense
- 4. subdurals and epidurals can also be subtle.

Canadian Head CT Rules	
High Risk for neurosurgical intervention	Medium Risk for neurosurgical intervention
<ul> <li>GCS score &lt; 15 at 2 hrs after injury</li> <li>Suspected open or depressed skull fracture</li> <li>Any sign of basal skull fracture*</li> <li>Vomiting (more than 2 episodes)</li> <li>Age greater than 65 years</li> </ul>	<ul> <li>Amnesia before impact &gt; 30 min</li> <li>Dangerous mechanism ** (pedestrian,occupant ejected, fall from elevation)</li> </ul>

#### \* Signs of Basal Skull Fracture: hemotympanum, ?racoon? eyes, CSF otorrhea/ rhinorrhea, Battle's sign

\*\* Dangerous Mechanism: pedestrian struck by vehicle, occupant ejected from motor vehicle, fall from elevation more than 3 feet or 5 stairs

Rule Not Applicable If: Non-trauma cases, GCS < 13, Age < 16 years, Coumadin or bleeding disorder, Obvious open skull fracture

## Treatment

Medical Treatment

Some tenets apply to all patients with intracranial hemorrhage:

- Assess and reassess the ABCD's
- Discontinue or reverse anticoagulation
- Prevent hypotension and hypoxemia
- Control ICP
- Prevent seizure: prophylaxis may be necessary depending on the type and extent of bleeding
- Treat fever and infection aggressively
- Control blood glucose (target 140-185 mg/dL)

#### ICP control can be managed by:

- Monitoring/lowering blood pressure in consultation with neurosurgery
- Elevating the head of the bead to 30 degrees
- Providing adequate sedation and analgesia
- If signs of rapidly rising ICP or herniation, considering mannitol or mild hyperventilation (target CO2 around 30 mmHg)

#### Surgical Treatment

In 2006, a comprehensive set of guidelines for the surgical management of intracranial emergencies was published in Neurosurgery. As a medical student, your focus should be on facilitating timely consultation with Neurosurgery for all patients with intracranial hemorrhage, unless it is clear that surgical or intensive care management would be against the patient's wishes.

### Disposition

The vast majority of patients with intracranial hemorrhage require close observation in either an intensive care unit or neurosurgical ward. Most will require intensive physiotherapy and occupational therapy before hospital discharge.

# **Pearls and Pitfalls**

- You will see far more patients who are worried that their headache represents an intracranial catastrophe than you will patients who actually have one. Learn the evidence so that you can reassure patients without having to CT everyone with a headache.
- Get that CT STAT! The sensitivity of the test and the wellbeing of the patient depend on it!
- Lumbar puncture is still an important part of management in patients whose CT scans are delayed more than 6 hours after the onset of symptoms.
- If your patient consents to an LP, be sure to warn them of the risk of post-LP headache (frequency ranges 10-20%) and other complications.

## References

- 1. Perry JJ, Stiell IG, Sivilotti ML, et al. Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: prospective cohort study. BMJ 2011; 343:d4277.
- 2. Connolly ES Jr, Rabinstein AA, Carhuapoma JR, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/american Stroke Association. Stroke 2012; 43:1711.
- 3. Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of acute subdural hematomas. Neurosurgery 2006; 58:S16.
- 4. Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of acute epidural hematomas. Neurosurgery 2006; 58:S7.

#### Selected Online Resources

- Stroke Center (http://www.strokecenter.org/): General information and neuroradiology files
- Ottawa Research Institute (http://www.ohri.ca/emerg/cdr/cthead.html): Information regarding Canadian CT Head Rules
- University of Hawaii Pediatric Radiology (http://www.hawaii.edu/medicine/pediatrics/pemxray/v5c06.html): Extensive teaching file on pediatric ICH
- University of Iowa Radiology (http://www.uiowa.edu/~c064s01/nradcerebrovascular.html): Cerebrovascular radiology teaching file