Intracranial hemorrhage made easy - a semiological approach on CT and MRI

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Learning objectives

1. To depict the basic CT and MRI semiology of intracranial hemorrhage (ICH).
2. To know how to identify the presence and age of bleeding.
3. To present the main etiologies of ICH depending on patient age, bioclinical predisposition or concurring pathology.

Background

Compartments of intracranial bleeding:

A. Intraaxial (=Intraparenchymal)

B. Extraaxial:
   - Subdural or epidural
   - Subarachnoid
   - Intraventricular

Chronology of bleeding:

- Hyperacute: < 12 hrs of evolution
- Acute: 12 hrs to 72 hrs of evolution
- Early subacute: 3 days to 7 days of evolution
- Late subacute: 7 days to 1 month
- Chronic: > 1 month

* some authors consider a hyperacute hemorrhage up to 24 hrs, acute between 1 day and 3 days, early subacute up to 7 days, late subacute between 7 and 14 days [1].

Pathology of bleeding:

- Hyperacute: ruptured vessel leads to accumulation of red blood cells in the interstitial space
- Acute: red blood cells give up all available oxygen to the adjacent cells
- **Early subacute**: intracellular proteins such as hemoglobin start to oxidize
- **Late subacute**: glucose reserves become depleted leading to red blood cell destruction
- **Chronic**: dead red blood cells constituents are cleared by macrophages

**Biochemistry of bleeding** *(Fig. 1 on page 6)*:
- **Hyperacute**: intact red blood cells containing oxygenated hemoglobin (iron in the ferrous state = Fe\(^{2+}\))
- **Acute**: red blood cells become desaturated, and oxyhemoglobin is converted to deoxyhemoglobin (iron in the ferrous state = Fe\(^{2+}\))
- **Early subacute**: intracellular deoxyhemoglobin within a hemorrhage is oxidized to methemoglobin (iron is oxidized to ferric state = Fe\(^{3+}\))
- **Late subacute**: red blood cell lysis and extravasation of methemoglobin (iron in the ferric state = Fe\(^{3+}\))
- **Chronic**: extracellular hemoglobin is oxidized by hemichromes to hemosiderin then phagocytized and accumulated in the lysosomes of macrophages (iron in the ferric state = Fe\(^{3+}\))

**Biophysics of bleeding**:
- **Hyperacute**: oxyhemoglobin has no unpaired electrons (diamagnetic): no magnetic moment and no proton relaxation enhancement
- **Acute**: deoxyhemoglobin contains the same Fe\(^{2+}\) iron but with four unpaired electrons (paramagnetic); heterogeneous distribution of deoxyhemoglobin due to confinement by the cellular membrane of the red blood cell; configuration change in the 3D-structure of the protein does not allow water molecules closer than 0.3 nm from the paramagnetic center
- **Early subacute**: methemoglobin has five unpaired electrons (highly paramagnetic); heterogeneous distribution of methemoglobin; configuration of the molecule allows approach within 0.3 nm of the paramagnetic center
- **Late subacute**: extracellular methemoglobin is no longer heterogeneous;
- **Chronic**: hemosiderin contains iron in the ferric state (strongly paramagnetic) and is insoluble in water

**Etiology**:

- **Hypertension associated intraparenchymal hemorrhage** affects patients in the average age of 50-60 years. Most commonly occurs in deep brain structures like basal ganglia, specially putamen [2] *(Fig. 2 on page 6)*.

- **Cerebral amyloid angiopathy** hemorrhage commonly occurs in lobar regions and affects the frontal and parietal lobes and is associated with microbleeds. Affects
particularly elderly patients. GRE or SWI sequences are very useful for the detection of small and diffuse hypointense regions (Fig. 3 on page 7).

- **Vascular malformations** present large hematomas and usually the presence of subarachnoid hemorrhage (Fig. 4 on page 8).

- **Hemorrhagic transformation secondary to ischemic stroke** is a relatively common entity, with combined features of a hematoma inside an ischemic stroke area.

- **Venous hemorrhagic infarct**

- **Trauma**

- **Coagulation deficiency**

- **Tumoral hemorrhage** usually involves more edema and mass effect compared to simple bleeding and the vasogenic edema is persistent on time (Fig. 5 on page 9).

Also, the hemosiderin rim has distinctive features, as presented below:

The hemosiderin rim configuration is an important factor in delineating a simple intra-axial hematoma from an intratumoral hemorrhage. In a simple intraparenchymal hematoma the hemosiderin rim is well defined, and continuous, when in the case of a hemorrhage associated with a tumor, hemosiderin deposits are discontinuous or with a random distribution because the blood-brain barrier is not intact, therefore hemosiderin-laden macrophages have access to the blood stream and the hemosiderin is resorbed [3].

- **Others causes**: venous stasis, vasculitis, eclampsy

**CT and MRI findings** [4]

**CT appearance of bleeding:**
- **Hyperacute**: slightly heterogeneous appearance with densities between 45-60 HU (similar to normal cerebral parenchyma areas)
- **Acute and early subacute**: increased density of the accumulation (80 HU) surrounded by an area of edema
- **Late subacute**: hematoma is isodense to normal cerebral parenchyma areas
- **Chronic**: hypodense appearance associating atrophy of surrounding parenchyma and ventriculomegaly

**MRI appearance of bleeding:**
- **Hyperacute**; hypo-/- isointense on T1-weighted images and high signal intensity on T2-weighted images.
- **Acute**: slightly hypo-/- or isointense on T1-weighted images and low signal intensity on T2-weighted images.
- **Early subacute**: high signal intensity on T1-weighted images and low signal intensity on T2-weighted images.
- **Late subacute**: Remaining high signal intensity on T1-weighted images. High signal on T2-weighted images.
- **Chronic**: Hemosiderin is slightly hypointense on T1-weighted images, and very hypointense on T2-weighted images.

**Differential diagnosis:**
On MRI, hemorrhage is occasionally confused with other pathologies or conditions that cause hyperintensity on T1-weighted images [5]. Examples are lesions containing fat, protein, calcification, and melanin.

- T1-weighted images can show a hyperintensity similar to that of intracellular and extracellular methemoglobin in metastases from melanoma, however, they less commonly display susceptibility on gradient recalled-echo images, and they typically show some contrast enhancement.
- Lesions containing fat, such as lipomas or dermoids, are also hyperintense on T1-weighted images, but the use of fat-suppression techniques, such as chemical shift imaging or inversion recovery sequences (e.g., short-tau inversion recovery = STIR) can help differentiate fat from hemorrhage. The presence of a chemical shift artifact may also indicate a fatty lesion.
- Hemorrhagic metastases usually show intense contrast enhancement, which is not seen in bland hematomas.
- Calcification may mimic hemorrhage, as both result in profound hypointensity on gradient-echo images. However, differences in the morphology and location of the abnormal signal intensity and in the clinical presentation suffice to distinguish the two. CT may also help differentiate these entities, as well as new and improved sequences from MRI machine developers.
- Residual gadolinium-based contrast material can resemble hemorrhage.

**Prognosis**
Important prognostic factors are:

- severity of stroke
- location of the hemorrhage
- size of the hemorrhage

*The intracerebral hemorrhage score* is used for evaluation of the outcome in hemorrhagic stroke [6]:


- GCS score 3-4: 2 points
- GCS score 5-12: 1 point
- GCS score 13-15: 0 points
- Age #80 years: yes=1 point; no=0 points
- Infratentorial origin: yes, 1 point; no=0 points
- Intracerebral hemorrhage volume #30 cm³: 1 point
- Intracerebral hemorrhage volume < 30 cm³: 0 points
- Intraventricular hemorrhage: yes=1 point; no=0 points

In a study by Hemphill et al, all patients with an Intracerebral Hemorrhage Score of 0 survived, and all of those with a score of 5 died [7].

Other prognostic factors:

- Nonaneurysmal perimesencephalic stroke has a better prognosis because of a lesser clinical impact
- The presence of blood in the ventricles is associated with a higher mortality rate
- Patients with oral anticoagulation-associated intracerebral hemorrhage have higher mortality rates and poorer functional outcomes

Images for this section:

**Fig. 1**: Biochemistry of bleeding. Evolution of a typical cerebral hematoma.
Fig. 2: Hypertensive Intraparenchymal hemorrhage. CT shows large deep hematoma in the right cerebral hemisphere, with capsulo-lenticulo-thalamic topography. Small area of edema in the right occipital lobe around a small dense nodular structure (arrow). Hematoma shows central restricted diffusion, and appears hyperintense on T2/FLAIR, with hypointense T1 periphery (methemoglobin), well delineated by a halo in hypointensity on SWI (hemosiderin). Mass effect on the right lateral ventricle with midline shift (5 mm). Findings are coherent with an subacute hematoma. Nodular enhancing lesion with adjacent edema (arrow) - metastasis from rectal carcinoma.
Fig. 3: Amyloidosis with associated lobar hemorrhage. Note the presence of numerous lobar microbleeds visible only on T2* GRE images, as well as a chronic hemorrhage in the right occipital lobe, entirely hemosiderin (no poroencephalic cavity).

Fig. 4: Subarahnoid hemorrhage. CT shows hyperdensities with extraaxial topography (intergyral spaces, tentorium, Sylvian fissure, basal cysternal spaces) compatible with acute bleeding. Areas of hiperintensity on T2/FLAIR WI, hypointensity on T1, intense hypointensity on SWI with subarachnoid distribution - subarachnoid hemorrhage. Small
accumulations in the optochiasmatic (arrow) and perimesencephalic cysternal spaces (arrowhead) which suggest subacute bleeding (methemoglobin).

**Fig. 5:** Tumor. Large mass in the right frontal lobe with an important mass effect and surrounding oedema, which crosses the midline, involving the corpus callosum and ventricular system, with heterogeneous structure associating intratumoral hemorrhage and necrotic areas, as well as intraventricular hemorrhage and small areas of SAH.
Findings and procedure details

Image acquisition protocols:

Routine head CT often requires additional spiral acquisitions in trauma, or when angio-CT is employed (detection of cerebral aneurysms or vascular malformations).

- Recommended acquisition parameters for routine head CT on a Siemens Somatom 16: sequential (axial) scan: 120 kV, effective mAs 400, rotation time = 1s, detector acquisition/collimation : 8x1,2, slice thickness 4,8 mm (recon: 2,4 mm), Kernel H31s.
- Spiral (helical) scan using fine detectors (16x0.6), rotation time = 1s, pitch = 0.85 slice thickness up to 1 mm for trauma/Angio-CT

The routine MRI protocol for the brain includes, on a GE Signa 1.5 T machine:

- Axial T2, Ax/Cor FLAIR, Axial T1 SE, 3D T1 FSPGR, DWI
- Selected cases benefit from SWI, T2 gradient recalled echo sequences, arterial and/or venous MRA (3D TOF/2D TOF), and if necessary, gadolinium administration.

CT appearance of bleeding (Fig. 6 on page 11):
- Hyperacute: extravasated blood has a slightly heterogeneous appearance with specific densities between 45-60 HU (similar to normal cerebral parenchyma areas)
- Acute and early subacute: blood clot retraction with increased density of the accumulation (80 HU) surrounded by an area of edema
- Late subacute: hematoma is isodense to normal cerebral parenchyma areas
- Chronic: hypodense appearance as the hematoma is progressively resorbed, and can associate atrophy of surrounding parenchyma and ventriculomegaly

MRI appearance of bleeding (Fig. 7 on page 12):  
- Hyperacute: long T1 and T2 relaxation times: hypo- or isointense on T1-weigthed images and high signal intensity on T2-weighted images = protein containing fluid [6]  
- Acute: preferential T2 proton relaxation enhancement shortens T2 but not T1: slightly hypo- or isointense on T1-weigthed images and low signal intensity on T2-weighted images (Fig. 8 on page 13)  
- Early subacute: proton-electron dipole-dipole interaction shortens T1 as well as T2; accentuated T2 relaxation (T2 proton relaxation enhancement) leading to high signal intensity on T1-weigthed images and low signal intensity on T2-weighted images (Fig. 9 on page 14)
-**Late subacute**: loss of T2 proton relaxation enhancement; proton-electron dipole-dipole relaxation enhancement leading to a decrease of T1. Remaining high signal intensity on T1-weighted images. T2 shortening leading to high signal on T2-weighted images = protein containing fluid *(Fig. 10 on page 15)*

-**Chronic**: no dipole-dipole interaction occurs; hemosiderin is slightly hypointense on T1-weighted images, and very hypointense on T2-weighted images, especially due to the inhomogenous distribution which leads to T2 proton relaxation enhancement. *(Fig. 11 on page 16)*

**Compartments of intracranial bleeding** *(Fig. 12 on page 17)*:

The location of the bleeding is important, as it brings information relevent for the cause of the hemorrhage, as follows:

A. **Intraaxial** (=**Intraparenchymal**) hemorrhage occurs most frequently due to hypertensive damage to blood vessel walls (hypertension, eclampsia, drug abuse), altered hemostasis, hemorrhagic transformation or other. *(Fig. 2 on page )

B. **Extraaxial**:

- **Subdural or epidural** - Subdural hematomas occur between the skull and the outer endosteal layer of the dura mater, involve meningeal or ethmoidal arteries or venous sinuses and have a typical biconvex lens appearance *(Fig. 13 on page 17)* Epidural hematomas occur between the dura and the arachnoid, involve bridging veins and have a typical crescent shape appearance *(Fig. 14 on page 18)*.

- **Subarachnoid** - occurs most commonly in head trauma; non-traumatic appears in the setting of a ruptured cerebral aneurysm or arteriovenous malformation (AVM) *(Fig. 15 on page 19)*.

- **Intraventricular** - is uncommon without parenchymal involvement; primary intraventricular hemorrhage has been noted in hypertension, anterior communicating artery aneurysm, anticoagulation, moyamoya disease and intraventricular neoplasia *(Fig. 16 on page 20)*.

**What is important to report?**

- Location and etiology of the hemorrhagic lesion
- Signes of gravity: mass effect, hydrocephaly
- Prognostic-evolution in time

**Images for this section:**
**Fig. 6:** CT appearance of ageing blood. Several factors which vary depending on the stage of the bleeding.
Fig. 7: MRI appearance of ageing blood in routine sequences.
**Fig. 8:** Acute stage hematoma. CT scan shows hyperdense hematoma in the right thalamic region, with slight mass effect on the right lateral ventricle. MRI shows the hematoma with surrounding oedema, marked hypointensity on T2 WI (susceptibility effect of deoxyhemoglobin), isodense on T1 = acute stage.
**Fig. 9:** Early subacute stage hematoma. CT demonstrates right cerebellar hemisphere hematoma with surrounding oedema. Relative hyperintensity on T1 WI appears due to the oxydation of deoxyhemoglobin to methemoglobin. DWI shows decreased signal intensity which also translates to the ADC map (T2 "dark-through") caused by low signal on the T2 images, a feature of the acute and early subacute hemorrhages [9].
Fig. 10: Late subacute stage hematoma. CT shows isodense lesion in the left lenticular-insular region, with fine hyperdense rim on the anterior side. MRI shows hyperintensity on the T2 WI caused by loss of susceptibility effect due to degradation of the red cell membranes. Note that the degree of vasogenic oedema is lesser compared to the earlier phases. Restricted diffusion on the DWI, typical of hyperacute and late subacute hemorrhages [9].
**Fig. 11:** Chronic stage hematoma. Right parietal hypodensity on CT (isodense to the CSF). The rim of hypointensity on T2* is caused by hemosiderin deposition. The rest of the hematoma has resolved to a poroencephalic cavity (note the signal intensity in the FLAIR images identical to the signal of the CSF). Surrounding gliosis visible on the FLAIR image.

**Fig. 12:** Frequency of bleeding sites in the brain (adaptation after Scarabino T et al. Emergency Neuroradiology, Springer 2006 [8]).
Fig. 13: Subdural hematoma. Band-like accumulation which follows the convexity of the right parietal, temporal and occipital lobes, left parietal and occipital lobes and surrounds the right cerebellar hemisphere (retrocerebellar and along the tentorium). Fine hypointensities on the T2* GRE sequences, suggesting hemosiderin deposits of a late subacute hemorrhage.
Fig. 14: Epidural hematoma. Small epidural hematoma in the left frontal meningeal pericerebral space, isointense on T1, hyperintense on T2/FLAIR and marked hypointensity on T2* GRE and DWI which suggests an acute hemorrhage.
**Fig. 15:** Subarachnoid hemorrhage. Linear hyperdenisties on CT, which appear hyperintense on T2/FLAIR, hypointense on T1 and T2* GRE respectively. DWI shows ependymal lining in increased signal intensity (also noticeable on FLAIR) which demonstrate deoxyhemoglobin at this level.
**Fig. 16:** Intraventricular hemorrhage. Diluted hyperdensity on CT in the lateral ventricles and basal cistern. Fluid-fluid levels also visible on MRI in T2 and T1 weighed images at the same levels.
Conclusion

1. CT is useful in evaluating recent intracranial hemorrhages, but MR is more sensitive especially in the subacute and chronic stages of bleeding and can provide superior information regarding the age, location, vessels status, and possible complications associated with ICH.
2. Important discriminants in the correct diagnosis of an ICH are the location, etiology and bleeding age.
3. Knowledge of ICH semiology in CT and MRI leads to a correct diagnosis, especially when presented as an acute neurological emergency.

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