Imaging of Intracranial Hemorrhage in Adults

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Learning objectives

The main objectives of this presentation are:

- To discuss the role of radiologists in the evaluation of patients with suspected ICHs, especially in the emergency department.

- Summarize the imaging characteristics of ICH on CT and MRI.

- Describe the main types of ICHs

Background

Intracranial hemorrhage (ICH) is a pathology that implies a poor prognosis and high mortality. Besides, it is one of the most common causes of acute focal neurological deficit. When non-lethal, it is a frequent source of complications that impact patient's quality of life and also burdens the health system. The imaging techniques are fundamental tools in the initial evaluation of these patients, being particularly useful in the emergency setting.

ICHs are a diagnostic challenge for the radiologist, due to their different forms of presentation. As radiologist we must know how to recognize the ICHs on CT and MRI to provide an early and precise diagnosis. For this purpose it is essential to incorporate the new advances in imaging techniques in order to increase our diagnostic accuracy. Moreover, a thorough knowledge of the pathophysiology and etiology may allow us to help establishing prognosis and optimize treatment.

How can we diagnose ICHs?

Computer tomography (CT) and magnetic resonance imaging (MRI) are the main imaging modalities in the diagnosis of ICH in adults.

What are the advantages and disadvantages of each technique?

Computed tomography (CT) has been the first line imaging technique in the evaluation of hemorrhages in the acute phase. The main advantages are:
- Speed.
- Reliable diagnosis of bleeding and possible complications.
- No contraindications.
However, CT is a limited technique to assess the etiology of the bleeding. It is also limited for the detection of chronic hemorrhages, as they can be isodense compared to normal brain parenchyma, thus difficulting the diagnosis.

Magnetic Resonance (MRI) is a useful tool in the diagnosis of hemorrhage. The identification of bleeding depends of the interaction between the different products of hemoglobin degradation and the pulse sequence used. A key role is played by the GRE (gradient echo sequence) and SWI (susceptibility weighted imaging) sequences, that are very sensitive to the paramagnetic products of the degradation of hemoglobin. The use of these sequences has brought great benefits, increasing our capacity to detect hemorrhagic lesions not only in the acute phase, but also in the chronic phases. An additional benefit of MRI over CT is its capacity to provide information about the evolutionary stage and the possible etiology of the hemorrhage.

The main disadvantages of MRI is the long duration of the studies, the need of certain degree of co-operation by the patients compared to CT, and the low availability of MRIs in certain settings.

Imaging findings OR Procedure details

How can we classify the ICH?
According to its:
  - Time of evolution
  - Location
  - Etiology

For a correct interpretation of the images it is important to know the pathophysiology and evolution of ICHs.

CT: the imaging characteristics are determined by the x-ray attenuation when the radiation beam crosses tissues with different densities.

MRI: The identification of bleeding depends on the interaction between the different products of hemoglobin degradation and the pulse sequence used.

The evolution of an hematoma has been divided into 5 stages: (Fig. 1 on page 7)
Hyperacute hemorrhage: < 12 hs of evolution
Acute hemorrhage: 12 hs to 48 hs of evolution
Early subacute hemorrhage: 2 days to 7 days of evolution
Late subacute hemorrhage: 8 days to 1 month
Chronic hemorrhage: > 1 month

Characteristics of the hematomas according to evolution stage on CT and MR:

CT: (Fig. 2 on page 8)
Hyperacute stage: the extravasated blood has a heterogeneous pattern with a density between of 40-60 Hounsfield units (HU). This density is equally to the adjacent normal brain parenchyma. It can be difficult to differentiate the density of the parenchyma and extravasated blood.

Acute and early subacute stage: In this phases there is going to be blood clot retraction with increased density of the hematoma. The hematoma is going to be detected as hyperdense in these phases (80 HU) with a hypodense halo due to vasogenic edema.

Late subacute stage: the hematoma is isodense to the adjacent normal brain parenchyma. This stage is characterized by the proteolysis of the globin protein.

Chronic stage: in this phase, the hematoma is hypodense and it is progressively reabsorbed. It can associate with atrophy of the surrounding parenchyma and enlarged ventricles.

MRI (Fig. 8 on page 13)
Hyperacute Stage: (<12 hs) Blood behaves as a fluid with 99% of intracellular oxigenated hemoglobin or oxyhemoglobin. In this phase the red blood cells are intact. Given that oxygenated hemoglobin has no unpaired electrons, it behaves as a diamagnetic molecule. For this reson on T1-weighted sequences, the hematoma is going to be isointense or hypointense. On T2-weighted sequences, the hematoma is going to be hyperintense. (Fig. 3 on page 8)

Acute Stage: (12 hs to 2 days) In this phase intracellular hemoglobin is progressively deoxygenated (Intracellular deoxy-Hb) and there is going to be clot retraction and reabsorption of serum. On T1-weighted images, the hematoma is going to be isoointense or hypointense in comparison to the surrounding brain parenchyma. On T2-weighted images, the hematoma is going to be hypointense due to paramagnetic susceptibility effects. The hematomas are markedly hypointense in the GRE and SWI sequences.
These sequences helps us to differentiate hemorrhagic collections of other masses. (Fig. 4 on page 9)

**Early subacute stage: (2 days to 7 days).** In this phase the deoxyhemoglobin is gradually converted to methemoglobin. The methemoglobin is paramagnetic and confined to the intracellular space. The oxidation of deoxyhemoglobin to methemoglobin starts from the periphery. On T1-weighted the hematoma is hyperintense and on T2-weighted is hypointense due to methemoglobin inside the intact red blood cell. (Fig. 5 on page 10)

**Late subacute stage: (8 days to 1 month):** In this phase, there is lysis of red blood cells and methemoglobin is released to the extracellular space. On T1-weighted the hematoma is hyperintense and on T2-weighted there is an increase in signal intensity of the hematoma. The appearance of the hematoma in both sequences is hyperintense. (Fig. 6 on page 11)

**Chronic phase: (> 1 month):** The liquid and the clot protein have been degraded and reabsorbed almost completely. Due to hemosiderin have nuclei with unpaired electrons, it is superparamagnetic. For this reason the hematoma is hypointense on T1 and T2-weighted images. On GRE and SWI sequences, it can be observed a persistent marked hypointense due to glial hemosiderin staining. (Fig. 7 on page 12)

**Location:** (Fig. 9 on page 14)
Intracranial hemorrhage can be intraaxial and extraaxial

1. **Intraaxial hemorrhage:** these can occur in the cerebral hemispheres, the cerebellar hemispheres or brainstem. (Fig. 10 on page 15)

2. **Extraaxial hemorrhage:** the blood can be located into of the ventricular system, subarachnoid spaces, subdural space, and epidural space.

**A Epidural hematoma:** it is a blood collection extra-axial, results more frequently from the laceration of the meningeal arteries.
Location: blood between the skull and the dura mater.
Cause: It usually occurs after a severe head trauma and the temporal lobe is affected more frequently.
Characteristics: It is a well defined biconvex collection. It does not exceed the skull sutures and it can cross the midline. (Fig. 11 on page 16)
B Subdural Hematoma: It is a blood collection extra-axial and generally it occurs by laceration of a cortical vein.
Location: They are located between the dura mater and the arachnoid.
Cause: Trauma is the most common cause.
Characteristics: Crescentic extraaxial collection. It can cross skull sutures. It never cross the midline due to it is reflected along the brain falx. (Fig. 12 on page 17)

C Subarachnoid hemorrhage: It is a blood collection extra-axial, can be traumatic (laceration of cortical veins or arteries localized in the subarachnoid space or cortical contusions with extravasation of blood into the subarachnoid space) or non-traumatic (ruptured aneurysms).
Location: There is an accumulation of blood within the subarachnoid space.
Cause: Trauma is the most common cause.
Characteristics: Blood within subarachnoid spaces between pia and arachnoid membranes.
CT is very sensitive in detecting hyperdense blood in the basal cistern and subarachnoid space. On MRI, the blood is diluted with the cerebrospinal fluid signal (CSF). For this reason it is difficult to detect on normal T2-weighted sequences. On the other hand on T2-FLAIR sequences subarachnoid hemorrhage is easily detected as its signal is not suppressed like in normal CSF. (Fig. 13 on page 18) (Fig. 14 on page 19)

D Intraventricular hemorrhage: May be due to an extension of intraparenchymal hemorrhage or from the reflux of blood from the subarachnoid spaces. (Fig. 15 on page 20) (Fig. 16 on page 21)

Etiology (Fig. 17 on page 22)
Intraaxial hemorrhage can occur for different causes. The most common causes are hypertensive hemorrhage, cerebral amyloid angiopathy, hemorrhagic stroke, aneurysms and vascular malformations, intracranial neoplasms, head trauma, coagulopathy, drugs, etc.

A- Intraparenchymal hemorrhage associated with hypertension
This entity affects patients in the average age of 50-60 years. Most commonly occurs in deep brain structures like basal ganglia, (specially putamen). (Fig. 18 on page 23)

B- Intraparenchymal hemorrhage associated with cerebral amyloid angiopathy
Commonly occurs in peripheral lobar regions and affects the frontal and parietal lobes. Usually associated with lobar microbleeds. Affects particularly elderly patients. GRE or
SWI sequences are very useful for the detection of small and diffuse hypointense regions. (Fig. 19 on page 24)

C- Vascular malformations
Are found in lobe regions and presents a larger hematomas associated.

D- Hemorrhagic transformation secondary to ischemic stroke
T2 FLAIR, GRE or SWI sequences, diffusion-weighted and ADC can give us information about non-hemorrhagic areas and show the blood within the infarct. (Fig. 21 on page 26) (Fig. 22 on page 27)

E- Haemorrhage secondary to tumors:
There is more edema and mass effect compared to simple bleeding and the vasogenic edema is persistent on time. (Fig. 20 on page 25)

Images for this section:
**Fig. 1:** Classification of hemorrhages according to the stages of evolution

**Fig. 2:** Axial CT in different patients
- **A:** Acute hematoma: hyperdense hematoma of 48 hs of evolution, characterized by hyperdense blood surrounded by a hypodense halo due to vasogenic edema.
- **B:** Late Subacute hematoma: subdural hematoma isodense with the brain parenchyma in a 67 year-old-male (1 month of evolution).
- **C:** Chronic hematoma: 80 year-old male with hypodense collections located in both hemispheres corresponding to evolved subdural hematomas (5 months of evolution).
Fig. 3: Hyperacute hematoma: In this image, it can be observed on sagittal T1-weighted MR a isointense hematoma and axial T2-weighted MR the hematoma is hyperintense. It is located in the right cerebellar hemisphere.
Fig. 4: Acute Hematoma: Sagittal T1-weighted MR shows an isointense hematoma (arrows) surrounded by a hyperintense halo. Axial T2-weighted MR shows a hypointensity. It corresponds to an acute hematoma (8 hours of evolution). There is also another hematoma of different time of evolution.
Fig. 5: Early subacute Hematoma: Same patient as in figure 3 in early subacute stage. The hematoma is hyperintense on T1-weighted MRI and hypointense on T2-weighted MRI, due to methemoglobin inside intact red blood cells.
Fig. 6: Late Subacute intracerebral Hematoma: 67 year-old-male. On sagittal T1-weighted and axial T2-weighted MRI a hyperintense intracerebral hematoma is seen, located in the right cerebral hemisphere. Hyperintensity is due to extracellular methemoglobin. (25 days of evolution).
Fig. 7: Chronic hematoma: Sagittal T1-weighted MRI and axial T2-weighted MRI in the same patient, show a hypointense hematoma in chronic phase (5 months of evolution). There is, also dilatation of the right lateral ventricle due to adjacent parenchymal atrophy.
Fig. 8: T1-weighted, T2-weighted, T2 FLAIR and SWI sequences. Hematomas in different stages of evolution.
Fig. 9: Classification according to the location.
Fig. 10: Intracerebral Hematoma. In these images, there are two adjacent hemorrhagic foci, located in right parieto-occipital region. These hematomas have different times of evolution as seen on CT and MR images. CT shows a hyperdense hematoma in the anterior and superior portion, indicating an acute stage (14 hs of evolution). The hematoma located in the posterior and inferior portion has a lower density, corresponding to an evolved hematoma. Axial T2-weighted MRI and sagittal T1-weighted MRI show a hypointense area in the upper hematoma, corresponding with the hyperdense area on CT. These findings indicate an acute hematoma. The inferior hematoma is hyperintense on axial T2-weighted, coronal T2 FLAIR and sagittal T1-weighted sequences, indicating a hematoma of late subacute evolution. There is vasogenic edema surrounding both hematomas and mass effect over the right lateral ventricle.
Fig. 11: Epidural hematoma: This patient suffered a severe head trauma after falling. In this axial CT image, it can be observed a typical presentation of epidural hematoma, located in the left temporal region (arrows).
Fig. 12: Subdural hematoma: Axial and coronal CT. Hyperdense hematoma of 14 hours of evolution located in the left side, corresponding to a subdural hematoma. Axial T2-weighted MRI and coronal T1-weighted MRI, in another patient, show a large subdural hematoma located in the right side. The hematoma is hyperintense in both sequences due to blood breakdown products, indicating a late subacute hematoma. This hematoma is causing mass effect with compression of the ipsilateral ventricular system and sulcal effacement.
Fig. 13: Subarachnoid Hemorrhage: Axial, coronal and sagittal CT in a 55 year-old-male, after a head trauma. Hyperdense collections in some left frontal and temporal sulci, indicating a subarachnoid hemorrhage.
Fig. 14: Another case of traumatic subarachnoid hemorrhage.
Fig. 15: Intraventricular hemorrhage: CT shows an acute intraventricular hemorrhage in a 60 year-old man. The blood is located in both lateral ventricles due to an extension of an intraparenchymal hematoma, located in the left frontal lobe. T2-weighted, T2 FLAIR and SWI show a hypointense hematoma and intraventricular hemorrhage. Sagittal T1-weighted image shows hyperintensity of the intraventricular hemorrhage (early subacute stage).
Fig. 16: Intraventricular hemorrhage: In another patient, axial, sagittal and coronal CT show a hyperdense hematoma located in the left caudate (arrow) with contamination of the ipsilateral lateral ventricle and third ventricle system of acute stage. The hematoma and the intraventricular hemorrhage are hyperintense on sagittal T1-weighted images. The intraventricular hemorrhage is hypointense on T2-weighted and SWI (arrow), indicating an early subacute stage.
Fig. 17: Classification according to the etiology.
**Etiology of bleeding**

**Hypertension**

Fig. 18: Intraparenchymal hemorrhage associated with hypertension. Sagittal T1-weighted and axial T2 FLAIR-sequences MRI, show a hyperintense hematoma located in the left putamen. This location is typical of hypertensive hemorrhage. SWI shows a halo of hemosiderin in the periphery of the hematoma (arrow. An hemorrhage of chronic evolution and of the same etiology is seen in the contralateral basal ganglia.
Fig. 19: Intraparenchymal hemorrhage associated with cerebral amyloid angiopathy. A 70 years-old-male patient with lobar hemorrhagic collections in different stages. SWI identified multiples hypointense foci (arrow) in relation to microbleedings. Their distribution and multiplicity are typical of hemosiderin deposits in the context of cerebral amyloid angiopathy.
Fig. 20: Intraparenchymal hemorrhage associated with tumor. This patient suffered a stroke in the right cerebral hemisphere few months ago. Subsequently he was admitted to our hospital for neurological deficit. T2-weighted, T2 FLAIR and SWI sequences show a hemorrhagic lesion in the left temporal lobe. The initial diagnosis was of a hemorrhagic infarction. 7 months later, this lesion had enlarged and presented pathological enhancement after the administration of contrast (arrow). The final diagnosis was a tumoral lesion with bleeding.
Fig. 21: Hemorrhagic transformation of an ischaemic stroke. Acute phase. Diffusion shows restriction to the movement of free water related to citotoxic edema (hyperintense in b1000 and hypointense in ADC). This finding is compatible with stroke of acute evolution in the territory of the left middle cerebral artery. An heterogeneous lesion located in left basal ganglia suggest an associated hemorrhage. SWI confirms a hematoma located in the left basal ganglia.
Fig. 22: Hemorrhagic transformation of an ischaemic stroke. Chronic phase. T2-weighted MR shows a hyperintense lesion located in the right occipital lobe with hemosiderin deposits (arrow) on SWI sequence, related to a cerebral parenchymal hemorrhagic infarct. There is no free-water movement restriction in ADC.
Conclusion

CT and MRI are useful tools for the diagnostic of intracranial hemorrhage in adults in the emergency services. CT is the technique of choice for the detection of bleeding of acute evolution because it gives us a quick and reliable information and because of its greater availability. MRI is also a useful tool in the detection of bleeding of acute stage but is more sensitive in the subacute and chronic phases of bleeding. In addition, allows us to know the time of evolution and the possible cause of the hemorrhage.

References


Personal Information