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# Neuroimaging in Intracerebral Hemorrhage

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Additional information is available at the end of the chapter

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## Abstract

Hemorrhagic stroke accounts for 15% of all strokes but results in nearly a third of the mortality. Neuroimaging forms the mainstay in diagnosis, which has resulted in improved treatment outcomes. The mandate of neuroimaging includes management, risk assessment, prognostication, and research. This involves rapid identification not only to direct treatment but also to discover the underlying etiology such as vascular malformations or tumors, monitor the evolving course of the hemorrhage and rapidly identify complications. While computed tomography (CT) remains the imaging of choice to rapidly detect acute hemorrhage, growing evidence shows that magnetic resonance imaging (MRI) is comparable to CT for detecting blood in the immediate setting and superior in this regard at subacute and chronic time points. Several advances have been made in the image sequencing protocols to detect bleeds at varying time points and to distinguish possible etiology. Initial and serial imaging is used to identify patients who may benefit from intervention. Advances in this field such as diffusion tensor imaging and functional MRI are being studied for their impact in understanding the extent of injury and possible recovery mechanisms, possibly allowing prognostication for patients.

**Keywords:** intracerebral hemorrhage, hemorrhagic strokes, neuroimaging, computed tomography, magnetic resonance imaging, vascular malformations

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## 1. Introduction

Hemorrhagic stroke is responsible for 15% of all strokes occurring annually in the United States and has a high mortality rate of 29% [1]. About two-thirds of these strokes are intracerebral hemorrhage (ICH) and one-third are subarachnoid hemorrhage (SAH) for which neuroimaging forms the mainstay in diagnosis; as history, clinical symptoms and signs are often nonspecific but have resulted in improved treatment outcomes.

## 2. Definitions

Intracranial hemorrhage is the accumulation of blood within the skull, the parenchyma and/or the meningeal spaces and/or other associated potential spaces (epidural and subdural). The term intracerebral hemorrhage refers to bleeding in the brain parenchyma (white or gray matter). The term subarachnoid hemorrhage is used for blood collection in the subarachnoid space (i.e., in the space between the pia and arachnoid meningeal layers). ICH is classified conventionally as primary or secondary, based on its causes, with primary ICH (80–85% of ICH) related to hypertension and amyloid angiopathy and secondary ICH having varied etiologies such as drugs, malformations, tumors, vasculitis, etc. [2]. This has given way to different systems of classification such as SMASH-U and lobar vs. deep.

Our chapter aims to:

- Explain the various modalities which can be used in the detection of ICH with a brief description of their mechanism.
- Provide advantages and disadvantages of each method.
- Provide image descriptions of common findings in ICH with sample images.
- Explain the modalities used in Detecting the etiology of ICH and complications with image findings.
- List some common sequences in practice today.
- Explain the expanded role of imaging from management to prognostication.

The goals of neuroimaging include:

The main goal of neuroimaging in a patient with suspected cerebral hemorrhage is to find a modality with perfect sensitivity and specificity. Rapid and accurate identification of hemorrhage is critical in planning therapy.

- Detecting intracerebral hemorrhage (ICH).
- Detecting etiology.
- Detecting tissue at risk.
- Detecting complications such as vasospasm, mass effect, and herniation.
- Detecting hemorrhagic complications in ischemic infarcts.
- Assessing risk factors for hemorrhage.
- Detecting resolution—monitoring and management.
- Prognostication of recovery.
- Assisting in research endeavors to advance both knowledge and treatment in ICH patients.

### 3. Early detection

#### 3.1. Need

ICH is a medical Emergency, with rapid diagnosis and management being vital due to early and rapid hematoma expansion and clinical deterioration. This increases the mortality to as high as 75% in patients with pre-hospital neurological decline and results in worsened long term outcomes [3]. Since clinical features such as severe headache, high blood pressure, vomiting, loss of consciousness, and rapid progression cannot always be relied upon as being specific for hemorrhagic stroke, neuroimaging is mandatory.

#### 3.2. Protocol

Rapid neuroimaging with noncontrast computed tomography (NCCT) or magnetic resonance imaging (MRI) is recommended to distinguish ischemic stroke from ICH [3]. Noncontrast CT (NCCT), perfusion CT, and CT angiography (CTA) are usually used in the hyperacute stroke setting. The imaging appearance of the ICH is closely linked to the physiological processes at play during and after the bleeding event.

### 4. ICH appearance on CT

#### 4.1. NCCT

Bleeding into the cerebral parenchyma results in a hematoma consisting of proteins, serum, platelets, white blood cells, and red blood cells (with hemoglobin), and the concentration of the latter (relative to plasma) is responsible for the degree of attenuation of the X-ray beam. The varied components of the hematoma give it a heterogeneous appearance. The attenuation of blood with a normal hematocrit (45%) is much higher (56 Hounsfield units—HU) than gray matter (37–41 HU) and white matter (30–24 HU) resulting in the 'brighter' or 'whiter' region in patients with a normal hematocrit [4] (**Table 1, Figure 1**).

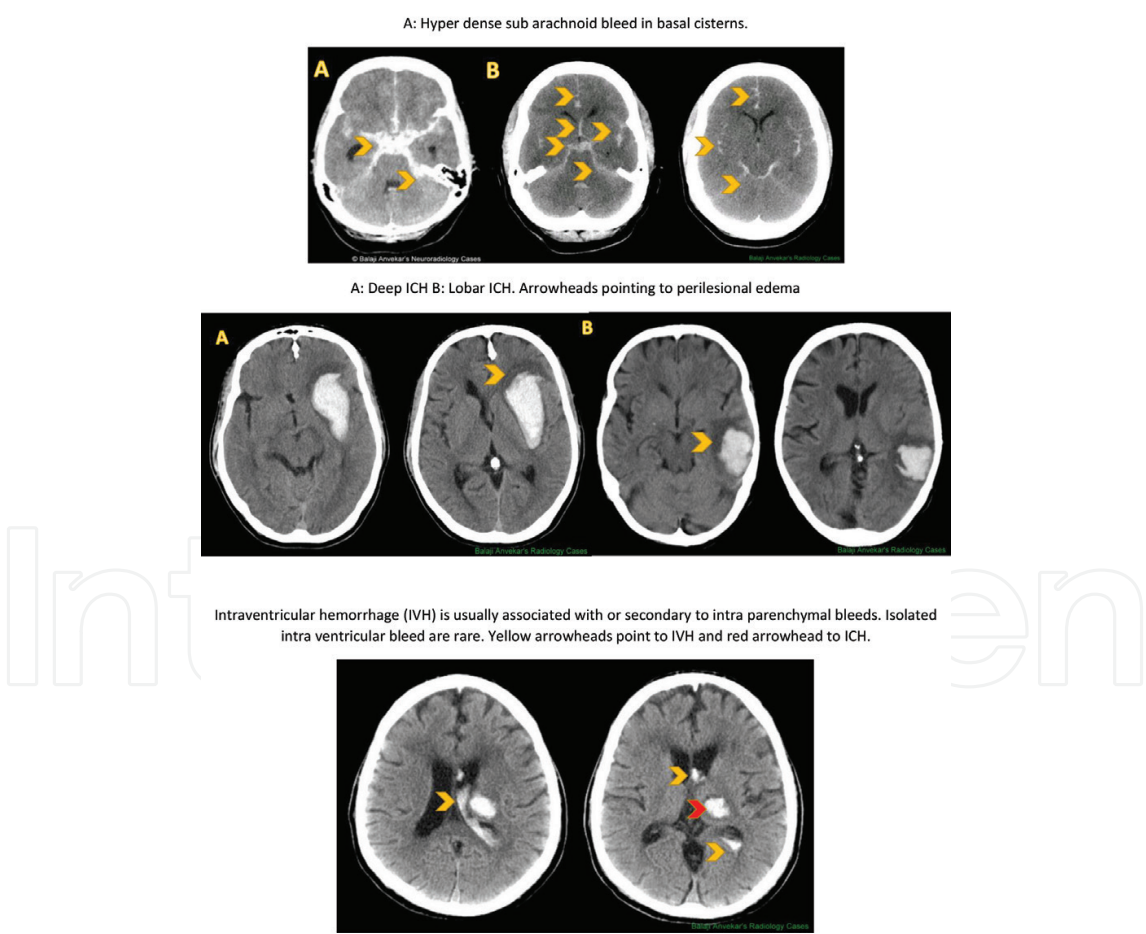
At the immediate onset of the bleed, (hyperacute phase) the blood has a similar attenuation as that of the cortex and is hard to distinguish. However, within minutes after a clot forms (platelet clumps, and proteins are consumed), the degree of attenuation increases and continues to increase over the hours as the clot retracts and extrudes serum, seen markedly in the center of the hematoma.

Within hours, the hematoma is surrounded by vasogenic edema which may last up to 2 weeks. Vasogenic edema is the extravasation of fluid and proteins into the extracellular spaces, due to the loss of integrity of the blood brain barrier and hence has a hypoattenuated or "darker" appearance on CT scan images, surrounding the hematoma.

In a large bleed, a fluid level may be visualized on imaging within hours of onset as the cellular debris collects in the more gravity-dependent portion, giving that area a higher attenuation.

Time	Process	CT
Immediately on extravasation	Bleeding into parenchyma	Hyperattenuated (brighter) lesion Heterogeneous due to varied cellular components
Minutes	Clot formation, serum extruded	Increasing intensity, marked in the center of hematoma
Hours–2 weeks	Vasogenic edema surrounds bleed	Hypoattenuated or “darker” appearance on CT scan images, surrounding the hematoma
Hours	Cellular debris settles in the gravity dependent part	Fluid level (with hyperattenuated dependent portion)
Days–weeks	Clot breakdown by scavengers (macrophages)	Decrease in attenuation beginning at periphery toward the center
2–3 weeks	Resolution of clot	Same intensity as white matter
Weeks-months	Cavity: collapsed or filled with cerebrospinal fluid	Small slit like cavity which may or may not be visualized
Months	Encephalomalacia	Hypointense (darker) area at lesion site

**Table 1.** CT imaging of intracerebral hemorrhage.



**Figure 1.** Immediate detection of hemorrhage using NCCT: the first image has yellow arrowheads pointing to hyper-dense areas representing sub-arachnoid bleeding in the basal cisterns. The second image has yellow arrowheads pointing to peri lesional edema around deep ICH (A) and lobar ICH (B). The third image shows Intraventricular hemorrhage (IVH) (yellow arrowhead) which is usually associated with or secondary to intra parenchymal bleeds (red arrowhead).

The breakdown of the clot by natural scavengers such as macrophages continues over several days and results in a decrease in attenuation beginning at the periphery and working its way toward the center, gradually over a period of 4–9 days having the same attenuation as cortical gray matter and eventually after 2–3 weeks, having a similar attenuation as white matter.

This explains the difficulty in using CT scan to detect subacute hemorrhages due to similar appearance (iso-attenuation) with the parenchyma, and it is often scarce or difficult to distinguish mass effect and edema, which is fortunately obviated by the use of MRI.

The hematoma is eventually resolved into a small or slit like fluid filled cavity which may or may not be appreciated on CT scan. Eventually the only evidence of the hemorrhage may be encephalomalacia (hypointense or 'darker' appearance) at the location.

The use of contrast material with CT scan, usually performed nonemergently for reasons other than initial detection does not usually show enhancement although it may develop after weeks or months at the periphery of the resolving hematoma, which may make it hard to distinguish it from tumors or abscesses [5].

#### **4.2. CT angiography (CTA)**

Contrast extravasation within the hematoma is used to identify patients at risk for hematoma expansion, which is commonly referred to as the "Spot Sign," which is used as a predictor of poor neurological outcomes. This can be used to institute prothrombotic therapies such as Factor VII and increased surveillance to avoid poor outcomes. CTA performed within 96 hours of the event has >95% sensitivity and specificity in identifying vascular malformations. However, this must be balanced with the severe contrast associated complications such as allergy and nephropathy as well as possible effects on the blood brain barrier.

#### **4.3. Quantification**

NCCT is used to quantify hematoma volume and monitor its evolution. ICH volume is calculated using the ABC/2 method. A = greatest hemorrhage diameter, B = diameter at 90° to A, and C = approximate number of CT slices with hemorrhage multiplied by slice thickness. This method however has been shown to have a large margin of error especially for irregularly shaped bleeds (by an excess of 7.33 cm<sup>3</sup> when compared to manual planimetric method [6]).

### **5. ICH appearance on MRI**

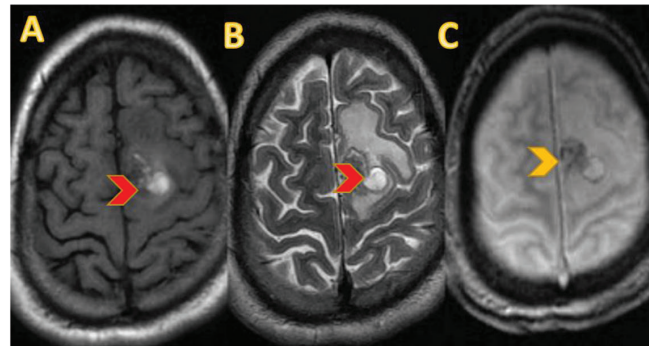
The MRI appearance of ICH is based on the evolution of the hematoma over time (as explained above for CT) and the corresponding signal characteristics. The MR signal characteristics in turn are dependent mainly on the chemical state of the iron molecules in hemoglobin as well as the state of the red blood cell membrane (Table 2, Figure 2).



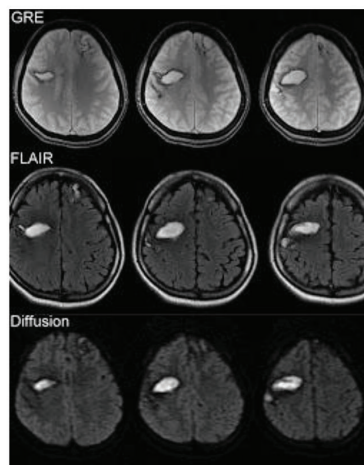
Time	Process	State of iron	State of membrane	T1 effect	T2 effect	T1 weighted images	T2 weighted images
<b>Hyperacute</b>							
Immediate -hours	Bleeding into parenchyma	Oxygenated (diamagnetic iron)	Intact	none	No susceptibility effect	Hypo- or isointense	Mildly hyper- or iso intense
	Deoxygenation at periphery begins	Deoxygenated at periphery (paramagnetic iron)	Intact	None	Susceptibility effect +	No change	Hypointense rim at periphery
<b>Acute</b>							
Hours–days	Deoxygenation from the outside in	Deoxygenated (paramagnetic iron)	Intact	None	Susceptibility effect +	Hypo- or isointense	Hypointense lesion
	Oxidation of iron at periphery	Met-hemoglobin at periphery	Intact	Decrease in T1 relaxation	None	Mild hyperintensity at periphery	None
<b>Early subacute</b>							
Days-weeks (usually 1 week)	Oxidation of iron to ferric state	Met-hemoglobin	Intact	Decrease in T1 relaxation	Susceptibility effect +	Hyperintense lesion	Hypointense lesion
<b>Late subacute</b>							
Week-months	Oxidation of iron to ferric state	Met-hemoglobin	Degraded	Decrease in T1 relaxation	No susceptibility effect	Hyperintense lesion	Hyperintense lesion
<b>Chronic</b>							
Several months	Protein breakdown	By-products	Degraded	Reduced signal intensity	Reduced signal intensity	Decreased hyperintensity	Decreased hyperintensity
	Iron deposited as hemosiderin at rim	Hemosiderin	Compartmentalized in molecule	None	Susceptibility effect +	Isoattenuation.	Hypointense rim
	CSF-filled cavity or slit-like cavity	—	—			Hypointense fluid- filled or slit-like cavity	Hyperintense fluid- filled or slit-like cavity
• CSF = cerebrospinal fluid.							

**Table 2.** MR imaging of intracerebral hemorrhage.

MRI shows a focal left parietal para sagittal bleed with low signal intensity (yellow arrowhead) on T2\*GRE (image C), T1 and T2 (A & B) show bright hyper-intensity (red arrowhead) due to Meth Hb, a blood degradation product indicating subacute stage



Subacute hematoma with restricted diffusion



**Figure 2.** MR appearance of ICH: (Top) MRI shows a focal left parietal para sagittal bleed with low signal intensity (yellow arrowhead) on T2\*GRE (image C), T1 and T2 (A & B) show a bright hyper-intensity (red arrowhead) due to Meth Hb, a blood degradation product indicating a sub-acute bleed. (Bottom) MRI showing sub-acute hematoma with restricted diffusion.

Iron within an intact red cell membrane causes shortened T2 relaxation times known as Susceptibility effect (which is lost when the membrane degrades and the hemoglobin/iron is no longer sequestered in the cell). Paramagnetic iron has a greater shortening effect on T1 relaxation times. Diamagnetic iron is an iron molecule with no unpaired electron in its outer orbit and has no exaggerated T1 or susceptibility effects.

### 5.1. Hyperacute

At the immediate hyperacute phase, (upon bleeding into the parenchyma) iron is still saturated with oxygen (diamagnetic) and cell membranes are intact. Hence, the hematoma produces slight hypointensity ('darker') or iso-intensity ('same') on T1 weighted images and iso- or slightly hyperintense ('brighter') on T2 weighted images. This makes it hard to distinguish a hematoma at the extreme initial stages; however, as hemoglobin gets deoxygenated rapidly toward the periphery of the lesion, it produces a T2 hypointensity at the periphery (a dark rim), which helps detection in the hyperacute phase.



## 5.2. Acute

Within hours of the bleeding event, hemoglobin is deoxygenated within intact cell membranes, from the periphery to the center of the lesion which is paramagnetic. This causes a Susceptibility effect, which is hypointensity ('darker') on T2. However, this structure of hemoglobin does not allow any effect on T1 images, which show a hard to distinguish iso-('same') or slight hypo ('darker') lesion. At times, there is a peripheral rim of T1 hyperintensity due to early oxidation of hemoglobin into met-hemoglobin.

## 5.3. Subacute

This phase begins after several days with the onset hemoglobin degradation. Due to the lack of energy in the cells, the iron is oxidized into the ferric state, which produces met-hemoglobin. This structure of iron atoms causes a decrease in the T1 relaxation times which is captured as a marked hyperintensity ('brighter') on T1 weighted images. Since the red cell membranes are intact, Susceptibility effect is in play causing a hypo ('darker') appearance on T2 weighted images.

Later on in the subacute phase (over days to weeks), the red cell membranes are degraded; hence the susceptibility effect is lost. This results in a T2 lengthening, which is seen as a hyperintensity ('brighter') on T2 weighted images.

## 5.4. Chronic

Over the course of weeks to months, the resolution process results in protein (met-hemoglobin) breakdown, which reduces the signal hyperintensity on both T1 and T2 weighted images. The iron atoms released in this process are picked up by macrophages and converted to ferritin for reuse elsewhere. However, the scavenging capacity of the macrophages is often overwhelmed especially in larger hematomas, which results in locally deposited hemosiderin molecules usually at the periphery. The structure of iron in hemosiderin exerts only a Susceptibility effect which is a hypointense ('darker') rim on T2 weighted images. The center of the hematoma may resolve into a cavity, usually filled with cerebrospinal fluid with the corresponding signal characteristics ('darker' on T1 weighted imaging and 'brighter' on T2 weighted imaging) or may collapse and be visualized as a narrow slit.

While the pathological processes usually follow a sequence with corresponding sequential imaging changes, these processes are also highly variable and dependent on a large number of factors such as size, presence of rebleed, oxygen tension, other concurrent conditions etc. Hence several stages of the hematoma may appear simultaneously on imaging which increases the complexity of determining the time of bleed.

MR is also a tool in neuroimaging to distinguish between a primary bleed and a hemorrhagic transformation, since area of the bleed is usually lesser than area of the infarct, and MR provides imaging of both. The shape (rounder) and larger edema around the bleed is another pointer toward primary ICH. Hematomas do not follow vascular territories but infarcts do and the occlusion is often visible on MR angiography.

MR is also one of the best diagnostic tools for secondary causes of hemorrhage, such as vascular anomalies, tumors, and cerebral venous thrombosis (comparable to conventional angiography) and is the choice of modality for cavernomas. It has a high diagnostic yield for etiologies especially in young nonhypertensive patients with lobar bleeds.

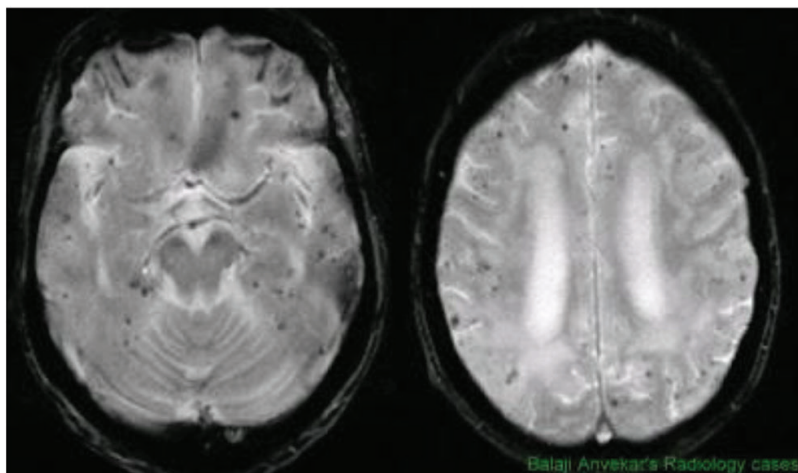
### 5.5. MRI-sequences in ICH

MR protocols in stroke include T1, T2, T2\* or GRE, fluid attenuated inversion recovery (FLAIR), contrast enhanced, diffusion weighted & perfusion weighted images, and MR angiography. Since the radiological appearance of the hematoma depends on both the hematoma and the MR signal characteristics, the latter can be varied to allow easier identification of hemorrhage. This is crucial as MRI shows minor and hard to appreciate changes in the hyper-acute and early-acute phases of ICH. By increasing the magnetic field, the susceptibility effect is increased, allowing easier and more rapid diagnosis. Sequences available commonly in clinical practice include fast spin echo (FSE), which due to a weaker magnetic field has less sensitivity to susceptibility effects (responsible for much of the lesion imaging) and is hence suboptimal initially in ICH detection. Using sequences such as gradient recalled echo (GRE) and echo planar imaging (EPI) increases the sensitivity to susceptibility effect.

### 5.6. GRE

Gradient recalled echo sequences (or T2\* weighted sequence) increases the hematoma detection in both acute and chronic stages. The strong Susceptibility effect results in extremely hypointense areas of hemorrhage on imaging (**Figure 3**).

T2\* GRE MRI sequence has high sensitivity in detecting cerebral microbleeds, which appear as small punctate (dot-like) hypointense lesions widespread in bilateral cerebral cortical white matter, basal ganglia, thalami, cerebellum as well as brain stem and are histologically characterized by hemosiderin deposits with tissue damage.



**Figure 3.** Gradient recalled echo. T2\* GRE MRI sequence has high sensitivity in detecting cerebral microbleeds, which appear as small punctate (dot-like) hypointense lesions widespread in bilateral cerebral cortical white matter, basal ganglia, thalami, cerebellum, as well as brain stem and are histologically characterized by hemosiderin deposits with tissue damage.

An advantage of GRE is that it can exclusively identify hemosiderin deposits from old and asymptomatic hemorrhages often referred to as microbleeds. A large number of microbleeds point to an etiology such as amyloid angiopathy or recurrent hypertensive vasculopathy. Since 80% of the hemosiderin deposits persist through a lifetime, it provides a snapshot of the hemorrhages across the patient's life span. These microbleeds are used as predictors of future ICH and a marker for small vessel disease especially in the basal ganglia region. The disadvantage in this sequence is occasionally the lesion size, which is inaccurate due to artifacts causing signal loss at the boundary of the lesions. Sinuses present in the skull enhance this signal loss and may not allow accurate identification of hemorrhagic lesions behind them [5].

## 6. Catheter angiogram in ICH

Certain clinical and radiological findings necessitate a conventional catheter angiogram, such as atypical configuration or location, excessive edema, evidence of masses or no obvious cause of bleeding; all of which necessitate pinpointing a secondary cause. The diagnostic yield of a conventional angiogram is high especially in younger patients with no hypertension. Often times, the vascular anomaly reveals itself over the course of time (upon resolution) and hence a follow up angiogram is recommended even after a prior workup reveals no abnormality. However, significant disadvantages of conventional angiography that include extremely high (5× more than CTA) radiation, cost, invasiveness, patient cooperation, and clinical stability as well as transient and permanent neurological deficits preclude its widespread use, giving preference to CT and MR angiography but remain the gold standard for aneurysms and arteriovenous malformations.

## 7. Detecting the etiology of ICH

An important step in the management of patients with ICH is determining the etiology and taking measures to correct and prevent further and future episodes of ICH. While medical history and demographics may help pinpoint a cause for the ICH, neuroimaging has a large role to play in this sphere.

Neuroimaging can provide a clue to etiology of the ICH based on the **location** and **imaging characteristics** of the hemorrhage.

Each **location or area** of the brain is associated with a list of common differentials as to possible etiologies. Lobar hemorrhage (bleeding mainly into the cortex through the sub-cortical junction) is mainly superficial and as the name states, deep ICH refers to bleeding mainly in the deeper structures such as thalamus, putamen, and head of the caudate. Lobar hemorrhages are usually not related to hypertension but are caused by cerebral amyloid angiopathy (including in patients with hypertension) and are present in the white matter of

the cerebrum and rarely in the cerebellum. Most deep or non-lobar ICHs are usually due to hypertension which is usually diagnosed based on the hemorrhage location. Hypertensive ruptures usually affect the smaller vessels such as lenticulostriate arteries, and perforating branches of the basilar artery, resulting in the characteristic sites of ICH. This lobar vs. deep structure based diagnosis does not hold true in patients less than 45 years of age where secondary causes such as vascular malformation, underlying tumor, vasoconstriction by sympathetic drugs are the usual culprits [7].

Hemorrhage in the brainstem (usually the pons) is usually associated with hypertension, vascular malformations (arteriovenous and cavernous).

Cerebellar hemorrhage is associated with hypertension, arteriovenous malformations, and the use of anticoagulants such as warfarin, with amyloid angiopathy being extremely rare.

Intraventricular hemorrhage (primarily intraventricular without involvement of the brain parenchyma) is associated with hypertension, aneurysm of the anterior communicating artery, vascular malformations, coagulopathy, and intraventricular tumors.

Hemorrhage at multiple sites is usually indicative of coagulation disorders, vasculitis, hypertension, tumors, and infarction.

Rupture of a saccular aneurysm may involve the parenchyma as well as the subarachnoid space, due to the pressure of the blood as it ruptures and its location (medial frontal lobe due to anterior cerebral or communicating artery aneurysm). Presence of ICH near the subarachnoid space near the base of the skull should prompt vascular imaging studies to rule out saccular aneurysms.

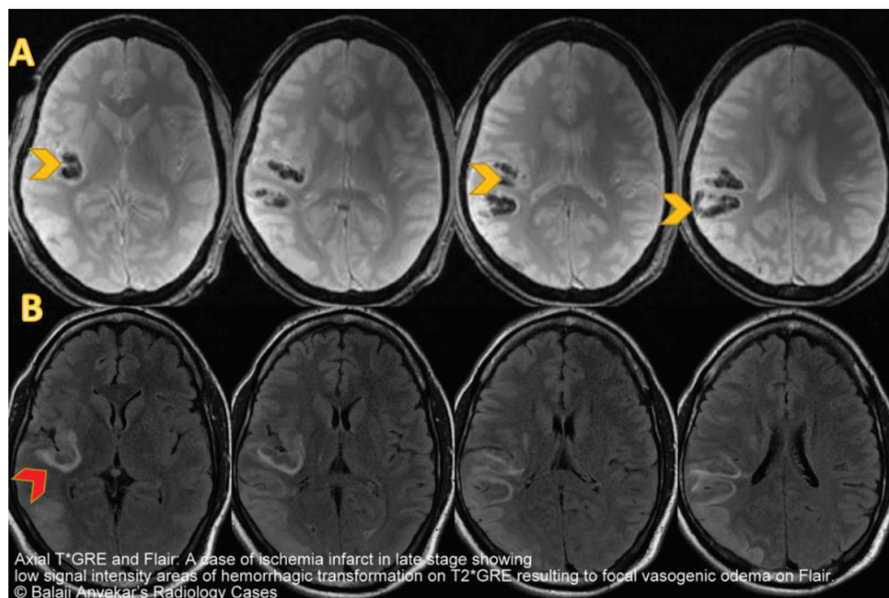
Common causes of ICH have distinctive **imaging characteristics** that point toward their diagnosis. The presence of multiple lobar microhemorrhages of differing ages, typically sparing the basal ganglia is a strong and specific indicator of cerebral amyloid angiopathy in the elderly which has been used in the clinical diagnostic criteria. The ICH in amyloid angiopathy often ruptures into the subarachnoid space but less commonly into the ventricles.

Arteriovenous malformations can often be suspected on conventional MRI and CT (T2 and MR and CT angiography) sequences by detecting dilated vessels to and from the malformation and at times patchy enhancement but are often times undetectable or silent. The presence of “popcorn” appearance of lesions on T2 weighted images suggests the presence of multiple small bleeds occurring at different time points in the same lesion such as a cavernous malformation. Presence of multiple micro- or larger bleeds on GRE sequence is also suggestive of a vascular anomaly as an etiology. The diagnosis of these malformations generally requires conventional angiography for confirmation.

Hemorrhagic transformation of an infarct is suggested by the surrounding cytotoxic edema which follows the arterial boundaries unless the hemorrhage is severe and early enough to blur the infarct visualization (**Figure 4**).



Hemorrhagic transformation of an ischemic infarct evident on MRI. A: T2 GRE showing areas of hypo-intensity (yellow arrowhead) corresponding to hemorrhage. B: FLAIR sequence showing the edema associated with bleed (red arrowhead).



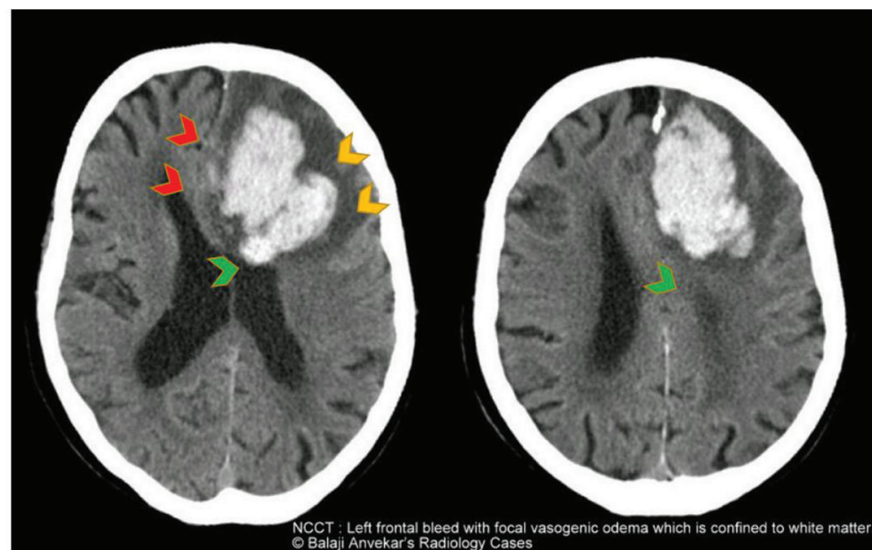
**Figure 4.** Hemorrhagic transformation. Hemorrhagic transformation of an ischemic infarct evident on MRI. A: T2 GRE showing areas of hypointensity (yellow arrowheads) corresponding to hemorrhage. B: FLAIR sequence showing the edema associated with bleed (red arrowhead).

Several neoplasms in the brain have a known propensity to bleed such as glioblastoma multiforme, and metastases of melanoma, lung cancer, renal cell cancer, etc. The imaging characteristics are variable due to the presence of often multiple hemorrhages at different time points and concurrent necrosis and cysts. Due to the low oxygen partial pressure in the tumor, MR signal changes are usually delayed. The location may be atypical for other common causes. The vasogenic edema present around a tumor is usually extensive and lasts longer as compared to primary ICH. Giving contrast almost always shows robust enhancement. A large hemorrhage may obscure the underlying lesion which may be visible on repeat imaging after its resolution [5].

## 8. Detecting complications of ICH

Complications of ICH include hematoma expansion, perilesional edema with increased intracranial pressure, and intraventricular extension of hemorrhage with hydrocephalus, seizures, venous thrombosis, hyperglycemia, autonomic fluctuations, and infections. Close monitoring is required for the prevention of these complications, and/or early detection and management, to reduce negative outcomes, the most emergent being mass effect resulting in herniation (**Figure 5**).

Left sided frontal lobe bleed showing perilesional edema (yellow arrowhead), mass effect (green arrowhead) and midline shift (green head)



**Figure 5.** Detecting complications. Left sided frontal lobe bleed showing perilesional edema (yellow arrowheads), mass effect (green arrowheads) and midline shift (green arrowheads).

Since vasospasm in SAH (and uncommonly in ICH with intraventricular extension) is a known risk factor from day 3 to day 12, transcranial Doppler is used prophylactically for its screening, with variable results as compared to conventional angiography [8]. CTA is also used for this purpose, with high sensitivity and specificity for severe spasms and in proximal vessels but reduced accuracy for distal vessels and mild spasms [9]. Irrespective of vasospasm, neuroimaging for ischemia can be performed using CT or MR perfusion studies or diffusion weighted MRI. CT perfusion studies show great prediction of vasospasm as compared to conventional angiogram [10] and studies exploring blood brain barrier permeability to guide future treatments using CTP are underway [11].

Size of the ventricles measured on CT and MR is variable and is not accurate to diagnose hydrocephalus, although serial changes in size on the same patient is more relevant toward detection. Periventricular edema seen with transependymal flow is a marker of hydrocephalus, better seen on MR than CT [12].

## 9. Sequences in practice

### 9.1. Prognostication

The initial volume of the hematoma assessed by various methods on neuroimaging, commonly the ABC/2 method, the presence of intraventricular blood, as well as the expansion of the hematoma indicated by the 'spot sign' are independent markers for clinical outcomes and mortality [2, 13, 14] (Table 3, Figure 6).



	Advantage	Disadvantages
<b>CT scan</b>		
Availability	Faster, cheaper, widely available.	
Contraindication	Can be performed in patients with contraindications to MRI.	Contrast allergy and impaired renal function (for contrast administration).
Side effects		High dose of radiation, contrast associated side effects: allergy, nephropathy and variable effects on blood brain barrier permeability and cytotoxic edema.
Imaging: detection of blood	Due to the differential attenuation of blood (proportional to the protein concentration in blood) vs. gray and white matter, blood in the parenchyma is detected immediately.	Since imaging is based on protein concentration; detection difficult anemic patients (hemoglobin <10 g/dl). Patients with higher hematocrit such as infants will show abnormal density in vessels.
Imaging: location		Bleeds in brainstem can be obscured by artifact. Flattened and thin blood collections (such as subarachnoid) are hard to visualize.
Imaging characteristics		Differential diagnosis of hemorrhagic tumor vs. infarct is difficult. Ring enhancement of the blood seen 1–6 weeks after the bleed is hard to differentiate from other ring enhancing lesions. Age estimation of hematoma is not as accurate.
Immediate detection of hemorrhage	Gold standard noncontrast CT.	
Detecting perfusion deficits	CT angiography and CT perfusion imaging can be used to detect ischemia, and vasospasm.	
Detecting hemorrhagic conversion of infarcts	Bleeding detected immediately.	
Detecting chronic micro-bleeds		Not ideal.
Detecting etiology	CTA performed less than 96 h from onset are highly sensitive and specific for vascular malformations.	
<b>MRI</b>		
Availability		Not as fast/cheap or widely available.
Contraindication		Several; including pacemakers, metallic implants and claustrophobia.
Side effects	No radiation.	Nephrogenic systemic fibrosis associated with gadolinium.
Imaging: detection of blood	MRI is sensitive to flow abnormalities in vessels and is ideal for detecting vascular malformations.	Immediate bleeding is isointense but due to rapid deoxygenation of hemoglobin at periphery, shows hypointense periphery on T2 and GRE.

	Advantage	Disadvantages
Imaging: location	Accurate at detecting exact location of hemorrhage.	
Imaging characteristics	Age estimation of hematoma is possible due to the differential magnetic properties of the different oxidation states of iron.	
Immediate detection of hemorrhage	MRI is sensitive if not more than CT in detecting acute ICH as per the HEME (Hemorrhage and Early MRI Evaluation) Study.	Gold standard remains noncontrast CT.
Detecting perfusion deficits	DWI/PWI mismatch.	
Detecting hemorrhagic conversion of infarcts	Ideal for distinguishing primary ICH vs. hemorrhagic conversion and shows the details of ischemic area.	
Detecting chronic micro-bleeds	More accurate than CT (GRE sequence). Lifetime history of hemorrhages is possible.	
Detecting etiology	Most sensitive and specific for detecting secondary causes such as vascular anomalies, venous thrombosis.	

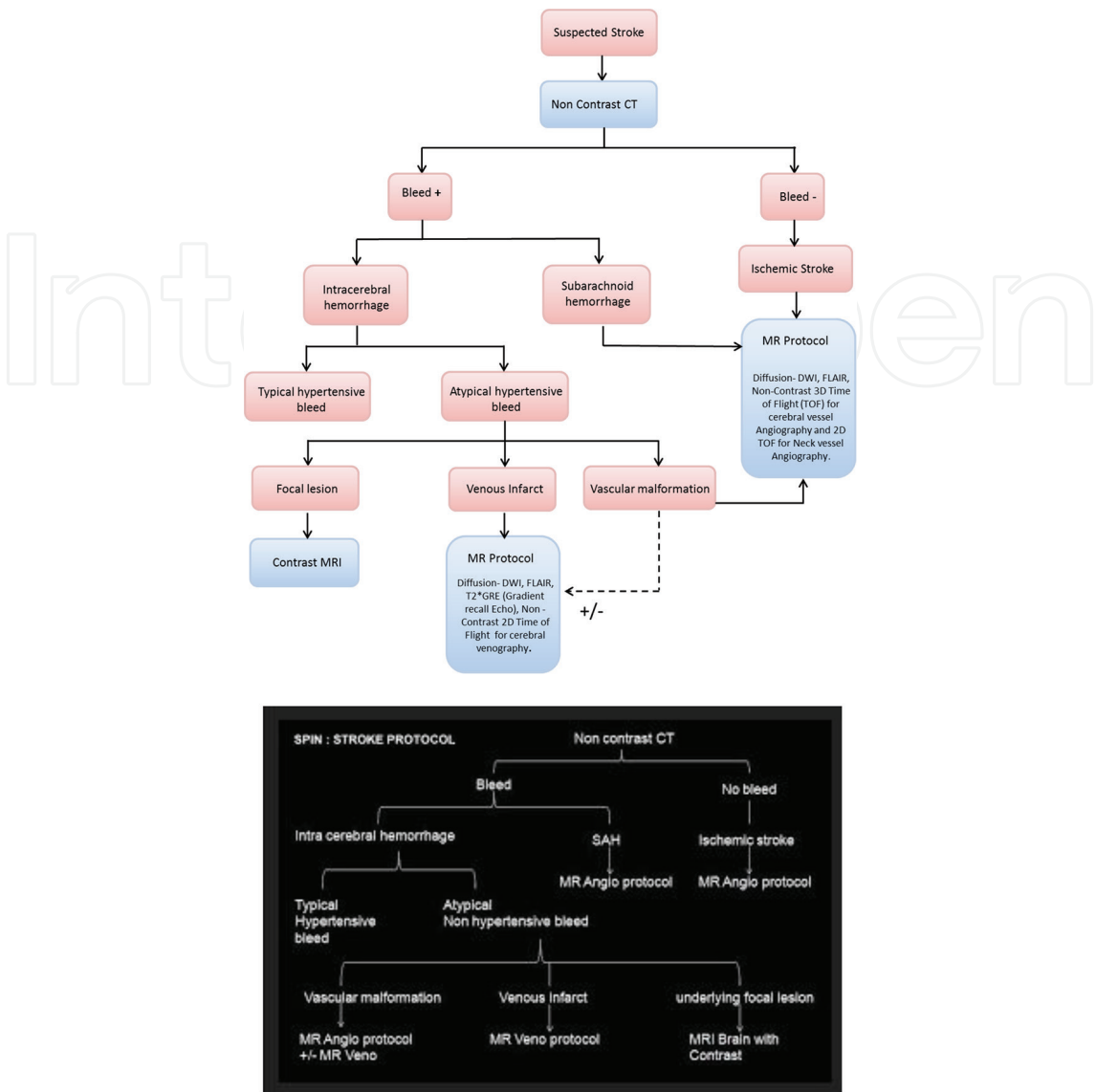
\*GRE = gradient recalled echo, CTA = computed tomography angiography, DWI = diffusion weighted imaging, PWI = perfusion weighted imaging.

**Table 3.** Advantages and disadvantages of CT and MRI in ICH imaging.

The risk of hemorrhage after ischemic lesions, notably after TPA administration, can be predicted by increased diffusion weighted imaging (DWI) lesion volumes, lower apparent diffusion coefficients, as well as decreased cerebral blood volume estimated using perfusion weighted imaging, the combination of the latter with DWI allowing one to identify infarcts with the colloquially termed ‘malignant profile’ for post thrombolytics bleeds. However, due to the time constraint of TPA administration which may not permit the above tools, CT perfusion imaging is being studied to predict similar hemorrhagic risk post thrombolysis based on the blood brain barrier permeability [15].

Using neuroimaging to correlate risk factors and measurable tissue states could allow greater precision in risk assessment. This could be utilized to predict the occurrence of ICH in patients of cerebral amyloid angiopathy (CAA) using PET and diffusion tensor imaging (DTI) [15].

While CT and MR provide the structural evidence of changes post ICH, aspects of brain function such as metabolism and absorption available through functional brain imaging may provide more granular details necessary to study the extent of injury and repair/recovery and may provide a new avenue to detect tissue at risk of hemorrhage.



**Figure 6.** Sequencing protocol. Adapted with permission from Neuroradiologycases.com by Dr. Anvekar B. (Neuroradiology unit, S P Institute of Neurosciences, Solapur, India).

DTI can be used to detect disturbances to the integrity and fiber counts of white matter tract, often damaged in ICH. Reduced fractional anisotropy or fiber counts may suggest worsened outcomes [15]. DTI can be used to predict motor recovery, as patients with preserved tracts on DTI at the time of lesion have shown greater recovery than their counterparts with compromised tracts [16] with similar studies being carried out using fractional anisotropy. A significant finding being that fractional anisotropy ratio did not correlate to size of the bleed but did correlate with recovery [17], which could serve as an important tool for the prediction of recovery post ICH. The predictive value of these studies has shown to be higher when carried out subacutely (2 weeks) rather than acutely (3 days) after the bleed which may be accounted for by the resolution of acute injury and inflammation and onset of repair and compensation [18].

Functional MRI (fMRI) is being evaluated as a means to evaluate the extent of injury and functional deficit post ICH based on functional connectivity rather than just a structural basis, with fMRI possibly evaluating the activity between physical and functional connections. Studies have pointed to the subcortical origin of redistribution of functional connection when cortical motor tracts are damaged [19]. This expands the possibilities to create a precise model to predict recovery post ICH.

## 10. Newer imaging techniques

Several newer imaging technologies such as CT and MR perfusion, positron emission tomography (PET), single photon emission computed tomography (SPECT) are used to study tissue injury such as perfusion deficits around the hematoma. Studies using PET have shown that this hypoperfusion does not result in hypoxia and ischemia, thus is not frankly ischemic in origin but likely to be due to secondary metabolic failure [20]. Diffusion tensor imaging (DTI), used to visualize white matter tracts is being used and studied for prognostication on motor recovery by assessing the integrity of major motor pathways such as the corticospinal tract [16, 21]. Newer technological advances in CT include dynamic angiography (4-dimensional CT angiography) which allows a detailed and comprehensive visualization of the intra and extra cranial vasculature and perfusion using a 320-row setup [22]. Magnetic induction tomography is being studied to measure tissue conductivity noninvasively, allowing identification of pathological changes and identification of extremely minute blood volumes [23]. Further exploration is underway to expand the mandate of neuroimaging, allowing image guided therapy at specific time points, using imaging biomarkers to assess edema, inflammation, and excitotoxicity.

## 11. Conclusion

Neuroimaging is a constantly evolving field to optimize the management, and prognostication after intracerebral hemorrhage and to advance research efforts. There are several choices available for neuroimaging in patients of ICH and familiarizing oneself with the techniques, indications, and disadvantages of each method allows the development of a rational imaging plan. Several advances have been made in the image sequencing protocols to optimize detecting, diagnosing, and selecting candidates for intervention and other therapies. Advances in this field such as diffusion tensor imaging and functional MRI are being studied for their impact in understanding the extent of injury and possible recovery mechanisms possibly allowing precise prognostication for patients. The mandate of neuroimaging is ever expanding with the ultimate goal of discovering tools that remain sensitive, specific, safe, rapid, and widely available, which allows optimized prognosis, prevention, and management for the best possible patient outcomes.

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