

# NEUROIMAGING 101

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## **Non-contrast Head CT in acute neurological change**

The most common initial neuroimaging study is a noncontrast head CT, which allows for the identification of acute hemorrhage. The high sensitivity and specificity of CT for identifying acute blood, along with its lower cost, feasibility in unstable patients, and wide availability make it the first neuroimaging modality of choice in most acute situations. In the setting of trauma, noncontrast head CT is usually obtained to identify surgically treatable lesions such as acute subdural or epidural hematoma as well as skull fractures. In the absence of trauma, rapid neuroimaging using CT is recommended to distinguish ischemic stroke from an intracerebral hemorrhage (ICH). Acute ICH is seen as a hyperdense lesion on head CT. However, as time passes the lesion will become isodense with the brain parenchyma, typically after one week, at which time the sensitivity of CT is lower than that of MRI. In addition to determining the location of hemorrhage, CT imaging also helps identify intraventricular extension, assess the extent of cerebral edema and mass effect, and estimated hemorrhage volume. Once an intracranial hemorrhage has been identified, the pattern of blood, the patient's medical history, neurologic examination, and laboratory studies lead the practitioner to pursue further neuroimaging studies to guide the medical, surgical, and interventional management.

Given the fact that hemorrhagic stroke comprises only 15-20% of all strokes, it goes without saying that many patients with acute neurologic dysfunction will have a noncontrast head CT which is unrevealing of the source of the patient's dysfunction. One such common scenario, is in the setting of acute ischemic stroke, where the purpose of the noncontrast head CT is to evaluate patients for the proper administration of tPA. Acute ischemic lesions cannot be identified on a noncontrast head CT within the first few of hours of symptom onset. Therefore, the patient's noncontrast head CT may appear normal in the first few hours of an acute ischemic stroke, and support the clinical decision to give tPA. When evaluating for acute ischemic stroke, the provider should use the physical exam and history to localize the ischemic stroke and then focus his/her attention on that portion of the head CT. For example, if a right-handed patient presents with aphasia, right hemiparesis, and right hemianopsia the provider should look for early findings which would support a left MCA ischemic infarction. Such findings would include flattening of the insular ribbon on the left, blurring of the gray-white junction on the left, and left hemispheric sulcal effacement. These subtle findings may initially be missed by the interpreting physician. However, the provider who is caring for the patient has the luxury of the physical exam and patient history to empower them to identify subtle abnormalities on head CT assisting them with their clinical decision making.

## **Noncontrast head CT in subacute neurological change**

The evaluation of the patient with subacute or progressive neurological change in the absence of focal neurological deficits typically begins with a noncontrast head CT. The lack of focality on the patient's neurological exam may be explained by bilateral lesions. There are some bilateral lesions which are easily identified on noncontrast head CT such as bilateral chronic subdural hematomas. However, the head CT may not be as revealing in patients with bilateral subtle gray-white junction abnormalities such as brain metastases, lymphoma, infectious processes, or demyelination, which may be due to the limitations of head CT.

## **Limitations of noncontrast head CT**

Head CT imaging has several limitations. It poorly visualizes the posterior fossa contents and (i.e., the brainstem and cerebellum) because of beam hardening artifact. In addition the hyper attenuation of acute blood on head CT is based on the protein content of whole blood in (i.e., hemoglobin). Therefore in patients who are anemic, in particular those with serum hemoglobin less than 10 g/dL, hyper attenuation may be limited, resulting in a reduced ability to identify the ICH. Similarly in patients with very elevated hemoglobin values, such as those with polycythemia or significant hemoconcentration, the vasculature may appear abnormally hyperdense, making acute diagnosis of ICH more challenging. When CT imaging is unrevealing, MRI imaging is usually the next step.

## **Brain MRI**

Unlike CT imaging, which uses x-rays to produce cross-sectional images and hence involves radiation exposure, MRI uses magnetic field and radio waves to produce images. Modern 1.5T MRI machines are equally sensitive at identifying acute symptomatic ICH as CT. In addition, MRI is more sensitive than CT at identifying acute ischemic change, subacute ICH, brain tumors, white matter lesions, and lesions of the posterior fossa and cerebellum. Although MRI imaging sequences can be tailored to the diagnostic question, there are several common sequences that are usually obtained on most brain MRI studies which are described below.

## **Limitations of brain MRI**

Limitations associated with MRI, other than cost, center on logistic and patient's specific characteristics. The most common logistic limitations associated with MRI is the ability to obtain the study in a timely fashion. Common patient's specific limitations of MRI included the presence of a pacemaker or ferromagnetic foreign object, claustrophobia, and large body habitus. Claustrophobia and large body habitus have, to some degree, been overcome by the advent of open and semi-open MRI scanners, but these may not be available at many centers. The duration of the study also poses some patients specific limitations in patients with more extensive illness who may develop hemodynamic instability. The patient must also be able to lie flat and still for the duration of the study without respiratory compromise, as motion significantly degrades the diagnostic information gained from an MRI.

## **Common brain MRI sequences**

T1- Has is similar in appearance to head CT wherein CSF is hypointense, gray matter is intermediate signal intensity, and white matter is hyperintense. However, the appearance of hemorrhage changes over time. Acute hemorrhage begins as isointense, progressing to hyperintense, and later to hypointense.

T2-Fluid is emphasized and thus CSF appears hyperintense as does edema as seen with acute ischemic stroke (cytotoxic) and neoplasms (vasogenic). The appearance of hemorrhage changes over time with acute hemorrhage starting out as hypointense then evolving to hyperintense, and later hypointense.

DWI-Diffusion weighted imaging allows for demonstration of restricted diffusion of water. When diffusion is restricted within a particular part of the brain that portion will be hyperintense. Such is the case in acute ischemic stroke wherein the hyperintensity of the infarcted tissue will remain visible for approximately 2-3 weeks. Hyperintensities on DWI imaging are seen in any condition in which there is breakdown of the blood brain barrier such as demyelination, diffuse axonal injury, or acute infection. Owing to the fact that some portions of the sequence are derived from T2, normal structures that are hyperintense on T2 will also be hyperintense on DWI in the absence of restricted diffusion, which is termed T2 shine through.

ADC-(Apparent Diffusion Coefficient) provides complementary information in assessing for restricted diffusion of water. Acute pathology such as acute ischemic stroke will be represented as hypointense in the same region that hyperintensity was noted on DWI.

GRE-Gradient recall echo allows for detection of small amounts of hemorrhage, which will appear hypointense on T2\* (two dimensional GRE) or SWI (susceptibility weighted imaging, three dimensional GRE).

FLAIR (Fluid Attenuated Inversion Recovery) is best for identifying cerebral edema. By suppressing the hyperintense CSF signal, parenchymal cerebral edema is easier to identify. This sequence allows for identification of lesions in the periventricular white matter as well as those in the periphery of the cortex.

## **Contrast enhancement**

CT- Iodinated contrast is the contrast media used in CT based imaging studies. Iodinated contrast has its greatest utility in CT based imaging when imaging the vascular system such as CT angiography. A contrasted head CT is of limited utility and is often only considered when the patient cannot obtain an MRI. Iodinated contrast places the patient at risk for acute kidney injury especially when they have an elevated serum creatinine. It is dialyzable and thus can be administered to patients who are on dialysis.

MRI-gadolinium is the contrast media used in MRI based imaging studies. In order to minimize the risk for nephrogenic systemic fibrosis, the use of gadolinium is limited to patients with a glomerular filtration rate greater than 30 and those who are not on dialysis as it is not dialyzable for the most part.

### **Common clinical scenarios**

Acute ischemic stroke- noncontrast head CT may be normal in the first few hours of symptom onset. This will allow for evaluation for tPA administration. Subsequent neuroimaging with brain MRI can better delineate the area or areas of infarction and provide insight into the etiology of the patient's acute ischemic stroke.

Acute head trauma- noncontrast head CT to identify skull fractures as well as intracranial hemorrhage for surgical evaluation. Brain MRI can identify areas of diffuse axonal injury which may explain a patient's poor neurological exam in the setting of an unrevealing head CT.

Acute hemorrhagic stroke- noncontrast head CT can identify acute hemorrhage and guide subsequent medical decision making. Brain MRI can identify chronic or subacute hemorrhages and microhemorrhages which are not easily seen on head CT.

Demyelinating disorders- noncontrast head CT is of limited value especially when the area of demyelination occurs in the brainstem or posterior fossa. Brain MRI especially with gadolinium enhancement can identify demyelinating lesions and distinguish between acute lesions which would enhance with gadolinium and chronic non-enhancing lesions.

Neoplasms- noncontrast head CT may reveal an area of cerebral edema suggesting an underlying structural lesion or may be unremarkable. Brain MRI especially with gadolinium enhancement can identify neoplasms as well as secondary satellite lesions seen in metastatic disease.

### **References**

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