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How to Read a Head CT

Recently published research suggests a concerning rate of head computed tomography (CT) scan misinterpretation by emergency physicians. This session will help emergency physicians improve their ability to read cranial CT scans. Expert faculty will explain the physics of CT scanning and review normal anatomy. CT scans of pathologic conditions frequently missed by emergency physicians will be presented. These cases will include fractures, hemorrhage, infarcts, edema, hygromas, and shear injuries. Methods to avoid errors of interpretation will be discussed.

- Briefly discuss the physics of CT scanning, including CT numbers, windows, and volume.
- Describe the CT appearance of normal brain anatomy.
- List the pathologic conditions most frequently misinterpreted by emergency physicians and the specific errors that resulted in the incorrect interpretation.

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FACULTY

Douglas L McGee, DO

Associate Director, Emergency Medicine Residency Program, Albert Einstein Medical Center; Assistant Professor, Temple University School of Medicine and Philadelphia College of Osteopathic Medicine, Philadelphia, Pennsylvania

How to Read a Head CT

Douglas McGee, D.O.

I. Course Description

Recently published research suggests a concerning rate of head CT misinterpretation by emergency physicians. This session will help emergency physicians improve their ability to read cranial CT scans. The physics of CT scanning will be explained and normal anatomy will be reviewed. CT scans of pathologic conditions frequently missed by emergency physicians will be presented. These will include fractures, hemorrhage, infarcts, edema, hygromas and shear injuries. Methods to avoid errors of interpretation will be discussed.

II. Course objectives

Upon completion of this course, participants will be able to:

1. Briefly discuss the physics of CT scanning, including CT numbers, windows and volume averaging

2. Describe the CT appearance of normal brain anatomy

3. Identify common pathologic conditions seen on CT scan encountered in the Emergency Department

4. List the pathologic conditions most frequently misinterpreted by emergency physicians and the specific errors made which resulted in the incorrect interpretation.

III. Course outline

Introduction

Godfrey N. Hounsfield is credited with the invention of computed tomography in 1972 but the mathematical model that allowed reconstruction of images based on their points in space was know by Radon as early as 1917. Conventional radiographs image all types of tissue in a similar manner, treating these tissues as if they had uniform radiographic density. These tissues are, of course, different in their chemical composition and structure. Computer enhanced images that define these differences and allow manipulation of the contrast and magnification form the basis of modern computed tomography.

A. Comparing various radiologic techniques

Conventional radiographs

Conventional radiographs rely on a summation of tissue densities penetrated by X-rays that are recorded on a monitor or film. Two-dimensional images created by three-dimensional objects (such as the heart) demonstrate poor contrast between tissues of varying density. Because the densest object attenuates, or absorbs the most x-rays, low contrast objects are often lost. X-ray beam scattering causes blurring of the x-ray image, further compounding the difficulty in visualizing low contrast objects. Blur can be reduced by directing the beam through a collimator, reducing the beam to narrow rays. Finally, the recorded image cannot be manipulated by computer to adjust image contrast and enhance areas of interest.

Classic tomography

Classic, or conventional tomography, attempts to minimize the difficulty of superimposing three-dimensional information on to two-dimensional film. The x-ray source is moved in concert with the recording device to blur structures which are not of interest. The object being studied remains stationary while the film and source are rotated around a point in the patient. This point is known as the focal point or fulcrum and is the clearest object on the film. This technology has limitations that result in less than optimal images. Adjacent tissue is often not completely blurred and is, therefore, never completely obscured. Blurred tissue, although difficult to discern, contributes to the overall "cloudiness" of the final x-ray image.

Computed tomography

A series of pencil thin x-ray beams are passed through the patient and are detected 180^{o} from the beam source. Moving the beam source 360^{o} around the patient and collecting information on corresponding detectors scans the patient. CT has largely overcome the difficulties with conventional radiography and classic tomography. This is possible because of several factors. First, small volumes of interest are scanned minimizing the degree of superimposition. Second, very narrow x-ray beams minimize the degree of scatter and blurring. Finally, computer manipulation of the information detected allows the data to be re-oriented to emphasize particular areas of interest.

Pixels
Each scan volume has a thickness. Each scan slice is further divided into smaller elements with areas described by *x* and *y*. These scanned volumes and the corresponding scanned areas (*x* by *y*) are referred to as pixels (*pic*ture *el*ement).

Attenuation coefficient
The tissue contained within each pixel absorbs and removes x-rays from the x-ray beam. This is referred to as attenuation; the amount of attenuation is assigned a number known as the attenuation coefficient. These coefficients can be mapped to an arbitrary scale where water is assigned a value of 0, bone a value of +1000 and air a value of -1000. This scale is known as the *Hounsfield scale* in honor of Godfrey Hounsfield. These Hounsfield numbers, or *CT numbers*, define the characteristics of the tissue contained within each pixel. Manipulation of these numbers on the contrast scale within the video monitor displaying the image allows the clinician to manipulate the image to highlight features of interest.

Windowing
One of the biggest advantages of CT scanning over conventional radiography is the ability to *window* certain tissues. Tissues of interest can be assigned the full range of blacks and whites available to the viewing monitor. This process allows the full gray scale to be assigned to a narrow range of CT numbers to maximize the differences in tissue appearance.

B. Normal brain anatomy seen on cranial CT

Like orthopedic injuries and plain radiographs in the Emergency Department, functional knowledge of relevant anatomy is compulsory when the Emergency Physician is interpreting CT scans. Specific neuro-anatomic structures or regions of the brain must be identified to correctly interpret associated pathology. Identifying injured or diseased structures and their corresponding neurologic function is useful when correlating CT findings with physical examination findings. In addition, the Emergency Physician must be able to effectively communicate with the consultant who may become involved with a particular patient's care.

Although detailed, intimate and subtle knowledge of brain anatomy may be desired, it is not required to identify the most important structures. Accurate identification of the following structures should allow for sufficient interpretation of most ED CT scans.

cranial bones: frontal, temporal, parietal, occipital
sinuses: frontal, ethmoid, sphenoid, maxillary, mastoid air cells
brain: cortex, cerebellum, ventricular system, basal ganglia, thalamus
subarachnoid space
vascular structures

Changes in the neuro-anatomic architecture occur with aging. As people get older, there is a loss in brain volume and functioning neurons. This is manifested on the CT scan as widening of the sulci and dilatation of the ventricles. Brain atrophy may occur in the gray matter, the white matter or both and may be generalized or focal depending on the etiology. The term **central atrophy** is often used to describe enlargement of the ventricles out of proportion to the sulci and is often associated with white matter disease. **Cortical atrophy** refers to widening of the sulci without ventricular dilatation and often represents "normal aging" of the brain.

C. Pathologic conditions seen on CT scans in ED patients

Skull fracture

Linear

Fractures of the calvarium are lucent relative to surrounding bone and are typically more lucent than vascular grooves or sutures which have not closed.

Linear fractures are wider at the midportion and narrower at the end of the fracture; usually no wider than 3 mm.

Most common in the temporoparietal, frontal or occipital bones and tend to extend toward the base of the skull.

Fractures may take 3-6 months in infants and 2-3 years in adults to heal.

Depressed

Degree of depression and interruption of the inner table of the skull can be easily identified on CT scan

May be associated with intracranial hematoma or underlying parenchymal injuries which must be searched for on the CT scan using the brain tissue windows

Skull base

Fractures at the base of the skull are hard to visualize on standard CT scans. High resolution, thin slice CT scans may demonstrate basilar skull fractures

Intracranial air and air-fluid levels, representing blood or CSF, seen in the basilar sinuses may provide indirect evidence of basilar skull fracture

Traumatic suture diastasis (traumatic separation)
Complete union of the coronal suture does not occur until age 30; union of the lambdoidal suture occurs near age 60; lambdoidal diastasis is most common

Occur when fractures extend into the suture; and should be suspected when the suture width is greater than 3 mm

Traumatic hemorrhage
Fresh blood within and around the brain appears as a bright white lesion usually measuring between 50 and 100 Hounsfield units. This brightness is due to the relative density of the globin molecule that effectively absorbs x-ray beams.

As blood begins break down, characteristic changes are seen on the CT scan. In the first few hours after hemorrhage, clot retraction causes a slight increase in radiographic density.

As the globin molecule breaks down, the blood appears to lose its density. Clot density decreases from the periphery and progresses centrally. A 2.5 cm clot becomes isodense in about 25 days. The clot is present but is no longer seen on the CT scan. As macrophage activity removes the remainder of the blood products, a cavity will be seen in the area of the hematoma.

Acute subdural hematoma
Seen on CT as a high density collection between the brain and the inner table of the skull; shaped like a crescent

Typically extend from front to back around the cerebral hemisphere and may enter the interhemispheric fissure or dissect under the temporal or occipital lobes to the base of the cranial vault

Loss of the sulci and narrowing of the ventricles often occurs and is due in part to clot volume; significant edema may be due to associated brain injury

CT scan may be limited in identifying certain types of subdural hematomas:

> thin hematoma adjacent to bone may be difficult to see because of x-ray beam distortion (known as beam hardening)

brain windows may not distinguish between the bone and the hematoma and require manipulation of the CT windows to make this distinction

small subdural hematomas over the convexity of the brain may be difficult to see because of signal averaging that occurs with adjacent bone mass

Chronic subdural hematoma
Chronic subdural hematomas are thought to be due to slow venous oozing between the brain and the dura; a fragile, vascular membrane often encases the collection and is subject to re-bleeding

CT appearance of a chronic subdural hematoma depends on the length of time since the last bleeding episode; old hematoma appears less dense than brain but because of the high protein content, has a higher signal than CSF

Re-bleeding may occur at any time and may be confined to loculated areas within the chronic collection. Fresh blood often settles to the most dependent portion of the hematoma; chronic subdural hematomas may be hypodense, hyperdense, isodense or mixed

Bilateral chronic subdural collections may be difficult to see if they are isodense; ventricles smaller than expected for age or white matter which appears too far from the calvarium may signal the presence of these hematomas

Contrast may highlight the vascular membrane surrounding a chronic collection

Epidural hematoma
Epidural hematomas are biconvex (lenticular or lens shaped) but vary in appearance because of several factors: source of bleeding (arterial or venous) and the length of time between the injury and the CT scan

Most epidural hematomas are caused by arterial bleeding and are represented by a hyperdense lesion that may cause effacement of the sulci, ventricular narrowing and midline shift. If brain injury is also present, edema and intraparenchymal hemorrhage may accompany an epidural hematoma

Because the dura is bound tightly at the suture margins, epidural hematomas do not cross sutures, bilateral epidural hematomas are exceedingly rare

Traumatic intraparenchymal hematoma
Intracerebral hematomas due to trauma are typically visible immediately following injury. They are hyperdense and can be associated with surrounding edema; often occurring at the white and grey matter interface

Usually found in the frontal and temporal regions; often associated with other injuries seen on the CT scan; may rupture into the intraventricular space

Subacute hematomas may become isodense overtime

Cerebral contusion
Contusions on the surface of the brain may be coup or contrecoup; coup lesions occur most frequently in the frontal and temporal regions

Superficial hemorrhagic contusions may be difficult to visualize on CT scan because of beam hardening artifact and signal averaging with adjacent bone

MRI is better suited for identifying these types of injuries

Diffuse axonal injury (DAI or shear injury)
Occurs when the brain is subjected to translational, torsional or rotational forces that stress the white matter axons

CT scan is often unremarkable; small focal hemorrhages may be seen with surrounding edema; typically occur at one of 4 sites: corpus callosum, corticomedullary junctures, upper brainstem and the basal ganglia

MRI is superior for evaluating DAI

Non-traumatic hemorrhage
Intraparenchymal hemorrhage
CT reliably identifies intracerebral hematomas as small as 5 mm and are identified as hyperdense lesions; may extend to the brain surface and cause a secondary subdural hematoma

Hematoma due to hypertensive disease are seen in older patients and typically occur at the basal ganglia and internal capsule but may be found in the thalamus, cerebellum or brainstem; typically dissects away from its site of origin along the white matter tracts

Hemorrhage may rupture into the intraventricular space allowing the blood to access any portion of the ventricular system including the subarachnoid space

Cerebellar hematomas may dissect into the pons, cerebellar peduncles or directly into the fourth ventricle

Intracerebral hemorrhages due to hypertensive disease are usually homogeneous. Heterogeneous hematomas should raise the suspicion of associated tumor, infarction or injury; this heterogeneity may be due to the presence of edema, abnormal or necrotic tissue

Subarachnoid hemorrhage
75% of patients with SAH have an aneurysm that may be seen on non-contrast CT studies if it is large enough, 5% have an A-V malformation and 15% have no cause identified

Blood on CT following SAH is hyperdense and can be detected with accuracy in 80-90% of SAH; very small hemorrhage or those which are several days old may not be seen; false negative CT scans are not uncommon in patients with high neurologic grades following SAH

The location of an aneurysm responsible for the SAH may cause a characteristic distribution of extravasated blood:

> **anterior communicating artery aneurysm:** blood and around the interhemispheric fissure, suprasellar cistern, cingulate and callosal gyri, the brainstem and the sylvian fissure
> **internal carotid/posterior communicating artery aneurysm:** tend to bleed into the Sylvian fissure and the suprasellar cistern
> **middle cerebral artery aneurysm:** bleed into the adjacent Sylvian fissure and the suprasellar cistern
> **posterior inferior cerebellar artery:** bleed in and around the brainstem but the scan is often falsely negative unless the bleeding is massive

The clinician must distinguish a normally calcified falx from recent bleeding; the posterior falx is seen in 88% of normal patients, the anterior falx is seen in 38% of normal patients; blood is typically seen in the paramedian sulci

SAH may be accompanied by a hematoma, often near the site of the aneurysm

Significant cerebral vasospasm may be present after SAH and will occasionally be manifest by cerebral edema or infarction; patients with severe vasospasm and clinical deterioration may have a normal CT scan

Mass lesions
Tumor
When interpreting a CT scan for tumor, 4 things should be determined: tumor

location and size, tumor appearance or character, degree of edema/mass effect and the presence of herniation

Tumors may appear as poorly defined or well-defined hypodense lesions on the non-contrast CT scan. Some brain masses may appear as hyperdense lesions and may vary in appearance even among tumors of similar tissue type. Calcification and hemorrhage may occur within a tumor mass.

Tumors may be suspected on a non-contrast CT scan when edema accompanying the mass is seen. Vasogenic edema occurs when the integrity of the blood brain barrier is lost and fluid passes into the extracellular space; increased intercellular water creates a low-density signal on the CT scan. There is more extracellular space in white matter than gray matter; edema occurs more readily in white matter. Cerebral edema is not confined to tumor. Edema results from tumor, hemorrhage, loss of the blood brain barrier or cellular edema often due to hypoxia.

Intravenous iodinated contrast may enhance the appearance of brain tumors with a high contrast ring because the contrast media is leaked through an incompetent blood brain barrier and concentrated in the extravascular space surrounding the tumor; contrast should be used when a tumor is suspected; MRI is superior in detecting the presence of a tumor

Intracranial masses can be grouped into 3 broad categories: primary neoplasms, secondary neoplasms, and cysts or tumor like masses. Generally, primary tumors of the brain are found in a 2:1 ratio compared to metastatic tumors

The location of brain tumors can be classified as intraaxial (such as an astrocytoma of glial origin) or extraaxial (such as a meningioma); most extraaxial tumors exhibit early and intense contrast enhancement

Although the tumor's location and particular appearance may allow a more specific diagnosis, this information is useful to the radiologist and neurosurgeon but rarely important to the Emergency Physician who has achieved the primary goal: identifying the presence of an intracranial mass.

Herniation
Although the brain can accommodate an expanding mass to a certain degree, the fixed dura compartmentalizes the brain and forces herniation of brain contents from one compartment into another in typical patterns:

<u>subfalcine</u>: the cingulate gyrus is pushed under the falx

<u>uncal</u>: a transtentorial herniation; the uncus of the temporal lobe is displaced medially and over the edge of the tentorium

<u>central:</u> a descending herniation of the hippocampus bilaterally through the incisura

<u>cerebellar tonsillar</u>: inferior displacement of the medial portions of the cerebellar tonsil through the foreman magnum and behind the cervical spinal cord, and compressing the medulla

Abscess/cerebritis

Abscess formation in the brain is at the terminal end of the spectrum beginning with cerebritis; as focal cerebritis progresses and the cerebral tissues soften, liquefaction and necrosis may occur

The brain attempts to wall off an abscess and forms a fibrous capsule around the collection

Cerebritis

Initially seen as focal areas of vague hypodensity on CT which is surrounded by regional or global mass effect due to edema; may enhance with contrast
As cerebritis evolves, small focal hemorrhages may give the area an indistinct heterogeneous appearance

Because of its greater sensitivity to changes in water content, MRI is more sensitive in the evaluation of early cerebritis

Abscess

An ill-defined hypodensity is seen on non-contrast CT scan; a faint ring of hyperdensity may surround this hypodense structure

Mass effect with compression of the ventricular system is seen in 80% of abscesses

The fibrous capsule is easily enhanced with contrast; a smooth appearance to the inner side of the ring is highly suggestive of abscess formation; the capsule is thin (3 to 6 mm) and is usually uniform in thickness

Most abscesses are supratentorial; cerebellar abscess account for no more than 18% of all abscesses

Ischemic infarction

Thrombotic infarction

CT may demonstrate ischemic infarction as soon as 3 hours after the injury but are typically not seen on CT for up to 24 hours; ischemic brain tissue appears hypodense on CT scan with loss of the grey-white matter interface

Ischemic infarctions become more distinct as time progresses and assume a wedge shaped lesion with its base pointed toward the base of the brain

Brain edema may be demonstrated as soon as 24 hours after the insult; mass effect may be seen in up to 70% of infarctions and is maximal between 3-5 days

Contrast may be used in the early stages of ischemic strokes to demonstrate lack of normal perfusion in the area of ischemia; this may identify ischemic brain tissue when the non-contrast CT is normal

Embolic infarction
CT scans immediately following an embolic stroke appear similar to thrombotic strokes. Recannulation and lysis of the embolus results in restoration of cerebral blood flow into injured brain tissue

Injured brain tissue loses its ability to autoregulate resulting in hyperemia or increased blood flow; previously hypodense lesions may now become isodense or hyperdense

Marked brain edema may result after embolic stroke and is maximal between the 2nd and the 5th days

Hemorrhagic infarction
Hemorrhagic infarctions often follow embolic strokes and are most likely when brain necrosis is present

Characteristic hyperdense lesions are seen on CT scan; unlike cerebral hematoma, hemorrhage following embolic strokes appears on CT to be indistinct and heterogeneous

Hemodynamic infarction
Occur after a period of decreased cerebral perfusion in patients with fixed vascular disease; ischemia is often best seen in the watershed areas or "border zones" of the brain (junctions of the anterior, middle or posterior cerebral artery circulations)

Lacunar infarctions
Small infarctions seen in patients with arteriosclerotic disease and hypertension in the basal ganglia-internal capsule area

Lacunar infarctions assume a conical or cylindrical configuration, extend through the basal ganglia or internal capsule, and terminate in the periventricular white matter

Reading a CT scan

Like reading an electrocardiogram or chest radiograph, a uniform, consistent approach to the emergency head CT interpretation is useful. Perron, et al have suggested the following mnemonic:

Blood Can Be Very Bad

Blood: Acute blood is bright white on the CT scan. Types include: EDH, SDH, intraparenchymal, intraventricular, SAH

Cisterns (Can): CSF collections jacketing the brain. Look for blood and effacement. Four key cisterns: circummesencephalic (ring around the midbrain), suprasellar (star shaped) Circle of Willis, quadrigeminal (W shaped), Sylvian (between the temporal and frontal lobes)

Brain (Be): Look for: symmetry, gray-white matter differentiation, shift, hyper/hypodensity, pneumocephalus

Ventricles (Very): CSF produced in the lateral ventricles (back to back commas) → III ventricle (slit shaped) → aqueduct of Sylvius → IV ventricle (helmet shaped). Look for: effacement, shift, blood

Bone (Bad): Note soft tissue swelling. Look for blood in the sinus/mastoid air cells, widened sutures.

Summary

Cranial computed tomography has altered that way Emergency Physicians manage and evaluate patients with head injuries, neurologic abnormalities or alterations in mental status. Increasingly, Emergency Physicians are called upon to interpret the studies they order. Accurate interpretation of these scans is of paramount importance. The following summarizes essential CT findings for various abnormalities seen on CT scans in the Emergency Department.

Skull fractures

seen best with "bone windows", more lucent than surrounding bone
less lucent than sutures or vascular markings
fractures at the skull base are hard to visualize
traumatic diastasis may occur with fractures

Hemorrhage

hyperdense, bright white lesions on CT
subdural = crescent shape; epidural = lens shaped
hemorrhage becomes less dense over time
cerebral contusions and diffuse axonal injuries best seen on MRI

subarachnoid hemorrhages seen about 80% of the time

Tumor

hypodense edema often the only finding on non-contrast CT scan
enhances with intravenous contrast
may be associated with herniation which follows predictable patterns

Abscess

hypodense lesions and a faint hyperdense ring seen on non-contrast CT scan
fibrous capsule enhances on contrast CT scan
most are supratentorial

Ischemia

appears hypodense on CT scan, contrast may show lack of perfusion
many types: thrombotic, embolic, hemorrhagic, hemodynamic, lacunar
CT typically normal for up to 24 hours

Additional Readings

The following are a partial list of references used in this presentation and additional readings on the general subject of CT scan interpretation in the ED.

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