





Slides

xplanation

Notes

Additions

Important

Objectives





- The inner Dura the (meningeal) is attached firmly to the outer one (periosteal) there is no space in between, so consider them one layer. The outer layer follows the bone and the inner layer reflects in the interhemispheric fissure or to the occipital lobe and cerebellum to make the Dural reflections (falx cerebri & falx cerebelli), So anything or any disease that is beneath the Dura should be called **<u>subdural</u>**, anything between the Dura and the bone is called **<u>epidural</u>**, Then after Dura is the white layer called **<u>the arachnoid</u>** then the **<u>subarachnoid space</u>** (which contains the CSF). the layer directly above the brain is the **<u>Pia matter</u>**.



CT: bone is white.

MRI: bone is black. Blood in CT: 1- Hyper-acute "within minutes" (black) 2-Acute: hyper-dens (white) 3-Sub-acute "after 2-3 days" isodense (gray) 4-Chronic: hypo-dense (black).

1)Intracranial Bleeding:

Types:

- I. Extradural(epidural)
- II. Subdural
- III. Subarachnoid.
- IV. Intraventricular.
- V. Intraparanchymal.



2- It vice versa with <u>subdural hematoma</u> when it reaches a suture it will cross it and spread because it's far from the bone. It will follow the Dural reflections and enter the falx and fissures. More dangerous because of attachment to underlying tissues.





1.1:Epidural Hemorrhage (EDH)

- □ Blood collection between inner table(Skull) and dura.
- Biconvex shape (lentiform).
- **Occur at site of impact(Site of the trauma).**
- **95% unilateral**, supratentorial.
- Does not cross sutures.
- Can cross falx and tentorium, doesn't cross sutures but crosses flax because it's not deep into the Dura.
- Skull fracture in 90%.
- Air seen in 20%.
- Cause of injury: Arterial 90%, Venous 10%
- □ Non-traumatic-rare
- □ Lucid interval- <u>50%</u> after head impact loss of consciousness then becomes awake for sometimes then lose it again.
- C/F: headache, nausea, vomiting, convulsions, herniation.
 If it's large > blurred vision, respiratory arrest.
 - Symptoms of increase ICP
- Mechanism: Trauma
- **Effect:** <u>artery(Middle cerebral artery)</u>



1.2)Subdural Hemorrhage (SDH)

- Blood collection between Dura and arachnoid.
- **Crescent shape.**
- **Bilateral**, Supratentorial.
- Cross sutures, but not Dural attachments.
- May extend along falx and tentorium The presentation of SDH is more symptomatic

(because of its large size and herniation)

- Causes:Traumatic : Most common. Non traumatic : Child abuse.
- Stages of bleeding(For any type):
 Acute: 6hr-3d.

Sub-acute: 3d-3w it will become darker (greyer).

- Chronic: >3w like CSF.
- □ Mechanism: acceleration RTA .
- Effect: veins .

Herniation-brain midline is shifted to the right-brain tissue is herniating to the other side >there is something inside the cranium but outside the brain (mass effect). it differs from the first case because:

- the shape is different and it's large in size (no Sutures)

- there are <u>black spots</u> inside it indicating active bleeding, so when blood is clotted it becomes white on CT,
- the shape is convex from outside and concave inside so it's lunar.

This is MRI because bone is black, and over it is the skin, which is white, here there is subdural hematoma, which is outside the brain and enters the Dural reflections.

T2WI (MRI), the fluid is bright and hematoma is bright, so to differentiate we do CT scan, on CT fluid is dark and hematoma is grey, if hematoma is grey it means it's subacute or chronic. Here it is bilateral subdural hematoma







Cont..1.2)Subdural Hemorrhage:



Bilateral subdural, with mass effect or chronic Atrophy.







This Subdural hematoma and not sub arachnoid, because the dark area is the subarachnoid space (which has CSF).



Blood in the suprasellar cistern



Blood in the ambient cistern

Blood in the interhemispheric fissure



Blood in the sylvian fissures

Baby with cerebral palsy who has bilateral subdural hematoma ,But has <u>active bleeding (black)=acute</u> and the blood has precipitated <u>(white)=chronic</u> in the dependent part of the cavity.there is ventriciloperito-peritonial shunt due to cerebral palsy.

1.3)Subarachnoid Hematoma (SAH)

- □ Blood between pale and arachnoid .
- Traumatic (most common), 2nd most common cause :HTN then <u>aneurysm</u> and malformation.
 Non-traumatic .
- Headache, vomiting, blurred vision, Neck rigidity (because of blood that irritates the meninges > patient presents with meningitis like picture without fever (not bacterial).
- Complication: hydrocephalus , (acute/delayed) (blood clots may occlude Foramen Monro ,Foramen of Magendie or Luschka or cerebral aqueduct and cause acute hydrocephalus or occlude the arachnoid villi which reabsorb the CSF and cause chronic hydrocephalus), vasospasm, re-bleeding. vessels will penetrate the Dura and will run in the subarachnoid space to penetrate the brain, so if blood touches the adventitia it will irritate it and cause spasm.

NOTE (S):

*When young patient come complaining of <u>worst</u> <u>headache in his life</u> think about <mark>aneuryism</mark>

*What might cause aneurism rupture? <u>Steroid</u>

*Most common site for aneurism is ACA

*How does SAH looks? Hyper density will <u>appear inside sulci</u>,<u>fissure</u> <u>and Subarachnoid space</u>(inter Pedinecular fossa).

1.4)Intraventricular Hematoma:



Blood in the 4th ventricle ,3rd ventricle ,and lateral ventricles.



Blood in the ventricles and the parenchyma, so intraventricular and intraparyncemal hemorrhage.

1.5)Parenchymal bleed:

Causes : HTN, trauma, AVM*, aneurysm, prematurity, tumors, infarction, coagulopathy.

NOTE (S) :

*Intracranial hemorrhage: the most common site > basal ganglia (sensory defect).

*Intra parenchyma > coagulopathy or anticoagulant



CT blood in the parenchyma only an when you do MRI we see <u>black spots</u>, which are micro-bleeds and are asymptomatic and can't be seen by CT scan

*AVM= (Arterio-venous malformations) they rupture in parenchyma

Trauma





Cont..Trauma



- Subarachnoid bleeding in the sylvian fissure (lateral sulcus).
- There are two skull fractures.
- Black areas of air (pneumo--cranium)=pneumo--cephalous, air enters the cranial cavity by fracture with skin laceration or a fracture in the paranasal sinus (in the base of the skull) or air cavity, so here if there is no skin laceration, the fracture in the frontal sinus explains the pneumo--cranium.
- There is bleeding inside the frontal sinus.



2)Brain infarctions



Artery(MCA).

Note:

Cerebral Artery(PCA).



Picture (2):

The inferior part of the cerebellum and the lateral part of the medulla are supplied by posterior inferior cerebellar artery (PICA).

If we go higher up, the anterior part of the cerebellum is supplied by anterior Inferior Cerebellar Artery (AICA).

•Higher up the at the top of the cerebellum it is supplied by Superior Cerebellar Artery(SCA).





If you see a lesion that follows vascular territory then think of Ischemia (thrombus)

•ACA supplies the medial aspect of frontal lobe. Basal ganglia including internal capsule is supplied by MCA. PCA supplies thalamus.

•The <u>medial aspect</u> of brain (around the sylvian fissure) is

The temporal & occipital lobes are supplied by posterior

supplied by Anterior cerebral Artery(ACA).



The putamen & the Globus pallidus are abnormal on the right side they are dark compared to left side which is normal.
The sylvian fissure which is normally widely open in the left side compared to right side which is small because there is ischemia so, if there is ischemia there is edema(fluid) so we lose density(lose appearance of the structure).





T2WI-MRI shows: Fluid in the ventricle and abnormal fluid in the area of infarction(edema). So this area (basal ganglia) and the sylvian fissure, which is surrounded by the insular cortex are supplied by the middle Cerebral Artery.



Case: This patient presented with <u>sudden right hemiplegia</u>, what are the findings in this CT?
The left side is hypodense (darker) in comparison to the other side.
The triangle (putamen & globus pallidus) is not clear.

Also the sylvian fissure is closed because of the swelling of the cerebral hemisphere so CSF will be squeezed from the sulci & that's a sign of ischemia.

•Again in the middles cerebral artery.



How they present clinically patients who have MCA infarction that involved basal ganglia ? They present with motor deficit (loss of motor function) either hemeparesis or hemiplegia.
If sylvian fissure get involved they present with aphasia (sylvian fissure contains wernicke's and broca's areas).



There are other infarctions in the cerebellum by the superior cerebellar artery and inferior cerebellar artery.



This is PCA infarction involved occipital lobe and it affects posterior part of thalamus .
The calcarine fissure is absent in the left side which indicates a swelling and when it is swollen it means acute not chronic.
There is bleeding in the ventricle. How they present clinically patients who have PCA infarction? Vision loss (hemianopia)







Infarction in the anterior cerebral artery

The calcarine sulcus in the left side is not seen here and the falx is pushed to the other side (edema)so this indicates that this is acute ischemia , but if there is shrinking of brain tissue >chronic ischemia.
How this patient will present clinically? Paralysis of the right leg (monoplegia) but the hand and face will not be affected because they are represented downwards.

Acute ACA infarction





There is **bilateral Anterior Cerebral Artery ischemia** due to anterior cerebral artery aneurysm, which has bled before & thus, caused spasm then infarction. How the patient will present clinically? **Paralysis of both legs (paraplagia)**.







There is MCA infarction.



Chronic Bilateral MCA infarction.

Multiple infarctions in SCA patients

Lower part of the cerebellum is infracted.
We know it's lower because the medulla oblongata is seen, and those structures are supplied by the PICA. This condition is known as lateral medullay syndrome



Infarction of basal ganglia (posterior limb of internal capsule) which is supplied by MCA.

FLAIR

DWI

3)Brain edema

Types:

1) Vasogenic:

Vasogenic cerebral edema refers to a type of cerebral edema in which the blood brain barrier (BBB) is disrupted.

In case of: Trauma/infection/inflammation/tumors Finger projection (white matter)

1) Cytotoxic:

cytotoxic cerebral edema where BBB is intact. It is an extracellular edema which mainly affects the white matter, through leakage of fluid out of capillaries. In case of: Ischemia/trauma

•Both could be generalized or localized

•Both may co-exist

Imaging findings:

Hypo density on CT Low signal on T1, high signal on T2 & FLAIR Loss of GM/WM interface Compressed ventricles Effaced sulci & cisterns Dense cerebellum Brain herniation Vascular compression-ischemia

Cont..Brain edema









Cont..Brain edema

	Vasogenic	Cytotoxic
Location	White matter	Gray matter
DWI	Non-restricted	Restricted
Shape	Finger-like	Diffuse

MCQs: from the slides

The cause of this hematoma is:

- A. Anticoagulation
- B. Hypertension
- C. Ruptured aneurysm
- D. Trauma



This CT shows:

- A. Epidural
- B. Subdural
- C. Subarachnoid
- D. Intraparenchymal



This CT shows:

- A. Epidural hematoma
- B. Subdural hematoma
- C. MCA infarction
- D. All of the above



MCQs: from the slides

This CT shows:

- A. Subdural hematoma
- B. Subarachnoid hemorrhage
- C. MCA infarction
- D. All of the above





SDH, IVH, SAH, Pneumocephalus, Edema

MCQs: from the slides This CT shows:

- A. Epidural hematoma
- B. Subdural hematoma
- C. MCA infarction
- D. Normal brain





Thank You!

We hope you found this helpful and informative.

Done by:

- Muhanad Alshiridah
- Muhannad alwabel

Reviewed by:

Kholoud Aldosari

•Abdullatif Ahassan

You can always contact us at Radiology433@yahoo.com