ISCHEMIC & HEMORRHAGIC STROKE

435 medicine teamwork

[Important | Notes | Extra | Editing file]

lecture objectives:

- Not given but ,[Dr.fawaz:said most imp things to know how to differentiate from the clinical presentations if the pt has stroke or no,how to localize the lesion,and the initial management]

Before reading the lecture check out this link to revise the neuroanatomy شيكوا عليه بيسهل عليكم حياتكم

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References: Doctors' Slides+Davidson+kumar

Introduction to Stroke

Definitions:

Stroke: is defined as a syndrome of rapid onset of cerebral deficit (usually focal) lasting >24 h or leading to death, with no cause apparent other than a vascular one.

- **Stroke onset:** Timing of **FIRST** neurological deficit OR last time patient was seen well. **What if they woke up from sleep with a stroke deficit? نحسبه من the last time they were seen well. Is it fair though?** Most research says that if they woke up from sleep with a stroke it most probably occurred within a half hour, because half of the body becomes heavy so the patient cannot move from side to side, so they wake up, and they know from the diffusion perfusion image: [abnormal perfusion of brain tissue can be imaged with CT after injection of contrast media (i.e. perfusion scanning),This can be useful in guiding immediate treatment of ischaemic stroke]:
  - ✓ If there's **MISMATCH** → Do **Thrombolysis.**
  - ✓ If there's **NO MISMATCH** → no point from giving thrombolytics > so do **MRI** instead (will be explained later in details)

Be careful, as some patients can wake up at night (before stroke happens meaning they were still well) فلازم تسألونه قمت باالليل؟ صليت الفجر أو رحت الحمام؟ اذا قالكم ايه قمت ورحت وجيت لازم تنتبهون وتعيدون الحسبة

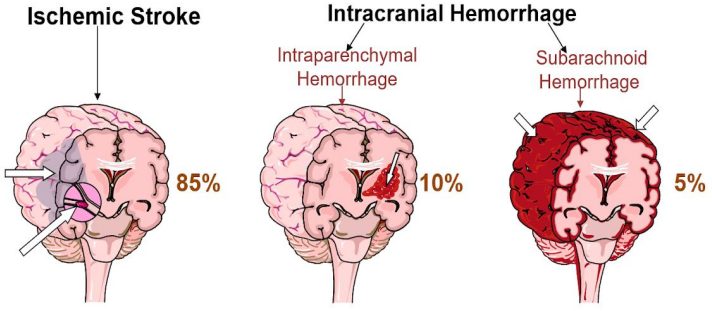
▸**What about sudden cognitive decline or behavior changes?** مشكلتها مانعرف متى أول مره لاحظ فيها المريض تغير في اطباعها :)) pt will not come immediately!That's why stroke scoring system (NIH)[1] doesn't include cognitive functions and behavioural changes in calculation!! NIH relies on (Motor functions- Sensory -language -level of consciousness).

Stroke in evolution: Is when the symptoms and signs are getting worse (usually within 24 hours of onset). **Why do stroke symptoms progress after several minutes or half an hour?** Because when the major artery closes, collaterals will open more and more but they will inevitably fail, so symptoms will be more severe leading to STROKE EVOLUTION!!

Transient ischaemic attack (TIA): Means a brief episode of neurological dysfunction due to focal ischaemia without infarction, usually lasting seconds or minutes with **COMPLETE recovery.** (Has to end with complete neurological recovery within one hour + Negative CT image). TIAs may herald a stroke. [what written in the book 'recovery within 24 hours' is wrong!!اذا شفت بكتبكم 24 ساعه اشطبها وحط بدالها ساعه وحده].

لو يفكونا من مصطلح TIA ابرك ومارح يفرق بالكلينيكال براكتس لاننا نعامل كل مريض جانا sudden neurological deficit على انه ستروك!

Types of Stroke:



Common Stroke Presentation:

Any SUDDEN neurological deficit: الكلمه المهمه المشتركة بينهم!! SUDDEN

- **SUDDEN** weakness.
- **SUDDEN** sensory loss/ visual loss.
- **SUDDEN** loss of coordination.
- **SUDDEN** aphasia.
- **SUDDEN** slurred speech.
- **SUDDEN** Loss of consciousness.
- **SUDDEN** headache characteristically with hemorrhagic stroke
- Others (depend on the affected vessel "will be discussed")

So, put a **STROKE** as DDx for any **Sudden** neurological deficit!! Stroke can present with wrist drop only, making you think primarily of radial neuropathy. So stroke recognition is difficult when there's an atypical presentation or atypical age

- e.g: 20 years old female MEDICAL student ياقلبي يالميدكل ستو دينت كلشي يحطونه فيكم :) with a headache and inability to talk, making u Think of migraine however, an inability to talk is a feature of stroke .

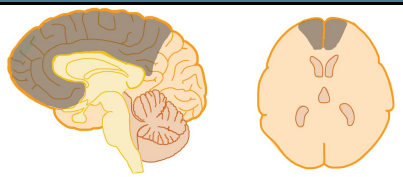
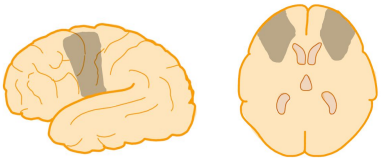
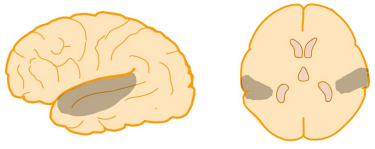
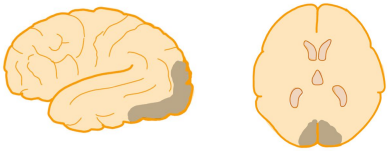
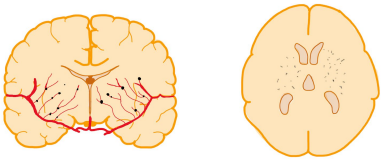
we can't know from the clinical presentation whether the stroke is hemorrhagic or ischemic الاعراض قد تشترك فيما بينهم,that's why we do CT with all pt who suspected to have stroke ,(ct will show u the blood if there hemorrhagic stroke)

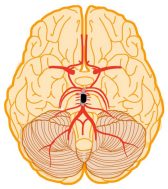
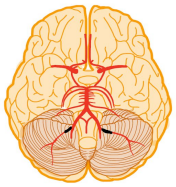
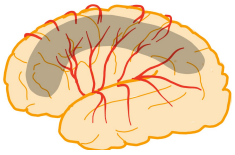
[1] National Institutes of Health Stroke Scale, or NIH Stroke Scale (NIHSS)

Ischemic Stroke

What is the typical presentation of the following arterial strokes?imp

#شكراً لمنيرة السلولي على رسمها الجميل للbrain ♡

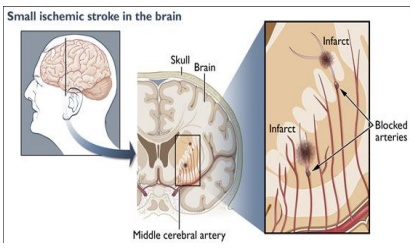
Artery occluded	Infarct Surface	Dominant Hemisphere	Nondominant Hemisphere
ACA	 frontal lobe	#Contralateral lower extremities weakness.Why? Because the motor area medial aspect is supplied by it. Why Leg? according to motor homunculus. #Abulia. (lack of initiation ,lack of interest). #cognitive dysfunction:will include a change in personality, withdrawal, any frontal lobe dysfunction, both can happen.	
MCA (ant. division)	 frontal lobe	#Expressive aphasia (broca's Area) يفهم عليك بس مايقدر يتكلم *"Video"* ***What's the difference between Aphasia & Dysarthria "Severe slurred speech"?*** *Aphasia*: Inability to formulate or intercept language symbols. NO muscle weakness. *Dysarthria*: Muscles used in speech r weak.Speaking's intact	#Aprosodia You can't know the person emotional status from his speech تصير نبرة صوته وحده سواء حزين سعيد خايف.
		#Contralateral hemiparesis(in Arm and leg)Why leg is involved?:الي قروا ملفي بيفهمون #Ipsilateral gaze deviation"Inability to move both eyes in the same direction" **Due to defect of frontal eye field.** مثلاً يقرأ كتاب أو يتابع فلم بدال ما يحرك عيونه ورا سه ثابت, لا يحرك راسه كامل	
MCA (post. division)	 Parietal lobe	#Conduction aphasia *"Video"* #Gerstmann's syndrome:very Rare ,not imp Gerstmann's syndrome Is characterized by four primary symptoms: 1)Dysgraphia/agraphia: deficiency in the ability to write. 2)Dyscalculia/acalculia 3)Finger agnosia: inability to distinguish the fingers on the hand. 4)Left-right disorientation.	#Anosognosia(not imp) Lack of recognition of sickness. #Apraxia(not imp)inability to perform a motor task that u used to do,NOT related to muscle weakness, however they respond to noxious stimuli # Contralateral Neglect.**اهم كوقنتف ديفيسيت أبيكم تعرفونه** (Doesn't pay attention to his/her left side & the left side of surrounded environment)يحلق شنبه اليمين بس يطنش! اليسار ,يكلم الشخص الي يمينه بس يطنش التي بيسار ه,جدا صعبين بالتأهيل **يمكن يجيكم سؤال مين أصعب بالتأهيل L MCA او R MCA؟** أكيد اليمين.الناس تظن الليفت اصعب عشان الافيزيا!بس لا النيقلكت(التطنيش) اصعب
		#Contralateral hypoesthesia.Reduced sense of touch or sensation. "Parietal lobe"	
MCA (post. division)	 Temporal lobe	#Receptive aphasia (wernicke's Area) *"Video"*مايفهم الكلام ويجاوبك شي ثاني ماله دخل باللي سألته وما يسكت كثير كلام	-
		#Contralateral homonymous hemianopia.'Due damage to optic tract which supplied by MCA'	
PCA	 Occipital Lobe	#Alexia without agraphia The patients are unable to read at all However,they are able to write. This is example of a disconnect syndrome in which information from the occipital lobe is not available to the parietal or frontal lobes to either understand or express what has been seen.	-
		#Contralateral homonymous hemianopia #Contralateral weakness. Why there's weakness?!! bc the P1 branch of PCA goes to cross cerbi,supplying the corticospinal tract. Why contra? bc it happens above the medulla where decussation happened)	
Subcortical lacunar الجلطة النقطية		Lacunes are SMALL infarcts seen on MRI or at autopsy. Hypertension is commonly present. Lacunar infarction is often symptomless,or with Minor stroke(Since it affects the internal capsule which contains tracts pass through it so there r various forms of presentations: pure motor stroke\or pure sensory stroke\or sensorimotor loss \or dysarthria from involvement of corticobulbar fibres)	

Basilar artery		Since it SUPPLIES the majority of the brainstem (which contains the reticular formation system 'the center of consciousness'). **THE MOST IMPORTANT presentation is loss of consciousness.**
PICA "Posterior inferior cerebellar artery"	 *PICA supplies the lateral medullary and inferior cerebellum.*	**Lateral medullary syndrome** (wallenberg syndrome):Sensory deficit in opposite side + Ipsilateral ataxia. (!!موضوع محبب بالاختبارات) ischemia in the lateral part of the medulla→contralateral Sensory deficit ischemia in inferior cerebellum→ Ipsilateral ataxia
Watershed Ischemia	 *Between ACA-MCA*	-Depends on the areas with distal branches, which are the first parts get affected by Hypoperfusion. -so if there was "strong or even moderate"**hypoperfusion** due to any cause (hypotension) will give you watershed ischemia. Most common is between MCA & ACA, and between MCA & PCA. there will be **PROXIMAL** muscle weakness in the arm and leg more than distal*(called man in the barrel syndrome).* **Q)Can there be WATERSHED between the PCA AND ACA?** Yes but it's in the medial part of the brain.

to localize the stroke حسيتونا طولناها عليكم :)) تبون شي أقصر:)) هنا ملخص للأشياء التي قال الدكتور انها مُهمة و هي الي يبينا نعرفها

What's the typical presentation of occlusion in of the following arteries ?

- **Right \left ACA:**contralateral lower extremities weakness +cognitive function deficit
- **left MCA:** aphasia+ contralateral sensory loss+ hemiplegia of upper and lower extremities +contralateral homonymous hemianopia.
- **Right MCA:** SAME presentation minus the aphasia, what is in its place? Neglect of whatever on his/her left side.
- **Right \left PCA:**contralateral homonymous hemianopia+contralateral weakness
  - ✓ PCA can mimic MCA but without hemispatial neglect nor aphasia
  - ✓ if u get a case pt has *Contralateral visual field loss +weakness,*What is the first artery u should think about? MCA,but keep in mind PCA can give the similar presentation, if u assess aphasia ,cognitive function and found them spared this mean it's PCA!
- **Subcortical lacunar:**small infarction usually Asymptomatic ,commonly found in HTN pt
- **Basilar artery:**THE MOST IMP presentation is loss of consciousness(can be only the clinical presentation).
- **PICA:**MCQ!! Lateral medullary syndrome 'Willingburg syndrome':(Sensory deficit in opposite side+ Ipsilateral ataxia)
- **Watershed Ischemia:PROXIMAL** muscle weakness in the arm and leg more than distal*(man in the barrel syndrome).*

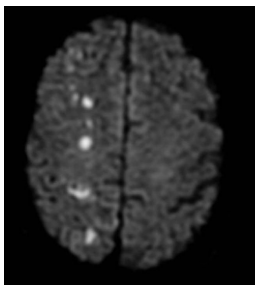


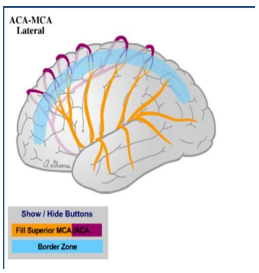
Lenticulostriate arteries supply the basal ganglia and internal capsule.

two issues make the post limb of internal capsule more vulnerable to stroke?No anastomoses+they all are end arteries

عندك ال MCA اكبر ب30 مره من الارتريز الصغار الي تطلع منها !!هنا مشكله انه هالارتريز السعونه ماحتتحمل الضغط العالي الي قادم لها فتنفجر بسهوله!!

you have 15 arteries, يعني ١٥ احتمالية





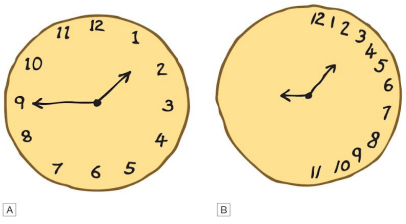
The MRI shows unilateral infarction between right(ACA), (MCA) and (PCA) territory.

اممم ليه منطقه وحده تأثرت بالهيبو بيرفيوجن؟!اول ماتشوفها يونيلاترال لازم تتسائل! مو المفروض كل الجهتين تتأثر ! فمعناته عندنا من قبل

right partial artery occlusion due to atherosclerosis

بما انه هذا الارتري عنده انسداد جزئي فعشان يوصل دم للمخ محتاج كميه دم كبيره !!فهو اول جهه تتأثر ,طيب إيش اول شي تسويه لما تشوف واترشيد في جهه وحده فقط,غير من انك تتأكد من ضغط الدم؟

Vascular image either carotid US or CT angiogram.



Clock drawing test:

[A] Image drawn by doctor.

[B]Image drawn by patient with left-sided neglect.

Treatment:

Whenever we say acute stroke, it means acute ischemic stroke.

1	**Primary Stroke Prevention**	Whenever we talk about stroke treatment, we mention the primary prevention, what does it mean? Preventing a stroke in a patient who has never had a CVA (neither TIA nor stroke)
2	**Acute Stroke Treatment**	Intervene within a few hours for clot lysis to ensure re-gain of function/recanalization/prevent necrosis
3	**Secondary Stroke Prevention**	Prevent stroke recurrence or prevent stroke after TIA
4	**Stroke Rehabilitation**	To minimize stroke physiological and psychological impact.

Sudden Neurological deficit = Acute stroke!

1) Activate Stroke Code.
2) HISTORY: Stroke Onset(very important), progression, fluctuation.
3) Check inclusion and exclusion criteria for IV T-PA(Tissue plasminogen activator) use.
4) Blood work up include:

CBC	INR & PTT "Make sure there's no bleeding disorders because of possible t-PA"	Creatinine "To know if we can give contrast"	Glucose[2] and electrolyte Hypo/Hyperglycemia can mimic stroke causing neurological deficits	Troponin "Mural thrombus or recent MI>Stroke. Also WHY we have to check it? If we give t-PA with post-MI"Wall Necrosis" will lead to "Cardiac Tamponade" due to rupture! In General, Any recent MI we prefer NOT give Thrombolysis.In Instead, we go to other modalities like endovascular.

& 12 lead ECG(bc Afib is the most common cause of ischemic stroke due to embolism) .

5) 2 Peripheral IV lines and Foley's catheter: Avoid urinary catheterisation unless patient is in acute urinary retention,If signs of dehydration give IV fluids "On IV line for Treatment & one for IV fluid resuscitation".
6) Urgent CT then CT angio brain:

- Non-contrast CT will demonstrate haemorrhage immediately but cerebral infarction is often not detected
- **1st:** Make sure no Hx of renal disease" because Contrast in CT angio is contraindicated"
- **2nd:** We need to do more then two modalities. Why? If a diabetic patient came with arm weakness and normal sensory, visual field and language. **Is this cortical or Subcortical?** it's ***SUBCORTICAL*** lesion like Lacunar. This one doesn't need CTA it's small vessels occlusion. "Lacunar stroke is higher in KSA because of DM"
- We do CTA if we suspect **LARGE vessel occlusion.** Why? Because from experience we know that some cases 80% don't benefit from IV t-PA only, the vessel won't open! So we have to prepare Angio-Suit in the ER also!! If the patient improved from IV t-PA only we can cancel CTA.

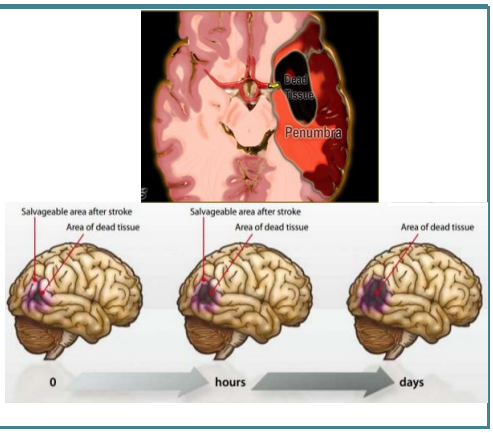
Stroke Penumbra

The Target of Acute Ischemic Stroke Treatment!!

- **Penumbra** is zone of **reversible/Symptomatic** ischemia around **core** of **irreversible/Symptomatic** infarction—salvageable in first few hours after ischemic stroke onset due to the collateral arteries that supply the penumbral zone. It's exists either due to collaterals or incomplete vessel occlusion. So, We have Hypoperfusion/Oligemia > Before Necrosis! And Occlusion of an artery can be less than 100%.

-The goal of Acute stroke Rx to rescue the penumbra by max use of available Rx strategie "TIME IS BRAIN: SAVE THE PENUMBRA"

#NOTE: More detailed studies involving perfusion-weighted images and diffusion-weighted MRI will differentiate the infarct core and the penumbral area which is potentially recoverable



[2] Elevated blood glucose is common in the early phase of stroke. Ischemia time seems to fly faster with patients with diabetes or grave hyperglycemia especially lacunar stroke. WHY? Hyperglycemia may exert direct membrane lipid peroxidation and cell lysis. Moderately and severely increased blood glucose has been found to further the metabolic state and mitochondrial function in the area of ischemic penumbra!! "Link"

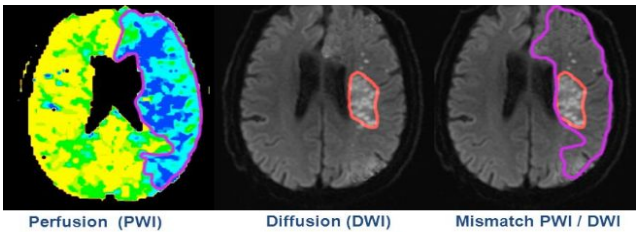
Modalities of Acute ischemic Stroke treatment:

IV t-PA	Intra Arterial t-PA	Mechanical Disruption	Surgical Rx (old)
"Standard" (Tissue Plasminogen Activator)	Not used any more	"standard"	Not used any more

Don't worry about the studies mentioned in slides.

IV t-PA (Alteplase)

- IV t-PA **standard** treatment until FOUR AND HALF hours except basilar stroke can be given until 8 hours, If we go beyond 4 hours we have to use modality to check diffusion perfusion ratio > we only treat if there is **MISMATCH**! *What's the diffusion/Perfusion in ischemic stroke?*
(Diffusion =The necrotic tissue), (**Perfusion=**Tissue at risk "Penumbra")

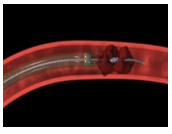


Inclusion criteria	Exclusion criteria
○ Clinical Dx of stroke. **"The most Imp. thing"** ○ Stroke onset less than 270 minutes. - **Patients:** *who improve in the 1st 90 min better then who improve in the 2nd 90 min and so on...* - 1st(90 min): 2 days → No deficit. Healthy! خلال يومين يطلع سليم - 2nd(90 min): 7 days يطول معه العلاج ويطلع خلال سبع أيام - 3rd(90 min): 13 days وهذا عالجناه بثالث 90 دقيقة رح يطلع من المستشفى تقريباً بعد 13 يوم! - **So, the late you start treatment the long hospital stay!!** ○ Age is more or = 18	○ **Intracranial Hemorrhage** in imaging or clinical presentation suggests SAH. What's the purpose of CT? **To rule out Hemorrhage.** ○ Active/ recent internal bleeding or on warfarin with **INR** > 1.7 or platelets < 100K. ○ Serum **Glucose** <50 or > 400. ○ Systolic **BP** > 185 or diastolic >110."At risk of bleeding and rupture" ○ **Recent MI** . ○ Recent major **surgery** or trauma. In cataract surgery you can do t-PA. ○ Recent **arterial puncture** at noncompressible site.

Mechanical Disruption:

- **Does recanalization benefit all patient?** NO, when there is already necrosis it's putting the pt on an increased risk of **HEMORRHAGIC TRANSFORMATION=** "Hemorrhage after ischemia"
  → This is treated as hemorrhagic in the first week and then TREATED as ischemic.
  → Treat underlying cause / give aspirin / if A-fib is the cause then you have to give anticoagulants. بتلقون تفاصيل احلى مع الهيمورجك ستروك

- **Endoarterial Mechanical Disruption:** الدكتور قالي هذي مره ادفانسد عليكم ومابيكم تعرفونها حطيتها بسلايداتي لاني ادرس سنه خامس و ريزدنت نفس الموضوع ومافيني اغير المحتوى لكل مرحلة:((((

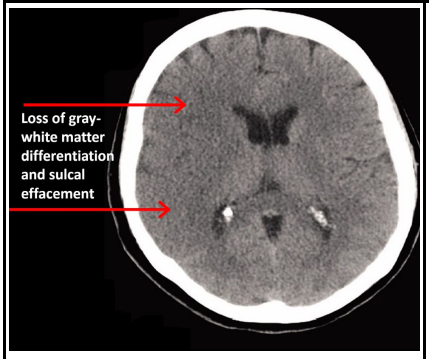
Merci Retriever *"1st FDA approved device"*	*Penumbra system* *"FDA approved"*	Solitaire Device *"3rd Generation device"*	Trevo retriever *"3rd Generation device"*
Increased recanalization rate and secondary clinical outcome when used for large cerebral arteries. AMAZING VIDEO 	It does: **clot suctioning**. Similar rates of recanalization and clinical outcomes to Merci retriever AMAZING VIDEO 	Solitaire was superior to Merci Like stent!It doesn't matter what kind of stent. AMAZING VIDEO 	Trevo was superior to Merci. Depend on experience. AMAZING VIDEO 

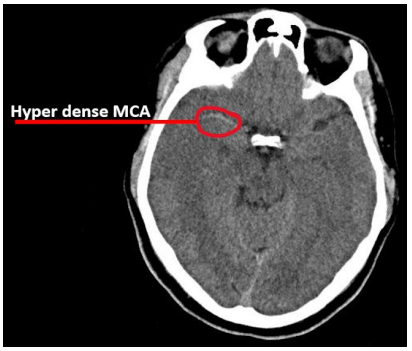
- **Endoarterial thrombolysis:**(Combined IA and Mechanical disruption) WHEN? In Contraindicated t-PA & large vessel occlusion.For large occlusion/selective cases/ whenever t-PA is contraindicated or Patient taking Warfarin and his INR is 2, Here we can not give t-PA! It's **ALTERNATIVE** FOR t-PA.

Barriers for Acute stroke therapy:

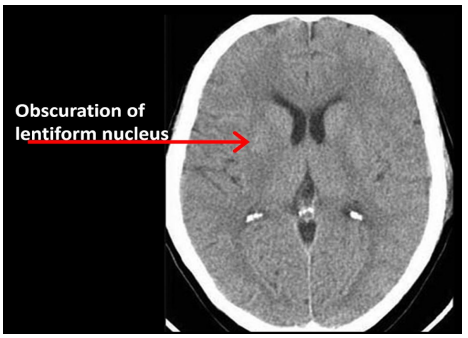
- Late patient presentation to ER (In USA; only 30% present within t-PA window)
- Poor stroke recognition and delayed triage at ER (mainly for un-usual stroke presentations)
- Lack of appropriate infrastructure. (((آه كلمة انفر استركتشر ذكرتني بماده غثيثه لين الحين و هم ناشيين بحلوقنا و الوركشوب حقتهم:
- Lack of acute stroke expertise
- Presence of a contra-indication
- Difficulty in getting patient's or family's verbal consent.

Acute Ischemic Changes in CT





in CT as the blood clot retracts it becomes more hyperdense(white)



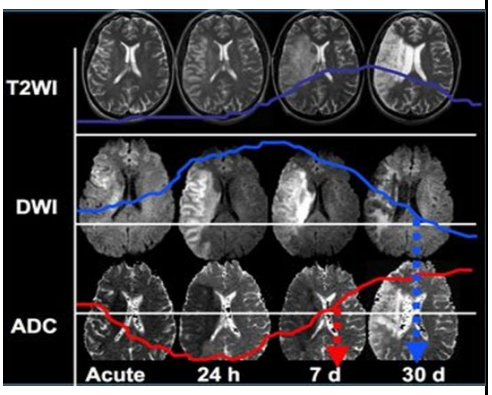
A 45 yr old male with weakness in Lt side for 2 hrs.

Acute Ischemic Changes in MRI

الي يهمني تعرفونه ويمكن اسألكم عنه الي هو ان

Diffusion-weighted MRI (DWI) can detect cerebral infarction immediately earlier than CT and other MRI sequences

More detailed studies involving perfusion-weighted images and diffusion-weighted MRI will differentiate the infarct core and the penumbral area which is potentially recoverable.

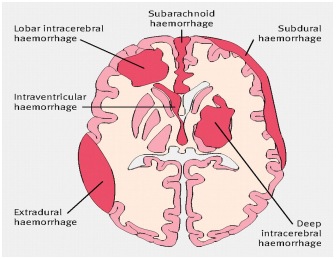


Hemorrhagic stroke

Intracranial haemorrhage(according to the anatomical location) ***Comprises:***

- Intracerebral and cerebellar haemorrhage.
- Subarachnoid haemorrhage.
- Subdural and extradural Haemorrhage/haematoma.
- Intraventricular.(if isolated from veins within the ventricles), if not isolated then it may be due to intracerebral hemorrhage.

#"The Most common cause of brain bleeding in Riyadh is RTA's (Road traffic accidents)"



Subarachnoid Hemorrhage(SAH):

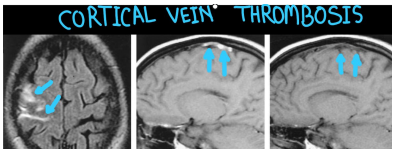
Features:	● Acute severe headache .**SUDDEN** onset of headache doesn't have to be the worst. You should ask the patient how it occured and what he/she was doing. Consider any SUDDEN is SAH until proven otherwise. ● High mortality – 50%; high morbidity. 20%-30% will arrive to the emergency dead. ● Subarachnoid haemorrhage (SAH) is less common than ischaemic stroke or intracerebral haemorrhage.
Diagnosis	⇒**First Do <u>CT</u>, CT is the best for blood** – 90% sensitivity,It can miss 10%: - The area of blood will be Hyperdense(white)in CT. ⇒**If CT negative <u>Do Lumbar Puncture</u>(LP):** - Lumbar puncture is not necessary if SAH is confirmed by CT, So, if CT is negative and you have a scenario of sudden severe headache do a spinal tap looking for blood in the CSF (**xanthochromia**). Within 12h of SAH and remains detectable for 2 weeks. - Why not to do CT angiogram before LP? you still can miss 7% so, CSF tap is more reliable (less than 3 millimeter will be missed),then If +ve do CTA. ⇒**If CT positive or,LP positive do <u>CT angio</u>:** - to demonstrates an aneurysm (the cause of the vast majority of SAH) - In the past **conventional angiogram** was done using a cerebral catheter, there is a correlation with silent ischemia,**Only used when CT angiogram is negative.** ⇒**lastly do <u>MRI</u> :**(FLAIR,DWI, hemosiderin Diffusion[3] etc ... لما نطلب ام ار اي يسون معها باكيج كامل) : - We don't do MRI in acute setting!!
Etiology	See table below

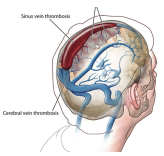
Aneurysmal SAH [85% of SAH]	Nonaneurysmal SAH [15% of SAH]
Ruptured saccular "Berry" aneurysms are the most common cause. الدكتور انهار على الانيورزم!!!EXTREMELY IMP	-perimesencephalic SAH[10% of SAH]it's A type of SAH (small hemorrhage around the brainstem)سببها موش معروف -Arteriovenous Malformation -rare causes: ○ vasculitis ○ Reversible cerebral vasoconstriction syndrome* ○ Cerebral Sinovenous thrombosis**it more commonly causeS IPH rather than SAH. ○ Dural Arteriovenous fistula (*see the img →*) 

For your information:

***Reversible cerebral vasoconstriction syndrome:**is a cerebrovascular disorder associated with multifocal arterial constriction and dilation Can cause: SAH(convexity SAH),epidural hemorrhage, or parenchymal hemorrhage ||Risk factors:women ,migraines,vasoconstrictive drugs

****Cerebral sinovenous thrombosis:**unusual cause of acute non-traumatic SAH,The exact mechanism of SAH in patients with CVT is still unknown. It is believed that venous hypertension causes dilatation and rupture of the cortical veins, with the subsequent bleeding into the subarachnoid space.



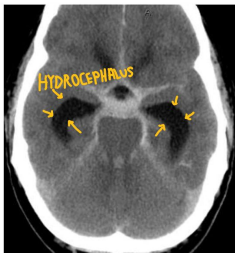


[3] ironcontaining blood products; useful for identifying microhemorrhages

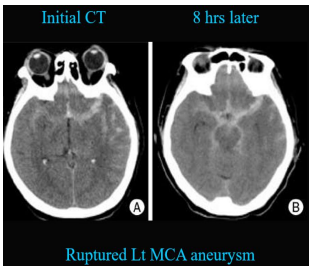
Complications of SAH
the complications only occur if there is bleeding!

Hydrocephalus

Since the CSF is being reabsorbed by arachnoid in the subarachnoid space, if there is bleeding the CSF won't be absorbed. device can be used to drain it or even a shunt.



Rebleed



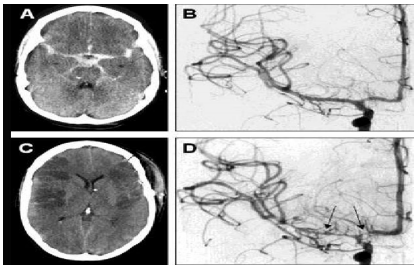
central hyponatremia:

etiology is unknown

Vasospasm/Ischemia
(most imp/dangerous)

Occur between day **4** and **14** it can lead to a diffuse stroke.وماحد يعرف ليه تطلع هالايام,لازم نراقب المريض هالفتره عشان نمنعها

Most commonly occurs around the vessel that ruptures. Nimodipine prevents it given for 21 days.



#NOW LET'S TAKE SOME CAUSES OF SAH AND DISCUSS THEM IN MORE DETAILS :

Aneurysm [85% of SAH]

Features:

- Mild to severe *(Fischer scale 1 -4):*[4 :All the subarachnoid spaces have blood.] [2 : two areas]. [1: only one area]. There is a proportional association between the score (extent of bleed) and prognosis.
- **Risk factors:** Family history , HTN, atherosclerosis , There is an association between polycystic kidney and the presence of an aneurysm. Connective tissue disease (marfan, ehlers-danlos), but the majority are not associated with any of them.

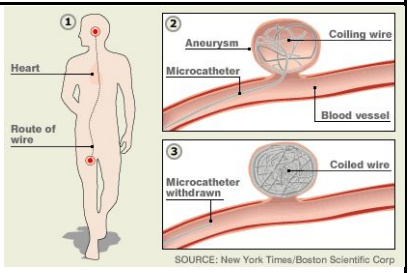
locations:

Common locations:the anterior communicating artery (30%)|posterior communicating artery (25%) |middle cerebral artery (20%)|Basilar tip(5%).

- Can be Multiple: in 20% of cases
- Localizing value:Location can be in the anterior communicating artery and Posterior communicating artery it's here because of the **pressure** due to the anatomical position.

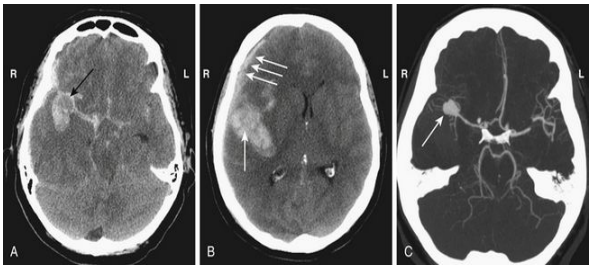
Diagnosis & RX:

Diagnosis: CTA – 93% "Quick, accurate, non-invasive".
RX: Ruptured berry aneurysm **should undergo CT angiogram** of the brain to look to for it once they identify it they call the interventionist to coil it. (it is done through a catheter through the groin and then inject coil it closes the aneurysm).Coilng is now the first line treatment.

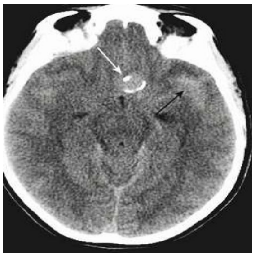


✓ What if you find it incidentally before it ruptures? single less than 5mm in size you watch more than 5mm you intervene.(some researches stated that, regardless to the size u have to intervene ,so it's controversial point)

Imeging:



Rt MCA Aneurysm



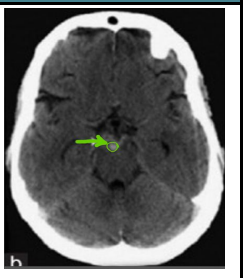
Calcification in aneurysms
Whenever you have a calcified aneurysm it's a **good** thing it means it won't rupture anymore.

Perimesencephalic nonaneurysmal subarachnoid hemorrhage (pnSAH)

Features:

Perimesencephalic means around the brainstem

- 10 % of all SAH
- subtle 'small' SAH Around the pons, midbrain.
- **CT:** SAH in the interpeduncular cistern (*see the img →*)
- **CT Angio negative:**no aneurysm
- the etiology is unknown can not be identified on CTA nor angiography
- Probably venous bleed.
- BENIGN outcome – no rebleed, vasospasm, or ischemia.



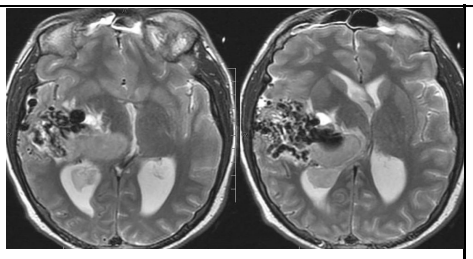
Arteriovenous Malformation (AVM)

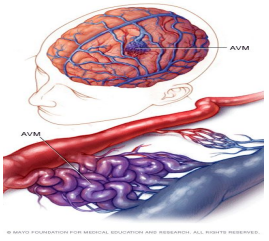
Definition:

- What is it ? AVMs are vascular developmental malformations, the blood goes directly from the arteries to the veins and the veins will dilate and with time will rupture. It usually causes intraparenchymal but occasionally causes subarachnoid.
- mixed Intraparenchymal hemorrhage + SAH

Imaging:

IPH + SAH due to AVM looks like a bunch of worms on imaging





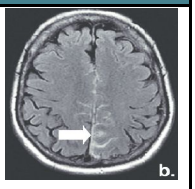
#There's a rare type SAH when the hemorrhage is confined only over the cortex,called:

Non-traumatic acute Convexity sulcal SAH [NOT imp]

What is it

Convexity subarachnoid haemorrhage is a rare type of spontaneous, non-traumatic, and nonaneurysmal SAH [AKA acute cortical subarachnoid haemorrahge] characterised by blood collections in one or more cortical sulci in the convexity of the brain,it's confined over cortical hemisphere and does not extend into the parenchyma, ventricles, or basal cistern

Complication are NOT rare (not rare مكتوب بسلايداته ريز بس هو يقول غلط الصح انها)



Etiology remains uncertain **[u can ignore them]**

- Cortical vein thrombosis (CVT)
- Cerebral amyloid angiopathy (CAA)[4] – *in **older patients***.it's more commonly causes IPH rather than SAH
- Posterior reversible encephalopathy syndrome (PRES)[5]
- Reversible cerebral vasoconstriction syndrome–*in **younger patients.***
- Vasculitis.

Pseudo SAH:

Pseudosubarachnoid haemorrhage is a sign related to apparent increased attenuation within the subarachnoid space which mimcs a true SAH,SO IT'S MORE RADIOLOGICAL THAN CLINICAL, the listed below r some conditions that cause hyperdensity in ct mimicking SAH:

- **Meningitis** – pyogenic.
- **Diffuse cerebral edema**, Global Hypoxia.In brain swelling due to acute hypoxic encephalopathy, the increased attenuation in the basal cisterns and subarachnoid spaces on CT scans is similar to findings of acute SAH"pseudo SAH"
- CT appearance of intrathecally administered contrast material, or leakage of high-dose intravenous contrast medium into the subarachnoid spaces.إفيمكن يللخبطو ا ويحسبو ا ان الكونترا ست بليدنق
- Venous angioma on NonContrast CT. بالذات في النونكونترا ست سي تي تبين هايبر دنس فالشخص ممكن يظن ان هذي الهايبر دينستي بليدنق ,لكن لما تسوي ام ار اي بيان عندك انهاماس (انجيوما)

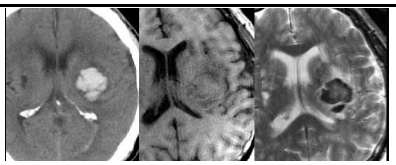
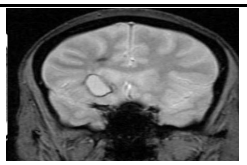
▸NOTES FYI: [kumar]

#migraine:SAH must be differentiated from migraine. This is sometimes difficult – a short time to maximal headache intensity and the presence of neck stiffness usually indicate SAH. Thunderclap headache is used (confusingly) to describe either SAH or a sudden (benign) headache

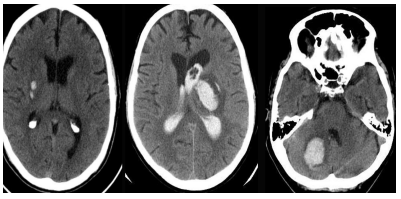
#Acute bacterial meningitis occasionally causes a very abrupt headache, when a meningeal microabscess ruptures; SAH also occasionally occurs at the onset of acute bacterial meningitis.

[4] **Cerebral amyloid angiopathy (CAA)**, also known as congophilic angiopathy, is a form of angiopathy in which amyloid deposits form in the walls of the blood vessels of the central nervous system.
[5] **(PRES)**, is a syndrome characterized by headache, confusion, seizures and visual loss.

Intraparenchymal Hemorrhage(IPH):

Diagnosis:	**The appearance of blood depends on its duration Brain bleeding CT:** ● **Acute** → High density (hyperdense). ● **Sub-acute** → Has a density similar to white matter density (isodense). ● **Chronic** → Decreased density (hypodense)
Imeging:	Acute  Chronic 
Etiology:	1. The most common cause is Hypertension,**Number one cause is HTN!!! you shouldn't forget!!** 2. The second common cause is Amyloid angiopathy– in elderly 3. Vascular :AVM, Cavernous Hemangioma 4. Neoplastic: Glioblastoma multiforme(GBM), Metastases 5. Coagulopathy **let's reclassify the etiology by Location:** - **Basal ganglia:** ○ Elderly - HTN ○ Young - Drug abuse. - **Lobar:**(any hemorrhage in cerebral white matter called lobar) ○ Elderly - Amyloid, HTN, neoplasm, Sinus Venous Thrombosis ○ Young – AVM, coagulopathy, Sinus Venous Thrombosis. - **Gray-white interface:** ○ Metastases, septic emboli, fungal infection ▶NOTE:Aneurysms and AVMs may bleed due to HTN surge.
Complications:	- Increased intracranial pressure. - Rebleed. - Edema. - Herniation. - infarcts. - Intraventricular extension, hydrocephalus. **4 poor prognostic signs:**elderly,large hemorrhage,extended to the ventricle,low GCS (Glasgow Coma score)

#NOW LET'S TAKE SOME CAUSES OF IPH AND DISCUSS THEM IN MORE DETAILS :

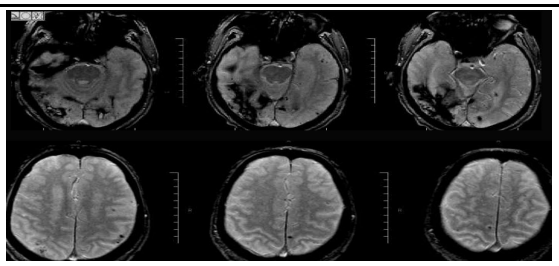
Hypertension hemorrhage: (80-90%)	
Locations:	1. Lentiform nucleus: 50% - 60%.What is the most common location? Putamen (basal ganglia)(**MCQ**) 2. Thalamus: 10% - 25%. 3. Pons: 5% - 10%. 4. Cerebellum: 5% - 10% 5. White matter. 
Features:	it occurs with **CHRONIC** HTN ,doesn't have to be malignant HTN ,some of the patients the HTN is controlled ✓ **CHRONIC** HTN causes degeneration of small arteries within the brain parenchyma, leading to micro-aneurysms, which can rupture easily. Found in older patients as risk increases with age.
Rx:	**How to treat?** Drainage doesn't work because the tissue is deep the only indication to drain is when its superficial or most importantly in the cerebellum (very accessible) (very serious may compress the brainstem). ✓ The only treatment available is admission to the neuro ICU to prevent complication. ✓ The best way to manage is to prevent and **treat HTN**

Amyloid angiopathy (80-90%)

Features:

- what is it?Deposition of amyloid protein on the vessel wall making it very fragile.
- Increased incidence with older age. Sometimes associated with alzheimer's and dementia.
- Lobar hemorrhage: Frontal>Parietal>Occipital>Temporal.
- Recurrent hemorrhages – 10% per year.
- No treatment available!
- Clinical diagnoses can't undergo surgery if you touch the vessel it will bleed.

Imeging:

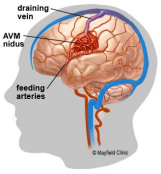


How can you differentiate between hypertensive and amyloid? Do a sequence MRI in amyloid you will see multiple old hemorrhages this is indicative of amyloid. It means that the patient has been bleeding every now and then (new and old bleeding).

Arteriovenous Malformation (AVM)

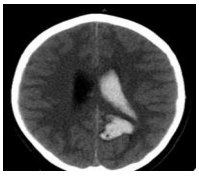
Features:

- Bleeding from the nidus or the proximal portion of the draining veins.
- **Hemorrhage could be in:** parenchymal, intraventricular, SAH.
- High chance of recurrence of hemorrhage.



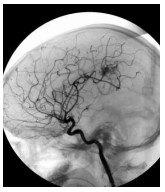
Diagnosis:

initially do ⇒**Noncontrast CT:**
shows hemorrhage, calcification



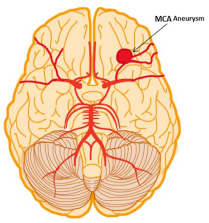
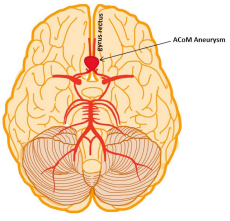
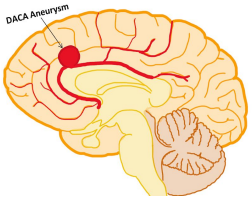
diagnostic⇒CT angiography:

- Can be negative initially (20%).
- Repeat angio after clot has resorbed.



Aneurysms

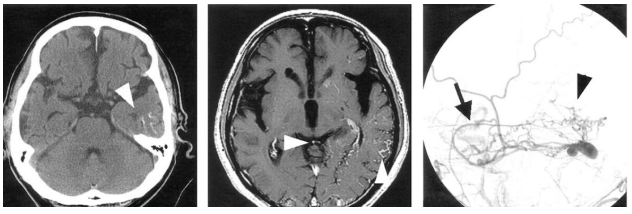
Specific sites:

MCA aneurysm - deep frontal lobe	**ACom aneurysm –** gyrus rectus.	**Distal Anterior Cerebral Artery(DACA)** – corpus callosum, medial frontal lobe.
		

imaging:

Noncontrast CT:curvilinear calcification. *(see the 1st img in the right →)*

#for sure u can't see the aneurysm in plain CT so, as mentioned earlier if u suspect aneurysm u should do CT angio ,to demonstrate the aneurysm "diagnostic"

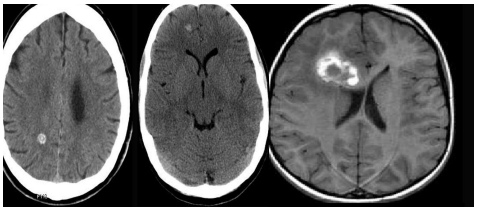


Cavernous angioma

Features:

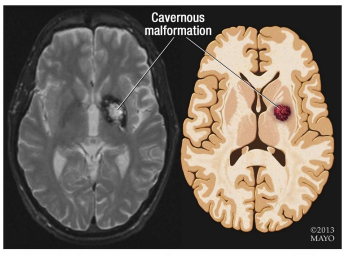
- It is a very benign form of vascular malformation causes **MINIMAL** bleeding every now and then we usually do nothing unless occasionally there is a large bleed.
- they may present with seizures so you just treat the seizures.
- Recurrent microscopic hemorrhages.
- Enlarge slowly; sometimes catastrophic(causing sudden great damage).

Imeging



it looks like popcorn

Noncontrast CT:slightly hyperdense, no edema.



Example of a cavernous malformation as seen on an MRI

MRI :diagnostic

Venous sinus thrombosis [very imp,i want u to know it]

pathophysiology: **how it can cause hemorrhage?**Cerebral venous sinus thrombosis (CVST) occurs when a blood clot forms in the brain's venous sinuses. This prevents blood from draining out of the brain. As a result, blood cells may break and leak blood into the brain tissues, **forming a hemorrhage**

Predisposing factors:

Dehydration, local neoplasm/infection, hypercoagulation, Polycythemia, drugs (OC pills, L-Asparaginase, Tamoxifen).

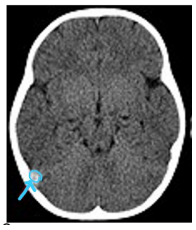
#Dural (cerebral) venous thrombosis: Common due to OCP , dehydration and commonly seen during pregnancy especially postpartum (due to the hypercoagulable state).

- Scenario: A 25 year old lady postpartum comes with headache not recovering and getting worse you do an MRI and find a dural venous thrombosis also hemorrhage.

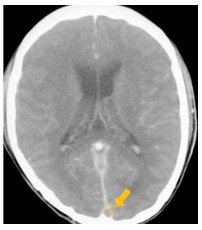
Rx:

Treatment : Anticoagulation. **ONLY indication to give anticoagulation in hemorrhage.**

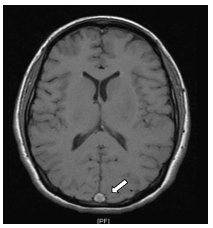
diagnostic images:



Noncontrast CT: cortical veins edema, hemorrhage, venous infarct.

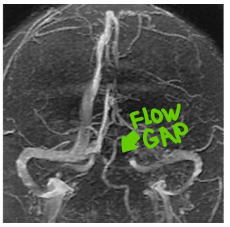


Contrast enhanced CT: filling defects.



MR:hyperintesty (SAGITTAL SINUS THROMBOSIS)"arrow"

diagnostic



MR venography:used to examine the intracranial venous system, particularly in the evaluation of dural sinus thrombosis[will show no flow in the veins]

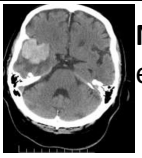
Neoplasms

Tumors can cause IPH:

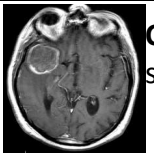
cancer invade the blood vessels → bleeding

- **Metastases:**any mets like **melanoma** (most common to bleed), renal, lung, breast Ca,choriocarcinoma,Pituitary adenoma.
- **Primary tumors:**GBM, choroid plexus cancer.

images:



Noncontrast CT: hemorrhage, edema +++

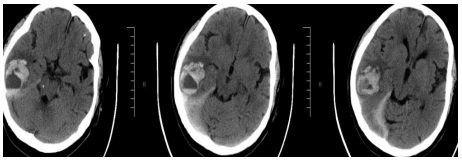


Contrast enhanced CT: some enhancement.

Sometimes the bleeding can mask the mass so you repeat the scan is a couple of weeks you will see the mass. For example if a SMOKER comes with bleeding look for lung cancer mets.

Hemorrhagic infarct [AKA hemorrhagic transformation]

- it's a complication of ischemic stroke → The infarcted tissue caused fluids to accumulate(Edema in arterial territory) *then* → Hemorrhage develops later (hours to weeks).
- Ischemic stroke can spontaneously bleed 30% of cases have hemorrhagic transformation within 1 week-2 week.
- It is believed that haemorrhagic transformation occurs as a result of preserved collateral perfusion or from reperfusion of infarcted tissues which have weakened vessels (i.e. from extravasation)



Infarct 3 weeks prior - Now on Anticoagulation

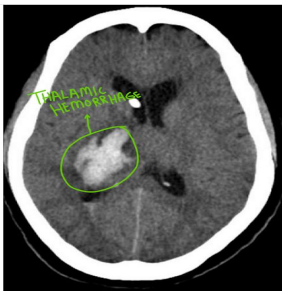
Coagulopathy

Anticoagulant Therapy:	- 7 to 10 times higher risk at therapeutic levels. - 2/3 are IPH: 1/3 are SubDural Hemorrhage If a patient presents with cardioembolic stroke from Afib how can you prevent another stroke? give anticoagulation(warfarin) but it can cause bleeding so you have to wait for 2 weeks due to the risk of hemorrhagic stroke. student said: دكتور معقوله الي عنده اسكميك ستروك نخليه اسبوعين بدون مسيلات دم :O The Doctor said: There is a risk of recurrence of the ischemic stroke within the 2 weeks but the risk of bleeding is much higher.
Thrombolytic therapy:	- **Thrombolytic therapy:** like STK, UroK, TPA - risk of intracranial hemorrhage when thrombolytic is give for acute Myocardial infarction is 1% -2 % .
Aspirin:	SLIGHTLY increased risk. not significant

Drug abuse

Any YOUNG patient coming with bleeding or ischemic stroke do a drug screen.

- **Drugs cause IPH:**Cocaine,Heroin, Amphetamines, phencyclidine.
- also drug abusers can have cerebral Vasculitis(angiography shows vasospasm due to vasculitis)
  - ✓ Cocaine:similar to HTN hemorrhage[Acute Thalamic hemorrhage *(see the img →)*]if young man comes to u with acute thalamic hemorrhage مابدا تفكير اكيد بسبب الكوكيين :×



Imaging guidelines in Intracranial hemorrhage:

1) **Noncontrast CT: initial** imaging of choice for suspected Intracranial hemorrhage .
2) Surgical candidates without known cause → **CT angio**.[in other words:Spontaneous Intracerebral hemorrhage without history of trauma or chronic hypertension, we need to proceed with CTA (CT angiogram) to assess for AVM]
3) If CT angio is not possible → **MRA or catheter angiography**.
4) **Old hypertensive patients :**no need for CT angio.
5) **Suspected mass/cavernous hemangioma:**MRI is **diagnostic** .

MCQs

1)A Patient presented with sudden weakness and aphasia. what is the most likely affected artery due his stroke?

A. Anterior cerebral artery
B. Posterior cerebral artery
C. Middle cerebral artery
D. Posterior communicating artery

2)a 60 years old male presented to the ER with 3 hours of central chest pain. His initial ECG shows ST elevation in lead 2, 3, and aVF which of the following is contraindicated for administering thrombolytic therapy:

A. A completely recovered ischemic stroke 2 months ago.
B. A blood pressure of 160/90
C. femoral fracture 6 months ago.
D. Severe chest pain associated with a short run of ventricular tachycardia.

3)64 years old lady with recurrent attack of severe headache diagnose to have migraine headache with aura, she is at increased risk of:

A. large artery ischemia stroke.
B. lacunar stroke.
C. hemorrhagic stroke.
D. cerebral aneurysms

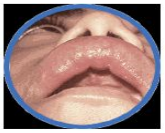
4)When treating patients with acute cerebral infarction. Thrombolytic therapy with intravenous tPA may be considered if the drug can be given within the following time window after the onset of symptoms:

A. 3 hours
B. 5 hours
C. 7 hours
D. 9 hours

SAQs

[taken from doctor's slides]

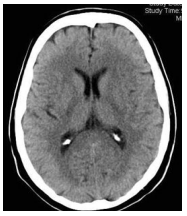
1) A 60 y.o lady with acute stroke few hrs post IV t-PA. She is known with HTN and controlled DM-2.



DX: Orolingual angioedema

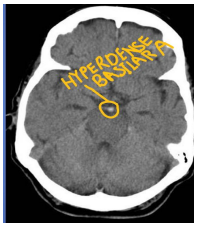
Alteplase is used to treat acute ischemic stroke. However, it has several documented adverse effects, including the development of orolingual angioedema (OA). Although, OA is a rare side-effect, it is thought to be life-threatening and is difficult to treat

2) Sample sample?21 y. o man, a university student presented to ER with Left sided throbbing headache and mild expressive aphasia.Nothing else.NIHSS: 2,PMHx: Migraine.



DX: Acute Left MCA (upper division) ischemic stroke with NORMAL CT brain

3) A 53 y/o male with sudden reduction in LOSS OF CONSCIOUSNESS, jerking in 4 limbs, and difficulty in breathing.Got intubated in ER then CT brain was done. PMHx: smoker, HTN.



DX: Acute Basilar artery stroke

Answer key:

1 (C) | 2 (A) | 3 (A) | 4 (A) |

➢ Extra MCQ's Very helpful. Check Your Understanding Dear. **Neurologist**! Link