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05 06

Pathology

(Cerebrovascular accident)

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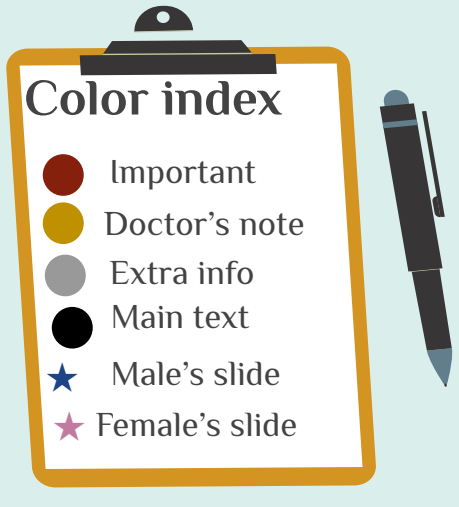
"اللهم لا سهل الا ماجعلته سهلا و انت تجعل الحزن إذا شئت سهلا"



Objective

01 Explain the concepts of brain “Hypoxia”, “Ischemia” and “Infarction”

02 Understand the pathogenesis of thrombotic and embolic stroke and be able to identify clinical risk factors

03 Identify the causes and consequences of subarachnoid and intracerebral hemorrhage

04 Build a list of the different causes that can lead to cerebrovascular accident

Helpful video

Overview

Cerebrovascular diseases

- **Definition** : the broad category of brain disorders caused by pathologic processes involving blood vessels.

- The three main pathogenic mechanisms are:

01 Thrombotic occlusion

02 Embolic occlusion
derived from thrombus

03 Vascular rupture

-All of these three pathogenic mechanisms can lead to stroke

- It is also the most prevalent neurological disorder in terms of both morbidity and mortality.
- Cerebrovascular disease is the third leading cause of death (after heart disease and cancer) in the United States.

Review the following terms

- **Hypoxia**: Deficiency in the amount of oxygen reaching the tissues.
- **Ischemia**: An inadequate blood supply to an organ or part of the body, **Ischemia** causes hypoxia which causes infraction
- **Infarction**: Obstruction of the blood supply to an organ or region of tissue, causing local death of the tissue.

Robbin's note :The brain is a highly oxygen-dependent tissue that requires a continual supply of glucose and oxygen from the blood. Although it constitutes no more than 2% of body weight, the brain receives 15% of the resting cardiac output and is responsible for 20% of total body oxygen consumption.

The brain may be deprived of oxygen by several mechanisms :	
Functional hypoxia	**Ischemia, either transient or permanent**
1-low partial pressure of oxygen. (e.g. High Altitude) 2-Impaired oxygen-carrying capacity. (e.g. severe anemia, carbon monoxide poisoning) 3-Inhibition of oxygen use by tissue. (e.g. cyanide poisoning)	1-reduction in perfusion pressure, as in hypotension, cardiogenic shock & local hemorrhage due to rupture of an aneurysm 2-Vascular obstruction. 3-Both

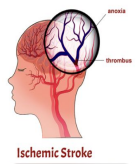
Stroke

❖ ⋆ **Stroke:** It is the clinical term for a disease with **acute** onset of a neurologic deficit as the result of **vascular lesions**, either hemorrhage or loss of blood supply

Etiology :

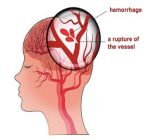
❖ **Thrombosis & embolism** have similar consequences for the brain , **loss of oxygen** & metabolic substrates, resulting in **infarction or ischemic injury** of regions supplied by the affected vessel .

❖ Infarction is complete loss of perfusion , hypoxemia (hypovolemic shock) or hypoglycemia



Ischemic Stroke

❖ **Hemorrhage** accompanies **rupture** of vessels and leads to direct tissue damage as well as **secondary ischemic injury** (**aneurysm** or trauma)

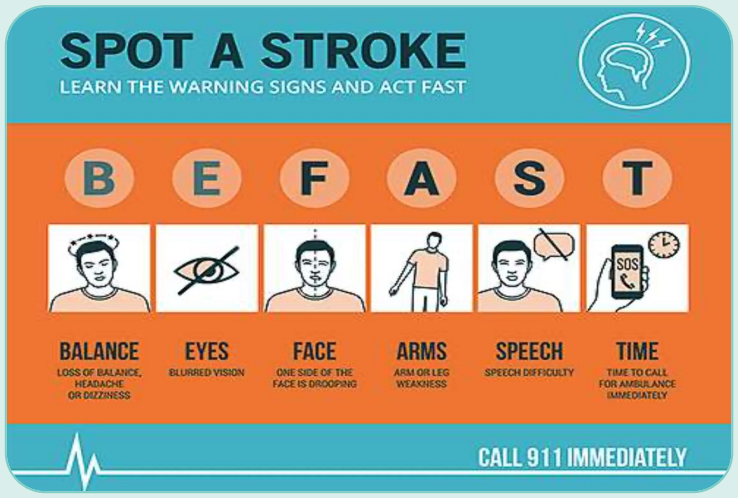


Hemorrhagic Stroke

Clinical Presentation

❖ Strokes can be asymptomatic or painless, however they may also present with symptoms depending on which part of the brain is injured, and how severely it is injured.

❖ It is very important to recognize the **warning signs (**BE FAST**)** of a stroke and to get immediate medical attention if they occur.

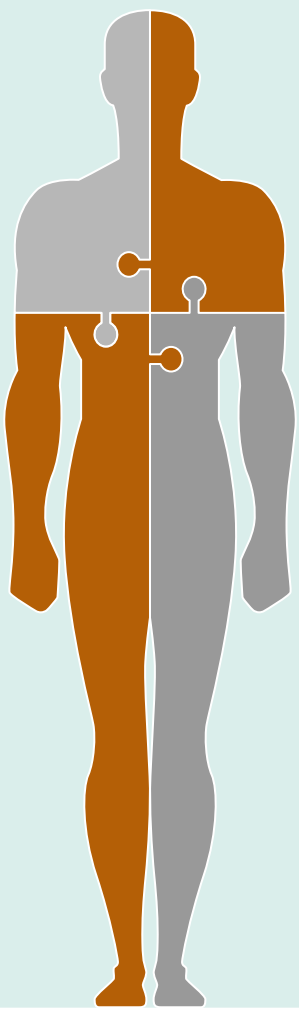


❖ If the brain damage sustained has been slight, there is usually complete recovery , but most survivors of stroke require extensive rehabilitation.

Sometimes people have a **headache** but stroke can also be **completely painless**

A stroke involving the **base of the brain** can affect **balance, vision, swallowing, breathing and even unconsciousness**

The most common is weakness or **paralysis of one side of the body (Hemiplegia)** with partial or complete loss of voluntary movement or sensation in a leg or arm



There can be **speech problems** and **weak face muscles**, causing **drooling** (uncontrolled salivation)

⋆**Sudden**
-**Numbness or tingling** is very common

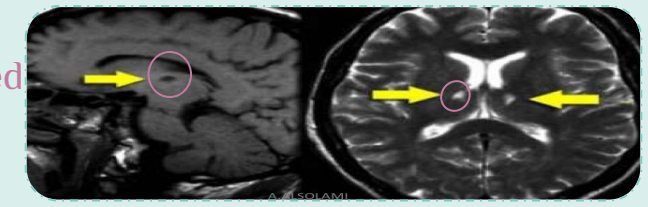
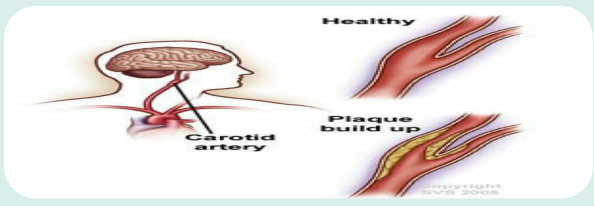
In cases of severe brain damage there may be **deep coma**, **paralysis of one side of the body**, and loss of speech, followed by death or permanent neurological disturbances after recovery

Risk Factors

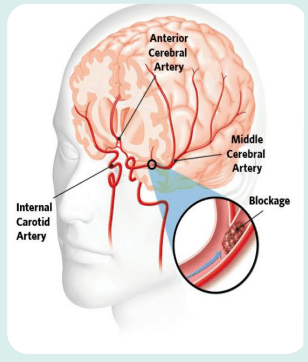
1. Hypertension
2. Atherosclerosis with its risk factors (diabetes)
3. Vasculitis
4. Vascular malformations
5. Venous thrombosis commonest locations (leg, pelvic vessels & brain sinuses)
6. Tumors
7. Embolic diseases
8. Amyloid angiopathy (leptomeningeal and cortical vessels) in old people & lead to subdural hematoma
9. Thrombophilia, e.g. Sickle cell anemia or hemophilia
10. Systemic hypoperfusion /Global hypoxia, e.g. shock

Types of ischemic stroke

Thrombotic stroke

Definition	Formation of blood clot (thrombus) inside a blood vessel, obstructing the flow of blood
Overview	-The majority of thrombotic occlusions causing cerebral infarctions are due to **atherosclerosis** -Thrombotic occlusions usually are superimposed on atherosclerotic plaques, accompanied by anterograde extension, fragmentation, and distal embolization. -Thrombotic occlusions causing small infarcts of only a few millimeters, so called "lacunar infarcts", occurs when small penetrating arteries are occluded. 
The most common sites of primary thrombosis	1- The **carotid bifurcation** 2- The origin of the **middle cerebral artery** 3- At either end of the **basilar artery** 

Embolic stroke

Definition	Intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin
Overview	-Embolic infarction are **more common** than thrombotic infarction
The sites of emboli	-The territory of distribution of the **middle cerebral arteries** (branches from the internal carotid arteries) most frequently affected by embolic infarction . -Emboli tend to lodge where vessels branch or at stenotic areas caused by atherosclerosis
Sources of emboli include	1- **Cardiac mural thrombi** (frequent): a.★Myocardial infarct , b. Valvular disease , c. Atrial fibrillation 2- **Paradoxical (unexpected) emboli** :★particularly in children with cardiac anomalies (e.g. patent foramen ovale) 3- **Arteries** :(often **atheromatous plaques** within the **carotid arteries** or the aortic arch) 4- **Emboli associated with cardiac surgery** 5 -**Emboli of other material** (tumor, fat, or air) 

Global cerebral ischemia

Definition

Widespread ischemic/hypoxic injury occurs when there is a **generalized reduction** of cerebral perfusion, ★usually below **systolic** (normally 120 mmHg) pressures of less than 50 mmHg.
Global means ischemia to the whole brain .

Etiology

- ❖ Cardiac arrest
- ❖ Severe hypotension or shock or bradycardia

Clinical outcome

- ❖ **Mild state :** When the insult is **mild** → there may be only a transient postischemic confusional state, with eventual complete recovery
- ❖ **Sever state :** In **severe** global cerebral ischemia, widespread neuronal death, and patients who survive often remain severely impaired neurologically & in :
- ❖ **persistent vegetative state :**

★ - individual who survive in this state often remain severely impaired neurologically and deeply comatose . They breath & may move their eyes but they aren't communicating .

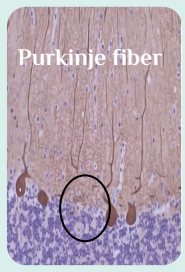
- ❖ **Respirator brain :**

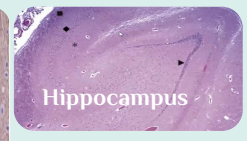
-Other patients meet the clinical criteria for **"brain death"** including evidence of diffuse cortical injury (isoelectric, or "**flat**" EEG) and brain stem damage, including absent pupillary reflexes and respiratory drive.
-When the patients with this irreversible form of injury are maintained on mechanical ventilation, the brain gradually undergoes autolysis, results in the so-called "**respirator brain**".

Overview

- ❖ **Neurons are much more sensitive to hypoxia than other glial cells**
- ❖ The most susceptible to ischemia of short duration are:

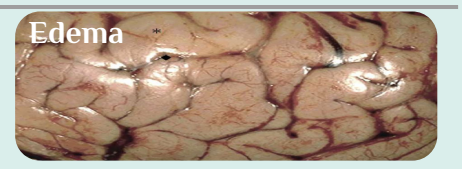
1-**pyramidal cells** of the Sommer sector (CA1) of the **hippocampus**
2-**Purkinje cells** of the cerebellum
3-**pyramidal neurons** in the neocortex





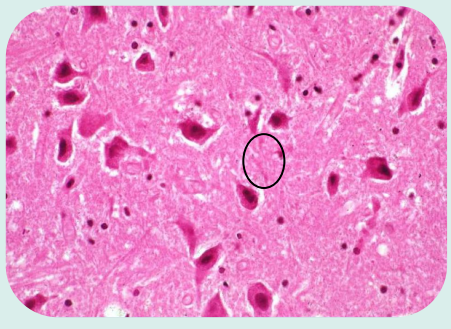
(Gross) Macroscopically pathology

1. The brain is **swollen** (edema), with wide gyri and narrowed sulci.
2. The cut surface shows **poor demarcation** between gray and white matter because of edema .

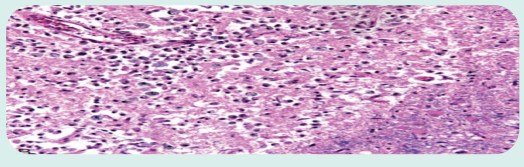


Microscopic Features
We don't call it granulation tissue due to the absence of fibrosis in the brain .

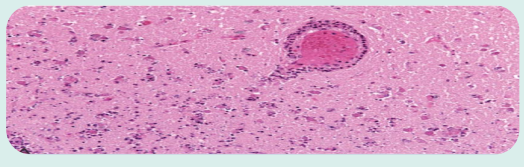
Early changes (12-24 H)	Subacute changes (24 H- 2 W)	Repair (after 2 W)
• **Red neurons :** characterized initially by microvacuolization, cytoplasmic eosinophilia, and later nuclear pyknosis and karyorrhexis • Similar changes occur later in glial cells.	• The reaction to tissue damage begins with the infiltration by **neutrophils**. • Necrosis of tissue, influx of **macrophages**, vascular proliferation and reactive gliosis.	• Removal of all necrotic tissue • Loss of the organized CNS structure • **Gliosis** • No scars, only shrunken areas.



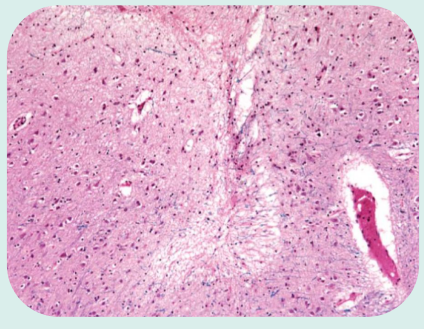
★ Red (angry) Neurons(Early)



★ By day 10, an area of infarction shows the presence of macrophages and surrounding reactive gliosis (Subacute)



★ Infiltration of a cerebral infarction by neutrophils begins at the edges of the lesion where the vascular supply is intact (Subacute)



★Old intracortical infarcts are seen as areas of tissue loss with a modest amount of residual gliosis.

All The pictures was found in both male's and female's slide , But the explanation was found only in female's slid, and Robins

Focal cerebral ischemia

Definition

- ❖ Cerebral arterial occlusion or cerebral hemorrhage lead first to focal ischemia then an infarction in the distribution of the **compromised vessels**. (not caused by hypotension or something affect heart)
- ❖ The size, location and shape of the infarct and the extent of tissue damage may be modified by collateral blood flow.

Collateral blood flow in focal cerebral ischemia

- ❖ The major source of collateral flow is the **circle of Willis** اكثر واحد يحب الفزعات
- ❖ Partial collateralization is also provided over the surface of the brain through **cortical-*leptomeningeal* anastomoses** pia & arachnoid meninges
- ❖ In contrast, there is little if any collateral flow for the deep penetrating vessels supplying structures such as: ماعندهم احد يساعدهم
  - Thalamus
  - Basal ganglia
  - Deep white matter

Infarcts

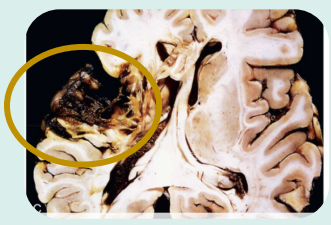
Infarcts can be divided into two broad groups based on their macroscopic and corresponding radiologic appearance:

Dr. Note: it is difficult to make classification of all infarction of the brain

- ❖ **Non hemorrhagic infarcts** : which result from acute vascular **occlusions**.
- ❖ **Hemorrhagic infarcts** : which result from **reperfusion** of ischemic tissue, either through collaterals or after dissolution of emboli, and often produce multiple, sometimes confluent petechial hemorrhages.
- ❖ usually manifest as multiple, sometimes confluent, petechial hemorrhages The microscopic picture and evolution of hemorrhagic infarction parallel those of ischemic infarction

(Macroscopically) Gross pathology Of Non - hemorrhagic infarct:

First 6 hours	the tissue is the tissue unchanged in appearance
By 48 hours	the tissue becomes **pales** (caused by occlusion), **soft** and **swollen**, ★ and the corticomedullary junction becomes **indistinct**
2-10 days	the tissue becomes **gelatinous**, **friable** and the boundary between normal and abnormal tissue **becomes more distinct as edema resolves** in the adjacent viable tissue.
Day 10- week 3	The **tissue liquifies** leaving a fluid-filled cavity (★ lined by dark gray tissue) which gradually expands as dead tissue is removed. مرحلة الذوبان



★ Old cystic infarct shows destruction of cortex and surrounding gliosis . Middle cerebral artery is blocked (loss of tissue, No scar or fibrosis)

The pictures was found in both male's and female's slide , But the explanation was found only in female's slid, and Robins

Focal cerebral ischemia

Microscopic Features for Non - hemorrhagic infarct:

❖ **After the first 12 hours :**

-Ischemic neuronal change **(red neurons)** and **edema**
-Endothelial & glial cells swell & myelinated fibers begin to disintegrate.
-**Loss** of the usual characteristics of **white and gray matter structures**
-**During the first several days** neutrophils infiltrate the area of injury.

❖ **Until 48 hours :**

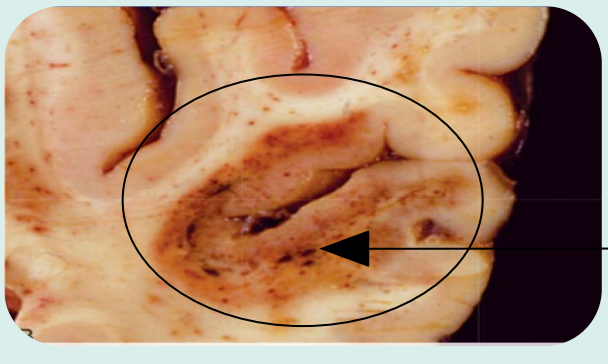
-There is some neutrophil emigration followed by **mononuclear phagocytic cells** in the ensuing 2 to 3 weeks.
-**Macrophages** containing myelin breakdown products or blood may persist in the lesion for months to years.
-As the process of phagocytosis and liquefaction proceeds, astrocytes at the edges of the lesion progressively enlarge, divide, and develop a prominent network of protoplasmic extensions. Gliosis at the edge of the stroke not in the middle

❖ **After several months :**

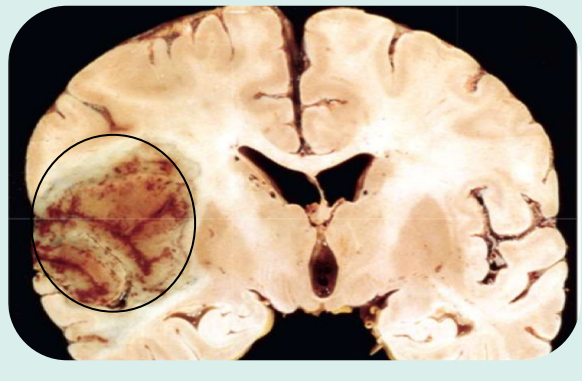
The striking astrocytic nuclear and cytoplasmic enlargement **recedes/disappears**.
-In the wall of the cavity, astrocyte processes form a dense feltwork of glial fibers admixed with new capillaries and a few perivascular connective tissue fibers.
-In the cerebral cortex the cavity is delimited from the meninges and subarachnoid space by a gliotic layer of tissue, derived from the molecular layer of the cortex.
-The pia and arachnoid are not affected and do not contribute to the healing process

Microscopic Features for Hemorrhagic Infarcts

-Hemorrhagic infarcts usually manifest as multiple, sometimes confluent petechial hemorrhages.
-The microscopic picture and evolution of hemorrhagic infarction parallel those of ischemic infarction, with the **addition of blood extravasation & resorption**.
-In individuals with coagulopathies or receiving anti-coagulants, hemorrhagic infarcts may be associated with **extensive intracerebral hematomas**.



★ An infarct with punctate hemorrhages, consistent with ischemia-reperfusion injury, is present in the temporal lobe. (Leptomeningeal vessels)

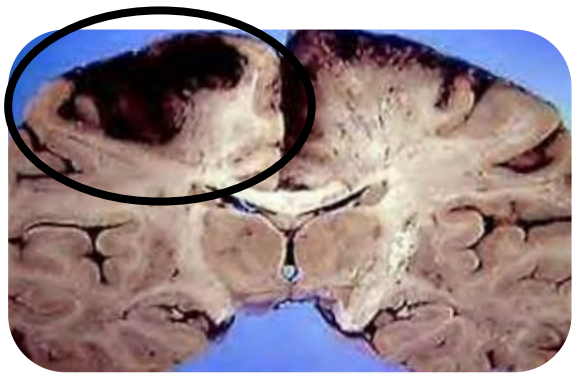


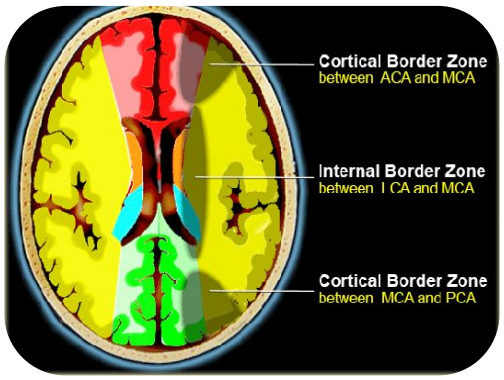
★ A section of the brain showing a large discolored focally hemorrhagic region in the left middle cerebral artery distribution (hemorrhagic or red infarction).

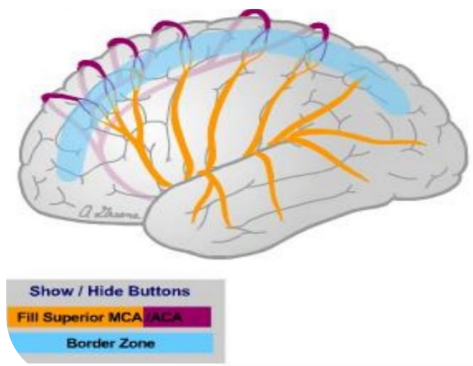
The pictures was found in both male's and female's slide ,
But the explanation was found only in female's slid, and Robins

Border zone (watershed) infarcts:

- It is not a disease but a term to describe to an infarction.
- **Wedge-shaped areas** of infarction that occur in those regions of the brain and spinal cord that lie at **the most distal** fields of arterial perfusion
- It is usually seen after hypotensive episodes
- In the cerebral hemispheres, the border zone between the **anterior and the middle cerebral artery** distributions is at greatest risk
- Damage to this region produces a band of necrosis over the cerebral convexity a few centimeters lateral to the interhemispheric fissure (في نص المخ)

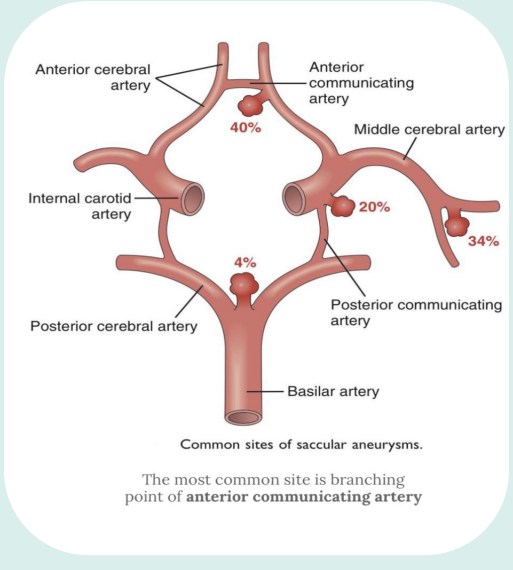






Intracerebral Hemorrhage

- Hemorrhages within the brain tissue (intracerebral) can occur secondary to :
  - **Hypertension (most common reason)**
  - Tumor
  - ⋆ CAA (Cerebral amyloid angioplasty)
  - Arteriovenous/vascular malformation
  - ⋆ An intraparenchymal tumor (can produce abnormal vessels)
  - vascular wall injury like vasculitis (Aneurysm)



Common sites of saccular aneurysms.

The most common site is branching point of **anterior communicating artery**

- Hemorrhages associated with the dura (in either subdural or epidural spaces) make up a pattern associated with trauma
- brain hemorrhage accounts for roughly 15% of deaths among individuals with chronic hypertension. Intracerebral hemorrhage can be clinically devastating when it affects large portions of the brain or extends into the ventricular system

★ Dr. Note: This is important Memorize the distribution with percentage

we will talk about this picture in the next slides in details

Subarachnoid Hemorrhage

❖ Bleeding into the subarachnoid space.

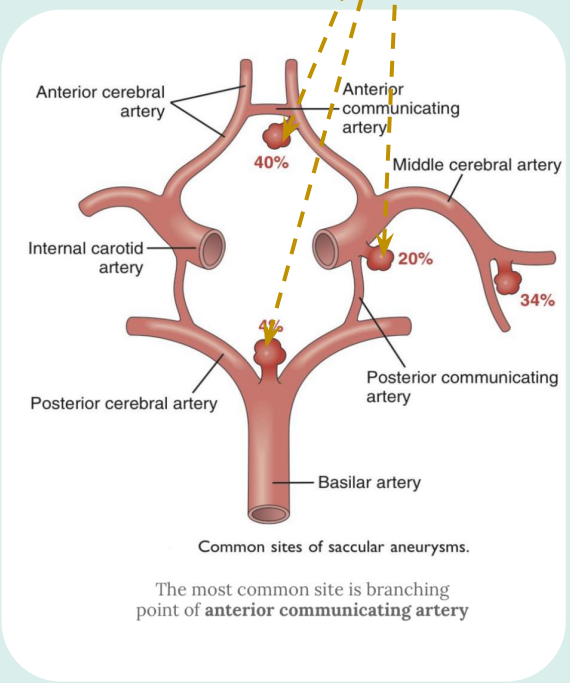
Causes

❖ **Rupture of a saccular (berry) aneurysm** (the most common frequent clinically significant cause)
if we know that he has subarachnoid hemorrhage we could say that he has berry aneurysm
❖ Vascular malformation
❖ Trauma
❖ Hematologic disturbances E.x: Hemophilia
❖ Tumors
❖ Rupture of an intracerebral hemorrhage into the ventricular system

General info.

❖ Rupture can occur at any time, but in about one-third (1\3) of cases it is associated with **acute increases in intracranial pressure**, (⋆such as with straining at stool or sexual orgasm) .
❖ Increased intracranial pressure is a medical term that refers to growing pressure inside a person's skull
❖ Sneezing and (constipation) can lead to Increase intracranial pressure

Berry aneurysm



Common sites of saccular aneurysms.

The most common site is branching point of **anterior communicating artery**

"This is very important subject especially in young people it's called **Berry aneurysm:**
It is an aneurysm around circle of willis , the bleeding will happen in the subarachnoid layer , it's congenital (but not from birth) It takes time to grow,
can lead to rupture and severe hemorrhage

MEMORIZE THE DISTRIBUTION.

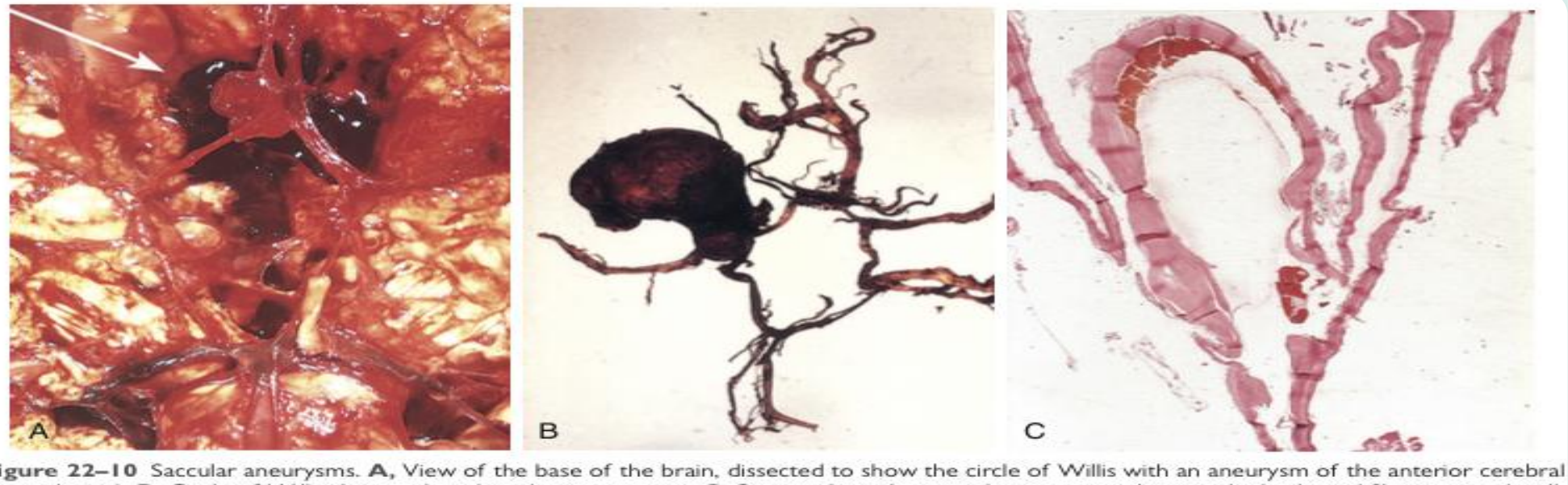


Figure 22–10 Saccular aneurysms. **A,** View of the base of the brain, dissected to show the circle of Willis with an aneurysm of the anterior cerebral artery *(arrow)*. **B,** Circle of Willis dissected to show large aneurysm. **C,** Section through a saccular aneurysm showing the hyalinized fibrous vessel wall. Hematoxylin-eosin stain.

Subarachnoid Hemorrhage

- Symptoms: Patient stricken with sudden, **excruciating headache** (very painful classically described as "**the worst headache I've ever had")** and **rapidly lose consciousness.**
- Location: About 90% of **saccular aneurysms** occur in the **anterior circulation** near major arterial branch points.
- Multiple aneurysms exist in 20% to 30% of cases.

-They are sometimes referred to as congenital but don't present at birth but develop over time because of underlying defects in the vessel media

- Diseases at risk of aneurysm: Ehlers-Danlos syndrome 2-Autosomal dominant polycystic kidney disease Said by Female's doctor
- The probability of **aneurysm rupture** increases with the **size of the lesion** such that , aneurysms greater than 10 mm have a roughly 50% risk of bleeding per year.
- In the early period after a subarachnoid hemorrhage, there is a risk of additional ischemic injury from **vasospasm** involving other vessels . The body reacts to hemorrhage by vasospasm (the constriction of arteries) so this contributes to an additional ischemic injury.

Prognosis

- The prognosis worsens with each episode of bleeding
- Between 25% and 50% of individuals die with the first rupture, although those who survive typically improve and recover consciousness in minutes
- Recurring bleeding is common in survivors; it is currently not possible to predict which individuals will have recurrences of bleeding and we don't know the risk factor of each individual .

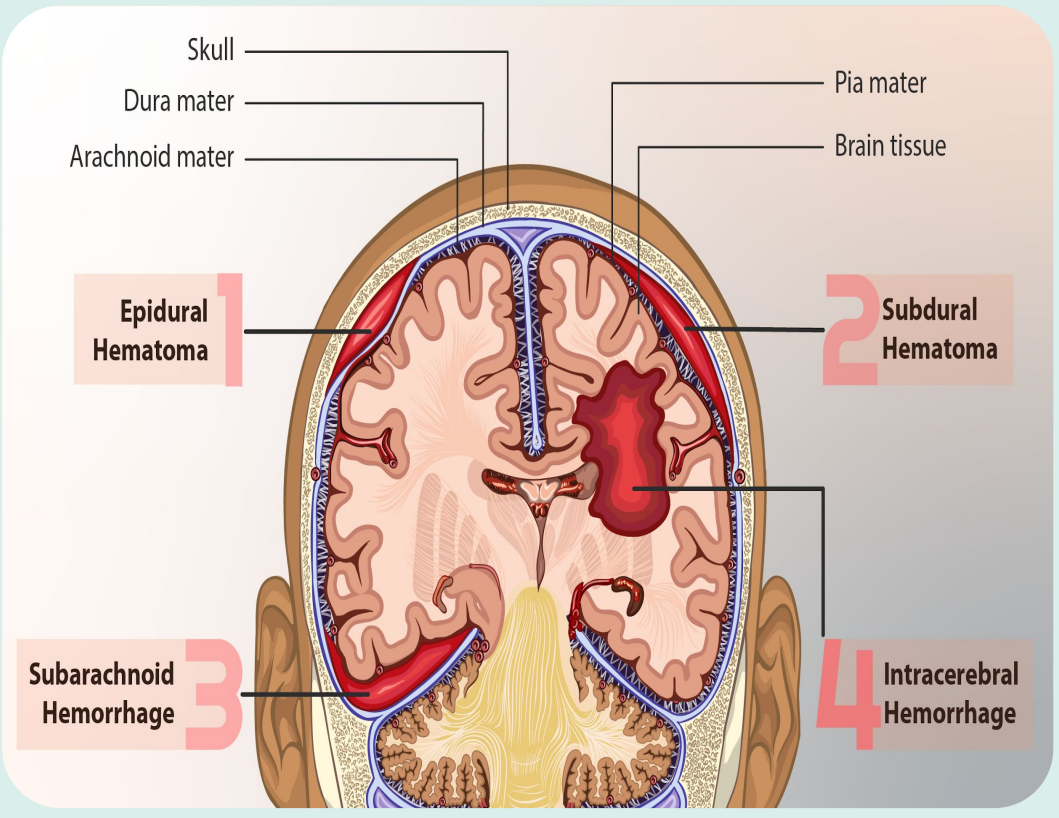
Healing phase of subarachnoid hemorrhage

01 repairing phase after hemorrhage , done by Meningeal fibrosis and scarring occur

02 Sometimes leading to **obstruction of CSF flow**

03 As well as interruption of the normal pathways of CSF resorption
Lead to secondary hydrocephalus

Type of brain hemorrhage



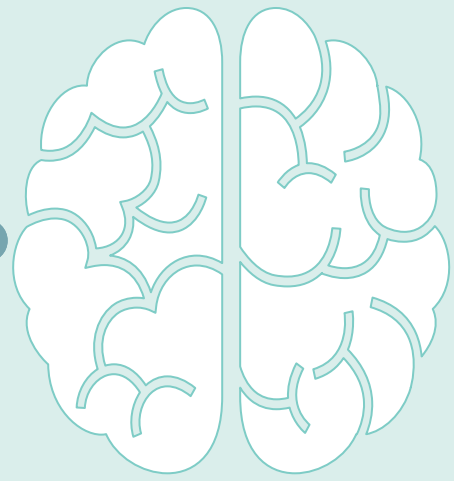
Hypertensive Cerebrovascular Disease

- Hypertension affects the **deep penetrating arteries** and arterioles that supply the **basal ganglia and hemispheric white matter** and the brain stem.
- Hypertension causes several changes, including **hyaline** (pink material) **arteriolar sclerosis** in arterioles → weaker than normal vessels and are more vulnerable to rupture.
- In some instances, chronic hypertension is associated with the development of **minute aneurysms** in vessels that are less than 300 µm in diameter → **Charcot-Bouchard microaneurysms** , which can **rupture**.
- Charcot-Bouchard microaneurysms are microaneurysms of **retinal** blood vessels & lead to loss of vision.
- Hyaline arteriolar sclerosis and Charcot-Bouchard microaneurysms are associated with hypertension .

The most important effects of hypertension on the brain include:

Massive hypertensive intracerebral hemorrhage (most important)

Lacunar infarcts



Hypertensive encephalopathy

Slit hemorrhage

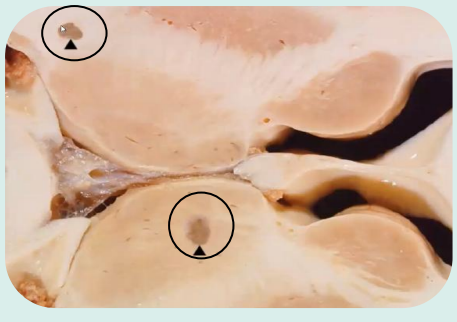
Lacunar infarcts

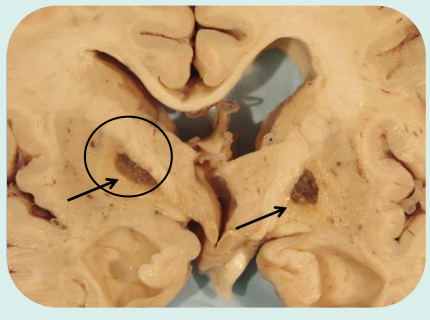
- Small **cavitary** infarcts

Location of lacunar infarct :

- Most commonly in **deep gray matter** (basal ganglia and thalamus), internal capsule, deep white matter, and pons
- Consist of cavities of **tissue loss** with, (repair stage) **scattered lipid-laden macrophages** and surrounding **gliosis** due to ischemia
- Depending on their location in the CNS, lacunes can either be clinically silent or cause significant neurologic impairment
- Caused by occlusion of a single penetrating branch of a large cerebral artery

Lacunar infarction can associated with chronic hypertension patient





Acute hypertensive encephalopathy

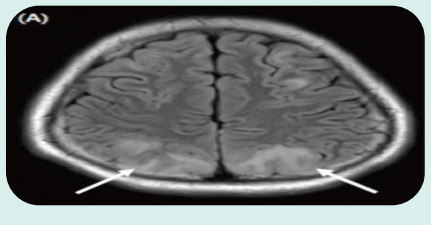
A clinicopathologic syndrome:

- Sudden stained Increase in diastolic blood pressure to greater than 130 mm Hg (very high):

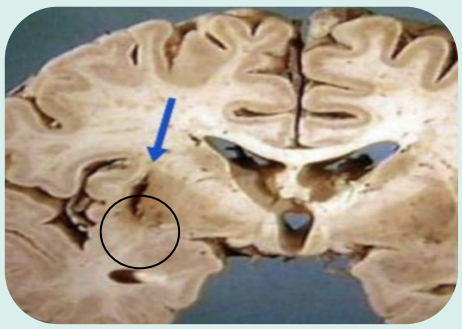
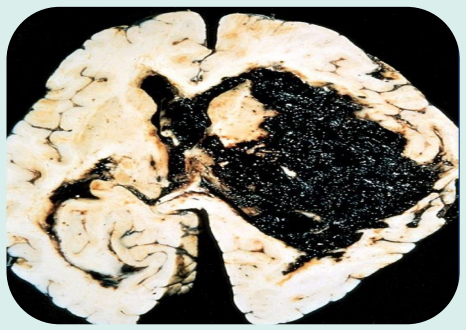
-Clinically: diffuse cerebral dysfunction, including headaches, confusion, vomiting, and convulsions, sometimes leading to coma.
- Does not usually remit spontaneously.

- **Grossly** : May be associated with an **edematous brain**, with or without **transtentorial** or tonsillar **herniation**.
- **Microscopically**: **Petechiae** (small hemorrhages) and **fibrinoid necrosis** of arterioles in the gray and white matter may be seen microscopically
- Most often associated with sudden sustained increase in diastolic blood pressure to greater than 130 mmHg

*the image is EXTRA



Hypertensive Cerebrovascular Disease

Slit hemorrhage	Massive hypertensive intracerebral hemorrhage (Most important)
❖ **Rupture** of the small-caliber penetrating vessels and the development of **small** hemorrhages ❖ in time, these hemorrhages resorbed , leaving behind a **slit like (needle like)** , cavity surrounded by brownish discoloration	❖ Massive hypertensive hemorrhage rupturing into the lateral ventricle. *Discussed earlier
	 Intracerebral & subarachnoid hemorrhage

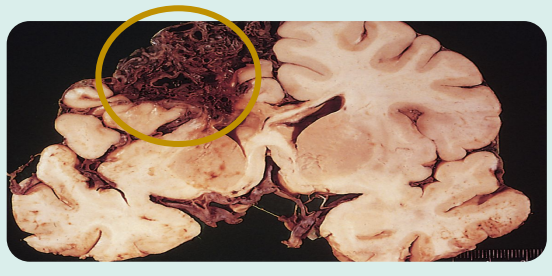
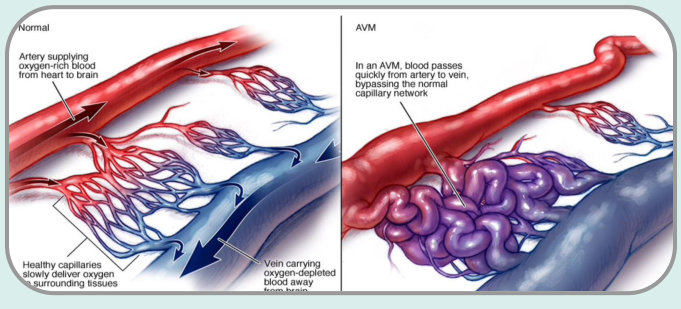
Vascular diseases

Vasculitis Inflammation		
Definition	Infectious arteritis of small and large vessels	
Etiology	Infectious	❖ Previously in association with syphilis and tuberculosis ❖ Now more commonly occurs in the setting of **immunosuppression** ex . (HIV patient , patient after transplantation) and opportunistic infection (such as **toxoplasmosis**, **aspergillosis**, and CMV **encephalitis**)
	Non Infectious	❖ Systemic forms of vasculitis, such as **polyarteritis nodosa**, may involve cerebral vessels and cause single or multiple infarcts throughout the brain
		❖ **Primary angiitis** of the CNS: ➢ Vasculitis of the brain blood vessels ➢ An inflammatory disorder that involves multiple small to medium-sized parenchymal and subarachnoid vessels – Affected individuals manifest a diffuse encephalopathic clinical picture, often with cognitive dysfunction ➢ Improvement occurs with steroid and immunosuppressive treatment

Vascular malformation

- They are classified into four principal types based on the nature of the abnormal vessels:
  a. Arteriovenous malformations (AVMs)
  b. Cavernous malformations (cerebellum, pons, subcortical)
  c. Capillary telangiectasias (pons)
  d. Venous angiomas (varices)

Arteriovenous Malformations

Definition	❖ arteriovenous malformation (AVM) is an abnormal tangle of blood vessels connecting arteries and veins, which disrupts normal blood flow and oxygen circulation
Overview	❖ AVM is the most common type of vascular malformations
Epidemiology	❖ affect males twice as frequently as females and most commonly manifest between 10 and 30 years of age with seizures, an intracerebral hemorrhage, or a subarachnoid hemorrhage. ❖ In the newborn period, large AVMs may lead to high-output congestive heart failure because of blood shunting from arteries to veins.
Location	❖ AVMs may involve subarachnoid vessels extending into brain parenchyma or occur exclusively within the brain ❖ The risk for bleeding makes AVM the most dangerous type of vascular malformation.
	❖ Multiple AVMs can be seen in the setting of hereditary hemorrhagic telangiectasia, an autosomal dominant condition often associated with mutations affecting the TGFβ pathway
	 Abnormal brain tissue loss with abnormal structure that can cause stroke & death 

Homework

Q1 . Define: Transient ischemic attack ?

Regional Ischemia that results in neurological deficits with symptoms lasting less than 24 hours. Is like a stroke, producing similar symptoms, but usually lasting only a few minutes and causing no permanent damage

Q2 . What are the risk factors of stroke ?

QUIZ!

MCQs

01 | Which of the following is not a common site of primary thrombosis in the brain:			
A) The **carotid bifurcation**	B)The **anterior cerebral artery**	C) The origin of the **middle cerebral artery**	D) At either end of the **basilar artery**
02 | Which one of the following functional hypoxia is due inhibition of oxygen use by tissue?			
A) High altitude	B) Severe Anemia	C) Cyanide poisoning	D) Carbon monoxide poisoning
03 |Early microscopic feature of global cerebral ischemia ?			
A)Infiltration of neutrophils and macrophage	B)Red neurons	C)Gliosis	D) both A , C
04 |What is the major source of collateral blood flow in focal cerebral ischemia ?			
A)cortical-leptomeningeal anastomoses	B)Basal artery	C)Circle of willis	D)middle cerebral artery
05 | which of the following hypertensive cerebrovascular diseases consists of cavities of tissue loss with scattered lipid-laden macrophages?			
A) Slit hemorrhage	B) Hypertensive encephalopathy	C) Intracerebral hemorrhage	D) Lacunar infarcts
06 | Autopsy was done to a patient with history of diffuse cerebral dysfunction, the histopathology report indicated a petechiae and fibrinoid necrosis in some arterioles in the gray and white matter, what is the diagnosis?			
A) Intracerebral hemorrhage	B) Lacunar infarcts	C) acute hypertensive encephalopathy	D) Slit hemorrhage

MCQs Answer key	01	02	03	04	05	06
	B	C	B	C	D	C

QUIZ!

MCQs

07 | Charcot-Bouchard Aneurysms are associated with :			
A) Chronic hypertension	B)Slit hemorrhage	C) Vasculitis	D) Lacunar Infarcts
08 | The most important effects of hypertension on the brain			
A) Massive hypertensive intracerebral hemorrhage	B) Lacunar infarcts	C) Hypertensive encephalopathy	D) Slit hemorrhage
09 | Which of the following triggers a Subarachnoid Hemorrhage with the highest clinical significance:			
A)Hematologic disturbance	B)Tumors	C)Hypertension	D) Rupture of a saccular aneurysm
10 | Regarding subacute changes of GCI, which of the following is not present in histopathological slide examination:			
A)Neutrophils infiltration	B)Reactive Gliosis	C)Red neurons	D)Scaring formation
11 | The watershed infarct happens in the cerebral hemisphere in:			
A) Between the anterior and the middle cerebral artery	B) Between the lateral and middle cerebral arteries	C) At the basal ganglia	D) At the occipital lobe only
12| In the healing phase of subarachnoid hemorrhage, occur:			
A) Scarring	B) Necrosis	C) Inflammation	D) Slit hemorrhage

MCQs Answer key	07	08	09	10	11	12
	A	A	D	D	A	A

"اللهم علمنا ماينفعنا ، وانفعنا بما علمتنا وزدنا علما يارب العالمين"

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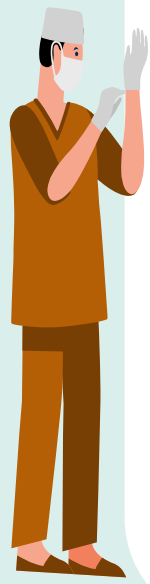
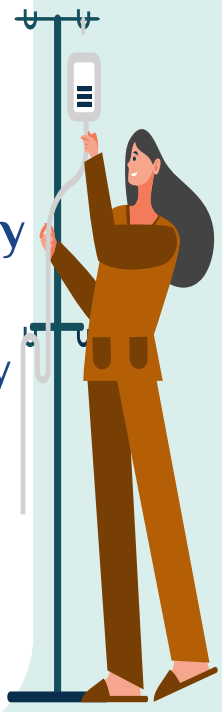
- Mona Alabdely
- Sara Alharbi

Any future corrections will be in the editing file , Click





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- Organizer
- Member
- Note taker
- Reviser

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