Stroke & stroke subtypes

BY ADEL HUSSAIN ALSHAMMARI
C11

WHAT IS A STROKE?

- A stroke is a life-threatening condition that happens when part of your brain doesn't have enough blood flow. This most commonly happens because of a blocked artery or bleeding in your brain.

- Strokes are a life-threatening emergency, and immediate medical attention is critical to prevent permanent damage or death.

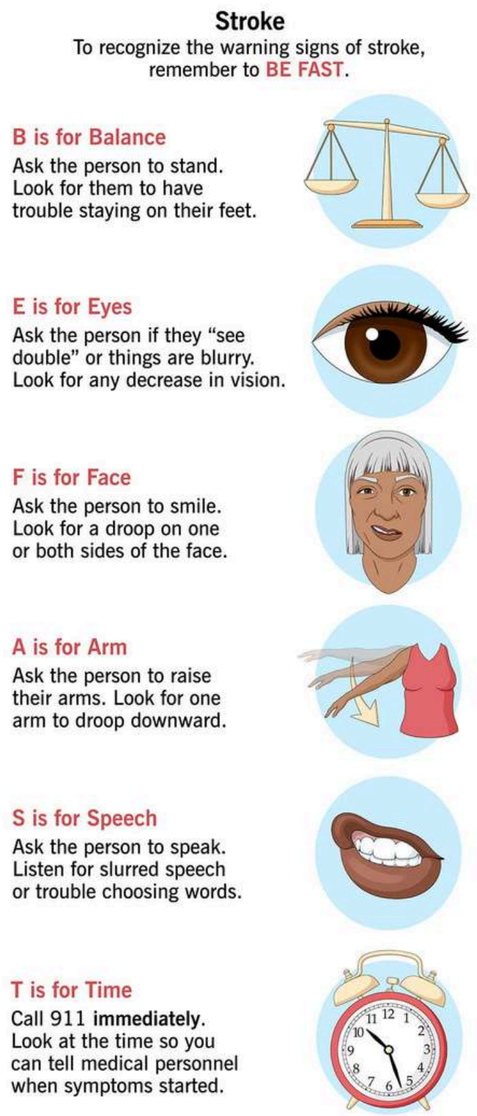
- Worldwide, strokes rank second among the top causes of death. In the United States, stroke is the fifth cause of death. Strokes are also a leading cause of disability worldwide.

TO RECOGNIZE THE WARNING SIGNS OF A STROKE, REMEMBER TO BE FAST:

- B. Be watchful for a sudden loss of balance.

- E. Look out for sudden loss of vision in one or both eyes. Are they experiencing double vision?

- F. Ask the person to smile. Look for a droop on one or both sides of their face, which is a sign of muscle weakness or paralysis.



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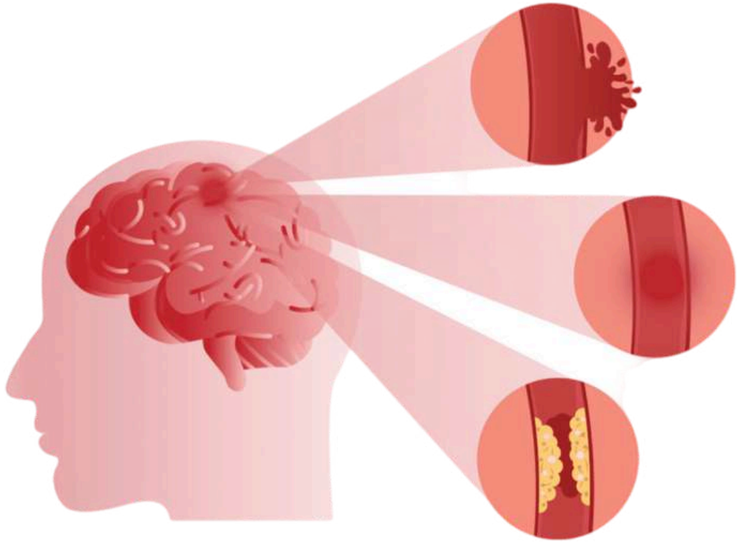
• A. A person having a stroke often has muscle weakness on one side. Ask them to raise their arms. If they have one-sided weakness (and didn't have it before), one arm will stay higher while the other will sag and drop downward.

• S. Strokes often cause a person to lose their ability to speak. They might slur their speech or have trouble choosing the right words.

• T. Time is critical, so don't wait to get help!

WHAT ARE THE TYPES OF STROKES?

- Ischemic stroke
- Hemorrhagic stroke
- Transient ischemic attack (mini-stroke)
- Brain stem stroke
- Cryptogenic stroke (stroke of unknown cause)



ISCHEMIC STROKE

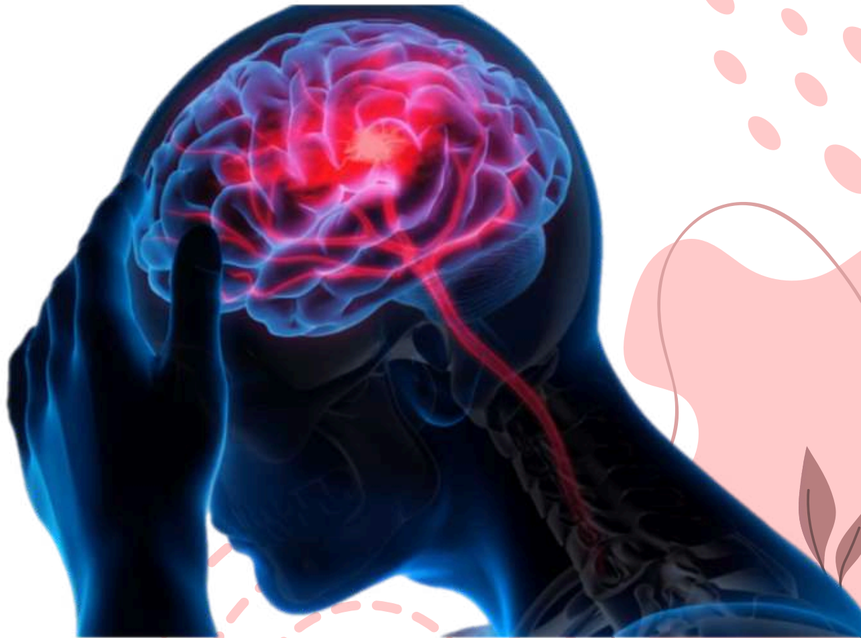
• An ischemic stroke happens when a blood vessel supplying blood to your brain gets blocked by a blood clot. Most strokes are this type.

• A fatty substance called plaque collects in your arteries and narrows them. This process is called atherosclerosis, and it slows the flow of blood. Plaque breaks off and lots of cells come to the rescue causing a clot and blocking blood flow through the artery.

• Besides atherosclerosis, other things that can raise your chances of getting an ischemic stroke are:

• Atrial fibrillation • Heart attack • Problem with your heart's valves • Injury to blood vessels in your neck • Blood clotting problems

• In general, the more severe an ischemic stroke is, the worse the damage. When brain damage is more severe, it's more likely that you'll lose certain abilities, at least temporarily. The faster you get medical attention for stroke symptoms, the better your chances that these effects are temporary or less severe.



THERE ARE TWO MAIN TYPES OF ISCHEMIC STROKE:

- **Thrombotic strokes:**

They're caused by a blood clot that forms in an artery that supplies blood to your brain, also called an atherosclerotic stroke.



atherosclerosis

- **Embolic strokes:**

They happen when a clot forms somewhere else in your body and travels through the blood vessels to your brain. It gets stuck there and stops the flow of your blood. Atrial fibrillation increases the risk of clots forming in the heart that can then travels to the brain.

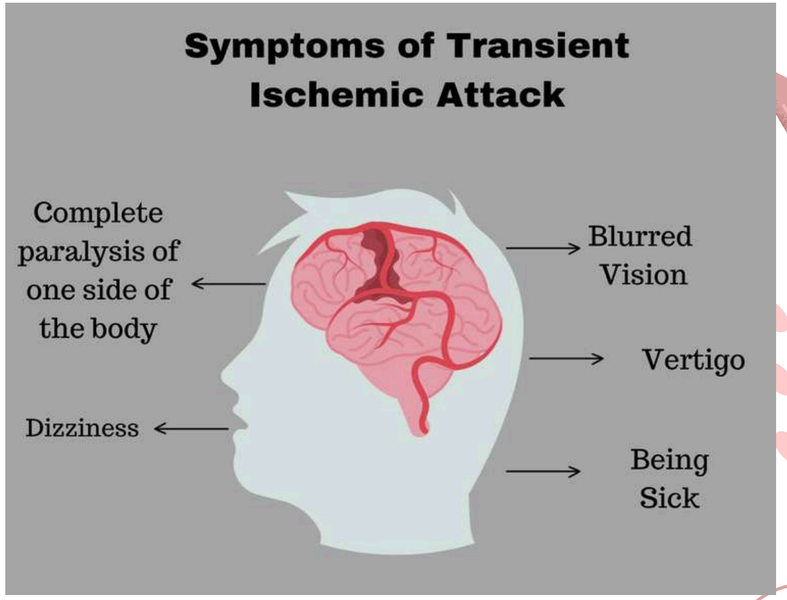


ischemic

TRANSIENT ISCHEMIC ATTACK OR MINI-STROKE

• Ischemic strokes also include something called a "mini-stroke" or a TIA (transient ischemic attack). This is a temporary blockage in blood flow to your brain. The symptoms usually last for just a few minutes or may go away in 24 hours.

• Causes and risk factors of TIA are the same as in an ischemic stroke. A TIA can sometimes be a warning sign that you'll have an ischemic stroke soon.

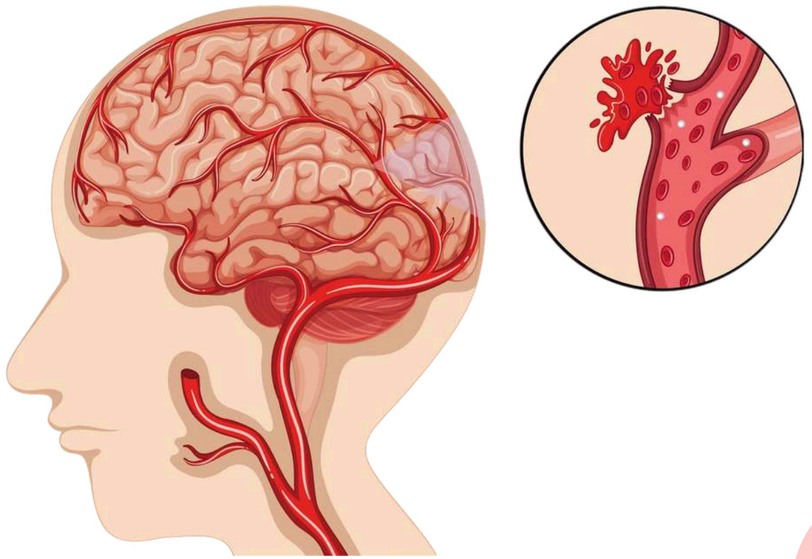


HEMORRHAGIC STROKE

• These happen when bleeding in your brain damages nearby cells.

• These strokes usually cause much worse symptoms, especially when bleeding is more severe. The symptoms of hemorrhagic stroke tend to get worse quickly. People with hemorrhagic strokes tend to cause severe headaches, seizures and coma.

• There are two types of hemorrhagic stroke. Which kind you have is based on where the bleeding happens.



THE TWO TYPES :

• Bleeding inside the brain (intracerebral). This happens when a blood vessel inside the brain tears or breaks open, causing bleeding that puts pressure on the surrounding brain tissue.

• Bleeding into the subarachnoid space (the space between the brain and its outer covering) . The arachnoid membrane, a thin layer of tissue with a spiderweb-like pattern on it, surrounds your brain. The space between it and your brain is the subarachnoid space (“sub” means “under”). Damage to blood vessels that pass through the arachnoid membrane can cause a subarachnoid hemorrhage, which is bleeding into the subarachnoid space, putting pressure on the brain tissue underneath.

• Hemorrhagic stroke symptoms usually increase gradually over minutes or a few hours, although a subarachnoid hemorrhage may come on suddenly. You might notice:

• Intense headache 

some people describe it as the worst headache they've ever had

• Confusion 

• Nausea or vomiting 

• Sensitivity to light 

• Problems with vision

• Passing out

BRAINSTEM STROKE

• This type happens in your brain stem. It can affect both sides of the body.

If this happens, you're left in a "locked in" state where you're unable to speak or move below the neck.

Stroke Risk Factors

By Layan Ali C11

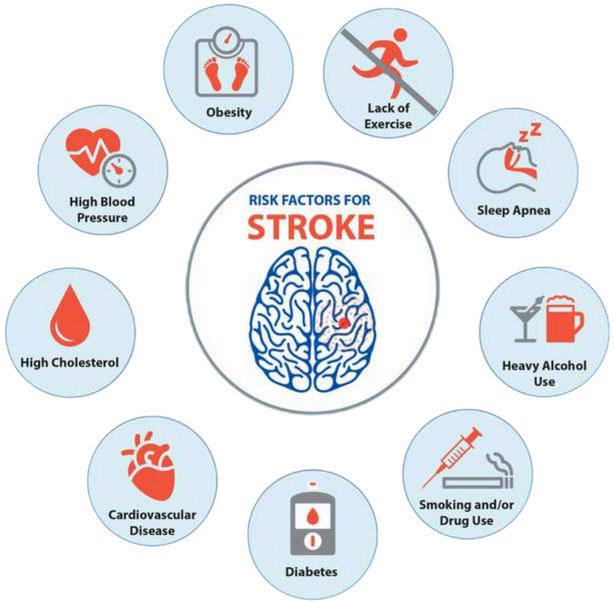
Modifiable Risk Factors:

These factors can be managed or treated to reduce stroke risk.

- **Hypertension (High Blood Pressure):** The most important modifiable risk factor for both ischemic and hemorrhagic strokes.
- **Diabetes Mellitus:** Increases stroke risk by 1.5-2 times, and effective glucose control can reduce the risk.
- **Hyperlipidemia (High Cholesterol):** Elevated levels of LDL cholesterol contribute to atherosclerosis, increasing stroke risk.
- **Smoking:** Nearly doubles stroke risk by accelerating atherosclerosis and increasing blood clotting.

Modifiable Risk Factors:

- **Atrial Fibrillation:** This irregular heart rhythm increases the risk of stroke by causing blood clots to form, which can travel to the brain.
- **Obesity:** Associated with other stroke risk factors such as hypertension, diabetes, and hyperlipidemia.
- **Physical Inactivity:** Sedentary lifestyle increases stroke risk, while regular exercise reduces it.
- **Diet:** A diet high in salt, saturated fats, and low in fruits and vegetables increases stroke risk.
- **Alcohol Consumption:** Heavy drinking increases stroke risk.



Non-Modifiable Risk Factors:

These factors cannot be changed but are important for identifying individuals at higher risk.

- **Age**: Stroke risk increases with age, particularly after age 55.
- **Gender**: Males have a higher risk of stroke, but strokes are often more severe in women.
- **Family History**: A family history of stroke increases an individual's risk, suggesting a genetic predisposition.
- **Race/Ethnicity**: African Americans, Hispanics, and South Asians have a higher stroke risk compared to Caucasians.

Other Contributing Factors:

- **Previous Stroke or Transient Ischemic Attack (TIA):** Having a history of stroke or TIA greatly increases the risk of subsequent strokes.
- **Cardiovascular Diseases:** Conditions like coronary artery disease and heart failure increase stroke risk.
- **Oral Contraceptives and Hormone Replacement Therapy:** Increase the risk, especially in women who smoke or have other risk factors.
- **Drug Use:** Use of illicit drugs like cocaine and amphetamines can significantly increase stroke risk.

ETIOLOGIES OF ISCHEMIC AND HEMORRHAGIC STROKE

By Redwan

overview

Vascular mechanisms causing stroke may be classified as:

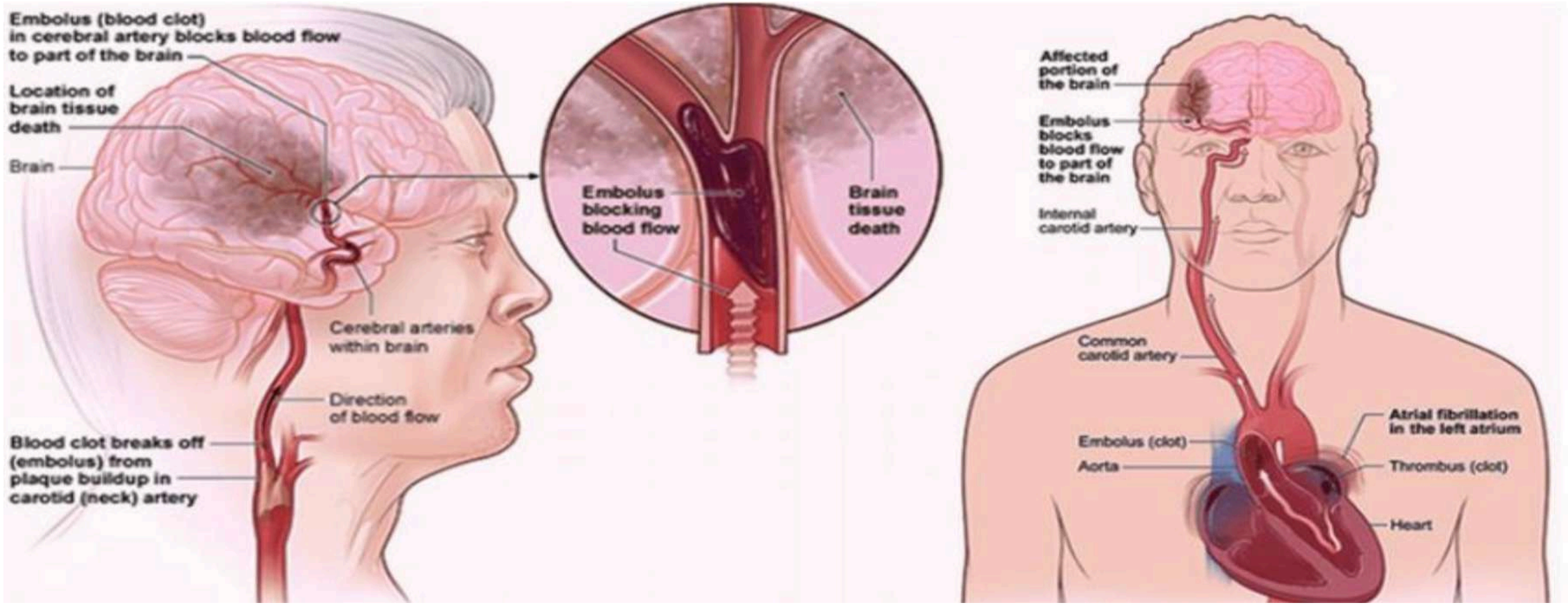
- infarction (embolic or thrombotic)
- hemorrhage.

etiology and pathogenesis:

•Cardiac Emboli .
•Atrial Fibrillation.
• Left ventricular thrombus.
• CHF with low ejection fraction

•This increases stasis of blood and risk of clots forming.
•Could be tricuspid in intravenous drug users
•Fibrinous material on mitral or aortic valves from rheumatic heart disease
•Mechanical heart valve (highly thrombogenic).

CARDIAC EMBOLI



"A **cardiac embolus** is a blood clot or debris formed in the heart that travels through the bloodstream and lodges in a distant artery, such as the carotid artery. When it obstructs blood flow to the brain, it can cause an ischemic stroke by depriving brain tissue of oxygen and nutrients."

ISCHEMIC STROKES

Ischemic strokes occur due to a blockage or reduced blood flow to the brain. The primary etiologies include:

1. Thrombotic Stroke:

•**Atherosclerosis**: Plaque buildup in the large or small arteries (such as carotid arteries) leading to reduced or complete blockage of blood flow.

•**Lacunar Infarcts**: Result from small vessel disease, often due to hypertension or diabetes, leading to microatheromatous changes.

•**Pathophysiology of Large Vessel Atherosclerosis :**

1. Plaque in vessel wall ruptures .
2. Thrombus develops over plaque decreasing lumen diameter → Decreased blood flow beyond thrombus.
3. Ischemia → Infarction.

Small Vessel Atherosclerosis

•**lenticulostriate arteries:**

a) these arteries are branches of the middle cerebral artery (mca).

b) they supply blood to the basal ganglia, internal capsule, and parts of the corona radiata.

c) infarcts in these regions can cause lacunar strokes, which may result in motor or sensory deficits depending on the area affected.

•**small pontine penetrating arteries:**

a) these arteries are branches of the basilar artery.

b) they supply blood to the pons.

c) infarcts here can lead to symptoms such as dysarthria, diplopia, or ataxia, And depending on the location and size of the lesion

d) Thrombosis of penetrating intracranial arteries is most often the consequence of hypertension, with hypertrophy of the media and deposition of fibrinoid material. Microatheromas can obstruct the penetrating artery origins.

Thrombosis refers to obstructed blood flow due to a localized occlusive process within one or more vessels. The most common vascular pathology is atherosclerosis, in which fibrous tissue and lipid materials form plaques that encroach on the lumen. Atherosclerosis affects mostly the large cervical and intracranial arteries. Less commonly, a clot forms within the lumen due to a primary hematologic problem, for example, polycythemia, thrombocytosis, or hypercoagulability.

HEMORRHAGIC STROKES

Hemorrhagic strokes occur due to bleeding in or around the brain, caused by rupture of blood vessels. The main etiologies from the most common are:

1. Intracerebral Hemorrhage (ICH):

- **Hypertension:** Chronic high blood pressure weakens small arteries, particularly in areas like the basal ganglia, thalamus, or pons.
- **Cerebral Amyloid Angiopathy (CAA):** Common in older adults, amyloid deposition in the vessel walls leads to fragile vessels prone to rupture.
- **Arteriovenous Malformations (AVMs):** Congenital malformations where abnormal connections between arteries and veins lead to rupture.
- **Trauma:** Head injury can cause vessels to rupture, leading to bleeding within the brain tissue.

Hypertension

•Chronic Hypertension is the most common cause of ICH. Elevated blood pressure leads to changes in small blood vessels, particularly in deep brain structures like the basal ganglia, thalamus, pons, and cerebellum.

•Pathophysiology: Long-standing hypertension causes lipohyalinosis (a degenerative process of the vessel walls) and microaneurysms (Charcot-Bouchard aneurysms), making these vessels prone to rupture.

Cerebral Amyloid Angiopathy (CAA)

• Common in elderly patients, particularly those with Alzheimer's disease.

• Pathophysiology: Amyloid protein deposits within the walls of cortical and leptomeningeal vessels, leading to vessel fragility and rupture.

• CAA typically causes lobar hemorrhages, often in the frontal, parietal, and occipital lobes.

Arteriovenous Malformations (AVMs)

• AVMs are congenital abnormalities where arteries directly connect to veins without an intervening capillary bed, leading to high pressure in the venous side, which can cause rupture.

• AVMs are a common cause of ICH in younger individuals

COMMON VASCULAR PATHOLOGY

Atherosclerosis:

• Characterized by the formation of plaques made of fibrous tissue and lipid materials that encroach on the lumen of the vessel.

• Primarily affects large cervical and intracranial arteries.

Primary Hematologic Disorders:

• Clots can form within the lumen due to conditions such as:

• Polycythemia

• Thrombocytosis

• Hypercoagulability

Vessel wall pathologies leading to thrombosis include: vasoconstriction, fibromuscular dysplasia, and arterial dissection.

Thrombosis of penetrating intracranial arteries is most often the consequence of hypertension, with hypertrophy of the media and deposition of fibrinoid material. microatheromas can obstruct the penetrating artery origins.

HEMATOLOGIC CAUSES

•Polycythemia:

• Characterized by an abnormally high number of red blood cells (RBCs), particularly in conditions like polycythemia vera (a myeloproliferative disorder).
• The increased RBC mass leads to hyperviscosity, slowing blood flow and predisposing to stasis.
• This sluggish flow promotes clot formation in both arteries and veins, increasing the risk of stroke, deep vein thrombosis (DVT), and pulmonary embolism (PE).

•Thrombocytosis:

• Excessively high levels of platelets, seen in both primary (e.g., essential thrombocythemia) and secondary thrombocytosis (e.g., from inflammation or iron deficiency).
• Elevated platelet counts increase the likelihood of spontaneous platelet aggregation and clot formation, particularly in the arterial circulation.
• Thrombosis typically occurs in small vessels but can also affect larger arteries, causing complications like stroke, myocardial infarction (MI), or limb ischemia.

•Hypercoagulability:

• Refers to a state of heightened blood coagulation due to an imbalance between pro- clotting and anti-clotting factors.
• Inherited causes include Factor V Leiden mutation, prothrombin gene mutation, and protein C or S deficiency, which increase the tendency to form clots.
• Acquired causes include conditions like antiphospholipid syndrome (APS), malignancy, or prolonged immobility.
• This results in intravascular clot formation, affecting both arterial and venous systems, with risks of stroke, DVT, PE, and even recurrent pregnancy loss in APS.

ARTERIAL DISSECTION

•**Carotid Artery Dissection:**
• Presents with severe retro-orbital headache, ipsilateral to the dissection.

• **Mechanism:** Stroke occurs from thrombosis of the internal carotid artery (ICA) or embolus from the dissection.

• **Physical Exam:**
• Ipsilateral Horner's syndrome due to involvement of the oculosympathetic tract.
• Preserved perspiration (sympathetic fibers travel with the external carotid artery).
•Vertebral Artery (VA) Dissection:
• Often caused by neck manipulation or trauma.
• Presents with ipsilateral neck pain and posterior circulation stroke symptoms.

•Horner's Syndrome

occurs due to disruption of the sympathetic pathway that controls eye and facial muscle function.

• Characterized by:

• Ptosis: Drooping of the upper eyelid (due to loss of sympathetic innervation to the superior tarsal muscle).

• Miosis: Constriction of the pupil (due to unopposed parasympathetic action on the pupil).

• Anhidrosis: Loss of sweating, typically on the affected side of the face.

•OculosympathetiC Tract:

• This sympathetic pathway originates in the hypothalamus and descends through the brainstem and spinal cord before traveling to the cervical sympathetic chain.

• The fibers reach the internal carotid artery and ascend to the eye, controlling pupil dilation and eyelid elevation.

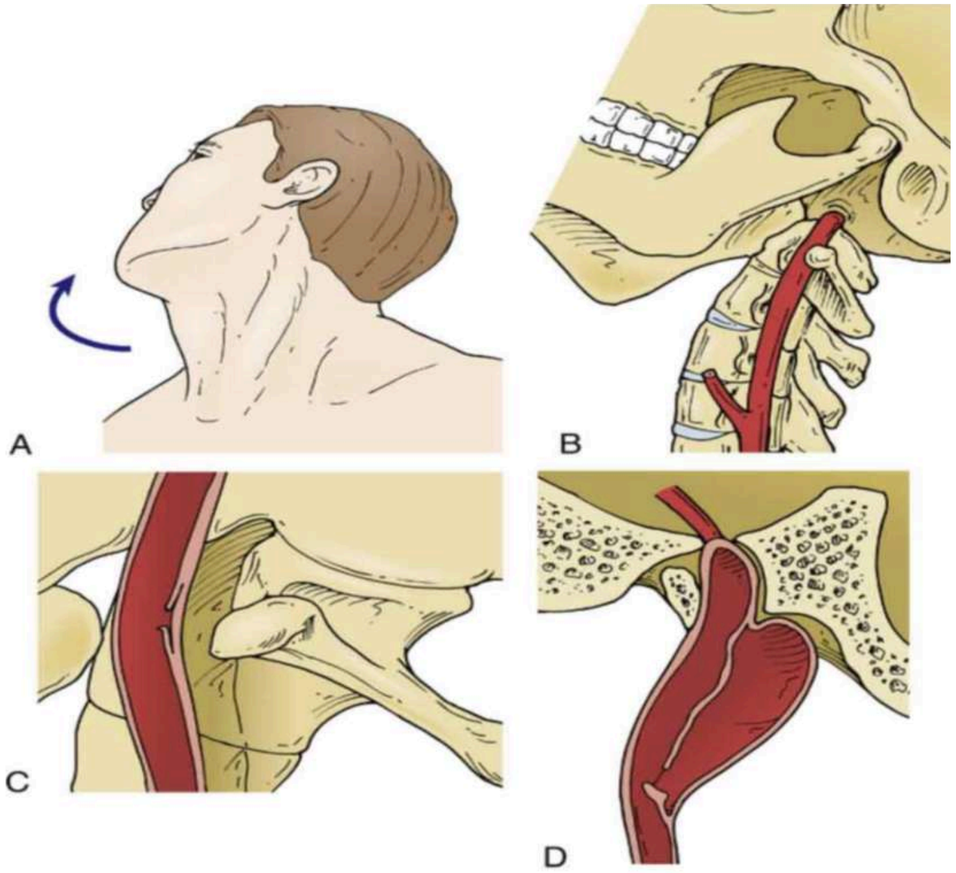
•Carotid Artery Dissection:

• Disrupts the sympathetic fibers traveling with the internal carotid artery.

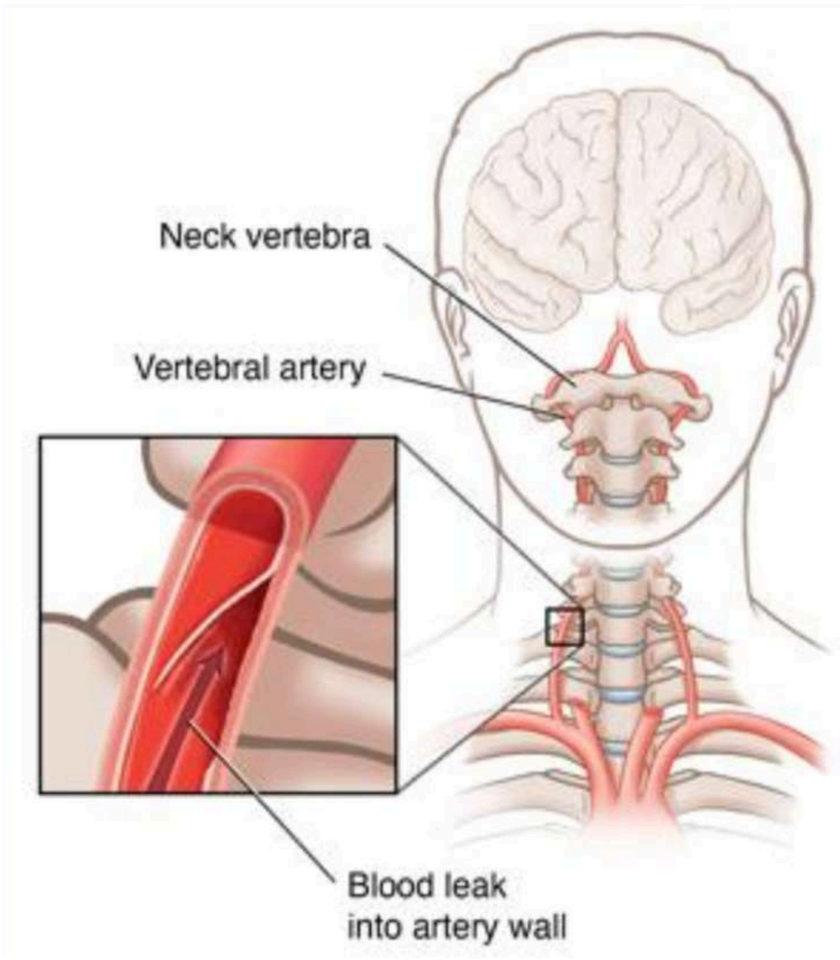
• Causes ipsilateral Horner's syndrome (same side as the dissection).

• In carotid dissection, perspiration remains intact on the face because the fibers responsible for sweating ascend with the external carotid artery, which is unaffected.

ARTERY DISSECTION IN CNS



One mechanism of carotid dissection caused by neck rotation. (Stringer WL and Kelly DL PMID 7366803)



CAA PATHOLOGY

CAA:
is a condition where beta-amyloid protein deposits accumulate in the walls of small- and medium-sized arteries of the brain's cortex and leptomeninges. This deposition weakens the vessel walls, making them prone to rupture, resulting in hemorrhage.

- CAA primarily affects elderly individuals and is especially common in patients with Alzheimer's disease.
- It is considered one of the leading causes of lobar intracerebral hemorrhage (ICH) in the elderly population.

Pathophysiology:
Amyloid deposits replace smooth muscle cells in the vessel walls, causing vessel fragility. This process leads to weakening and thinning of the blood vessels, predisposing them to spontaneous rupture.
CAA predominantly affects the superficial cortical arteries, including those in the frontal, parietal, and occipital lobes, leading to lobar hemorrhages (superficial rather than deep brain structures). Unlike other causes of ICH, such as hypertension, CAA rarely involves the deep white matter or basal ganglia.

AVM PATHOLOGY

•AVMs are congenital vascular malformations where there is a direct connection between arteries and veins without the normal intervening capillary bed. This abnormal connection
results in high-pressure arterial blood flowing directly into veins, leading to venous dilation and increased risk of rupture.

Epidemiology:

• AVMs are relatively rare but are a significant cause of intracerebral hemorrhage (ICH) in younger patients, particularly those aged 10 to 40.
•AVMs are usually diagnosed incidentally or after a hemorrhagic event.

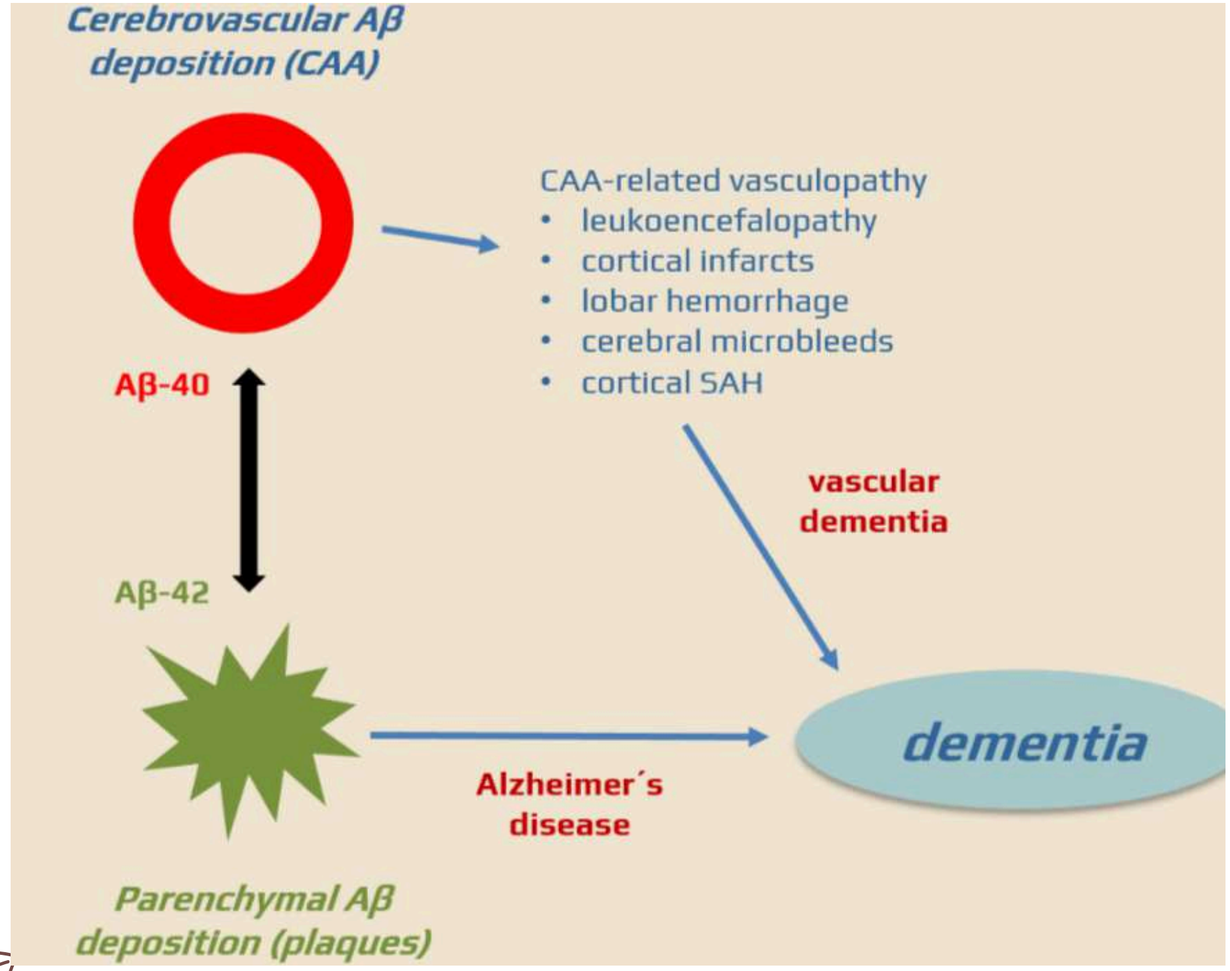
Pathophysiology:

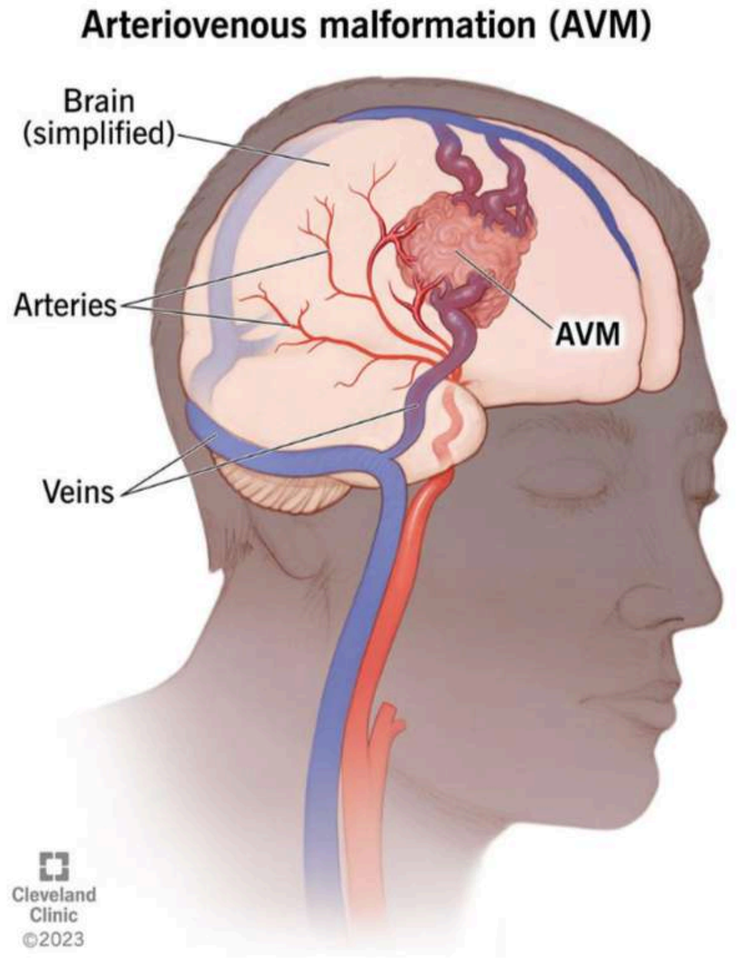
The absence of a capillary network in AVMs means that arterial blood, which is under high pressure, flows directly into the low-pressure venous system. Over time, this creates vascular stress on the veins, leading to their dilation, thinning, and eventual rupture, resulting in hemorrhage.

AVMs can cause two main types of symptoms:

1. Hemorrhage: The most common and serious complication, leading to intracerebral bleeding.
2. Seizures: Result from the irritation of the surrounding brain parenchyma due to abnormal vascular structure and possible ischemia.

CAA AND AVM


Cerebrovascular Aβ deposition (CAA)
CAA-related vasculopathy
• leukoencefalopathy
• cortical infarcts
• lobar hemorrhage
• cerebral microbleeds
• cortical SAH
Aβ-40
Aβ-42
vascular dementia
dementia
Alzheimer´s disease
Parenchymal Aβ deposition (plaques)


Arteriovenous malformation (AVM)
Brain (simplified)
Arteries
Veins
AVM
Cleveland Clinic ©2023

clinical feature of stroke in different vascular territories

By Heba Hmedan

ANTERIOR CIRCULATION

1. Left cerebral hemisphere strokes lead to:

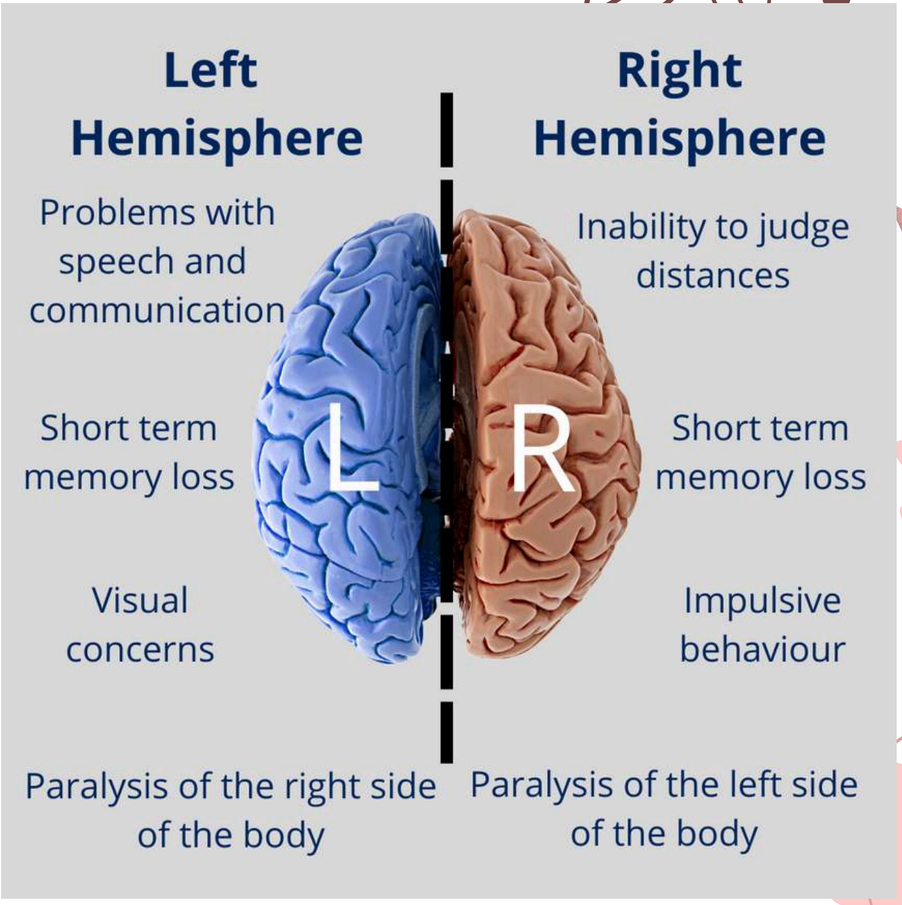
a. Right hemiparesis:often arm , hand, and face>leg

b. Right hemisensory loss

c. Aphasia

d. In large lesions, conjugate deviation of the eyes to the left; right hemianopia or hemi-inattention

e. When caused by ICA occlusive disease, transient left monocular visual loss mayalso occur.



ANTERIOR CIRCULATION

2. Right cerebral hemisphere strokes cause:

a. Left hemiparesis: often arm , hand , and face>leg

b. Left hemisensory loss

c. Poor drawing and copying

d. Neglect of the left visual field

e. In large lesions , conjugate deviation of the eyes to the right ,left hemianopia

f. When the signs are due to ICA occlusive disease, transient right monocular visual loss may accompany the brain signs.

POSTERIOR CIRCULATION

1. Lateral medullary stroke (Wallenberg syndrome, usually due to intracranial VA occlusion) causes :

a. Ipsilateral facial pain , or reduced pain and temperature sensation on the ipsilateral face, or both

b. Loss of pain and temperature in the contralateral limbs and body

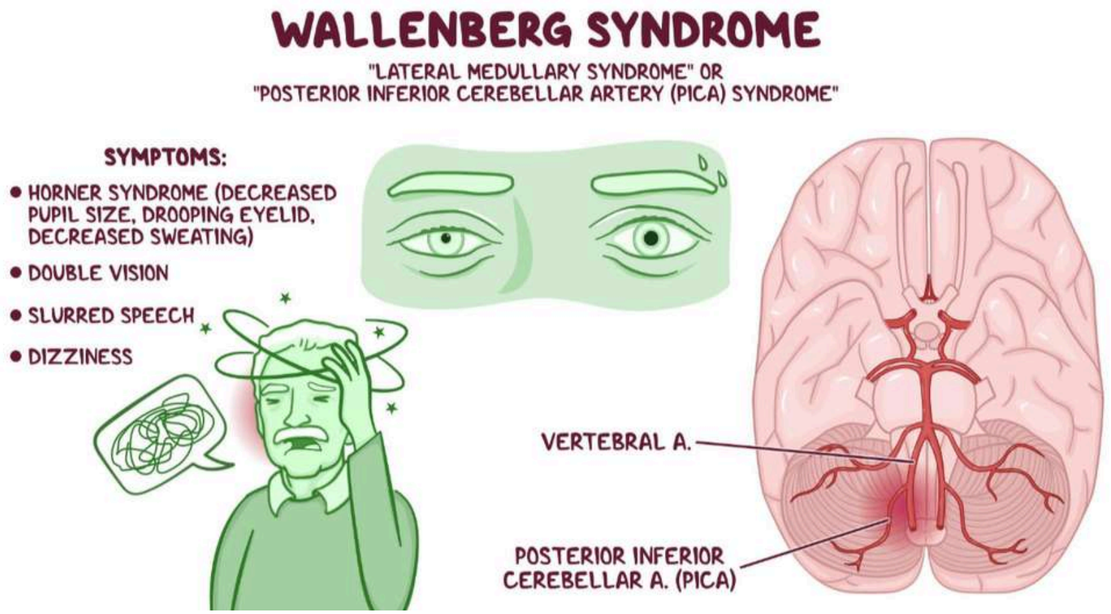
c. Ipsilateral Horner syndrome

d. Nystagmus (involuntary eye movements)

e. Incoordination of the ipsilateral arm

f. Leaning and veering while sitting or walking, with gait ataxia

g. In deep lesions, dysphagia and hoarseness



POSTERIOR CIRCULATION

2. Bilateral pontine base and often medial tegmentum stroke (usually due to basilar artery occlusion, or pontine hemorrhage) causes :

a. Quadriparesis (weakness in all four limbs)

b. Unilateral or bilateral conjugate gaze paresis; sometimes internuclear (person loses the ability to
move both eyes together in a coordinated manner towards one side)
ophthalmoplegia or sixth nerve palsy

c. When the medial tegmentum is involved bilaterally,coma

3. Cerebellar infarction (usually due to embolism to the PICA or SCA, or cerebellar hemorrhage) causes :

a. Gait ataxia; often inability to walk

b. Dysarthria (difficulty in articulating words due to problems with the muscles used for speech)

c. Ipsilateral arm dysmetria (inability to control the distance of the movement)

POSTERIOR CIRCULATION

4. Left PCA territory stroke causes :

a. Right homonymous hemianopia (person loses vision in the right half of the visual field in both eyes)

b. At times, amnesia (partial or complete loss of memory)

c. Alexia without agraphia when the splenium of the corpus callosum is involved (Inability to read , The ability to write is preserved)

5. Right PCA territory stroke causes :

a. Left homonymous hemianopia

b. At times, left-sided visual neglect (person is unaware of or does not respond to objects, people, or stimuli on the left side of their visual field, even though there is no problem with their vision)

LACUNAR SYNDROMES

• Pure motor lacune: weakness of the contralateral arm, face, and leg without sensory, visual, or cognitive or behavioral signs. Common locations of lacunes producing pure motor stroke include the corona radiata, posterior limb of the internal capsule, and pons.

• Pure sensory lacune: paresthesiae of the contralateral body, limbs, and face without motor, visual, or cognitive abnormalities. The most common location of a lacune producing pure sensory symptoms is the ventral posterior thalamus.

LACUNAR SYNDROMES

• Sensorimotor lacune: combination of motor and sensory lacunes. This syndrome is due to infarction in the ventral posterior thalamus and adjacent posterior limb of the internal capsule.

• Dysarthria—clumsy hand syndrome : slurred speech and clumsiness of the contralateral hand. The most common location of a lacune producing this syndrome is in the base of the pons.

• Ataxic hemiparesis: weakness and ataxia of the contralateral limbs, often greater in the leg and foot than in the arm and hand. The most common locations for lacunes producing ataxic hemiparesis are the base of the pons, the posterior limb of the internal capsule, and the corona radiata.

Tissue plasminogen activator (t-PA)

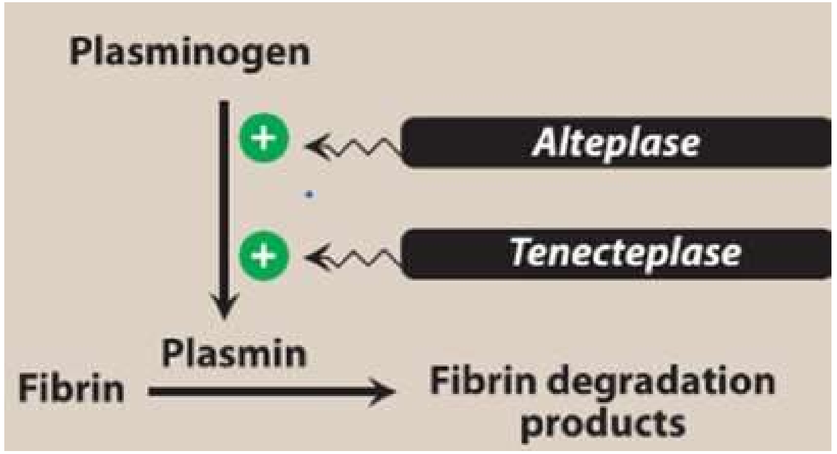
By Aya Abdullah

Tissue plasminogen activator (t-PA)

• t-PA is a protein(serine protease) that is naturally produced in the Body by the endothelial cells.

Mechanism of action:

- **Accumulation of fibrin → clot**
- **Plasmin is responsible for fibrinolysis**
- **t-PA: plasminogen(inactive) → plasmin (active)**



• If intravenous t-PA administered within 4.5 hours of the onset of an acute ischemic stroke, improved clinical outcome is seen at 3 months.

• Do not give t-PA if the time of stroke is unknown, if more than 4.5 hours have passed, or if the patient has any of the following: uncontrolled HTN, bleeding disorder, is taking anticoagulants or has a history of recent trauma or surgery. also in pregnancy, brain tumors and metastatic cancer.

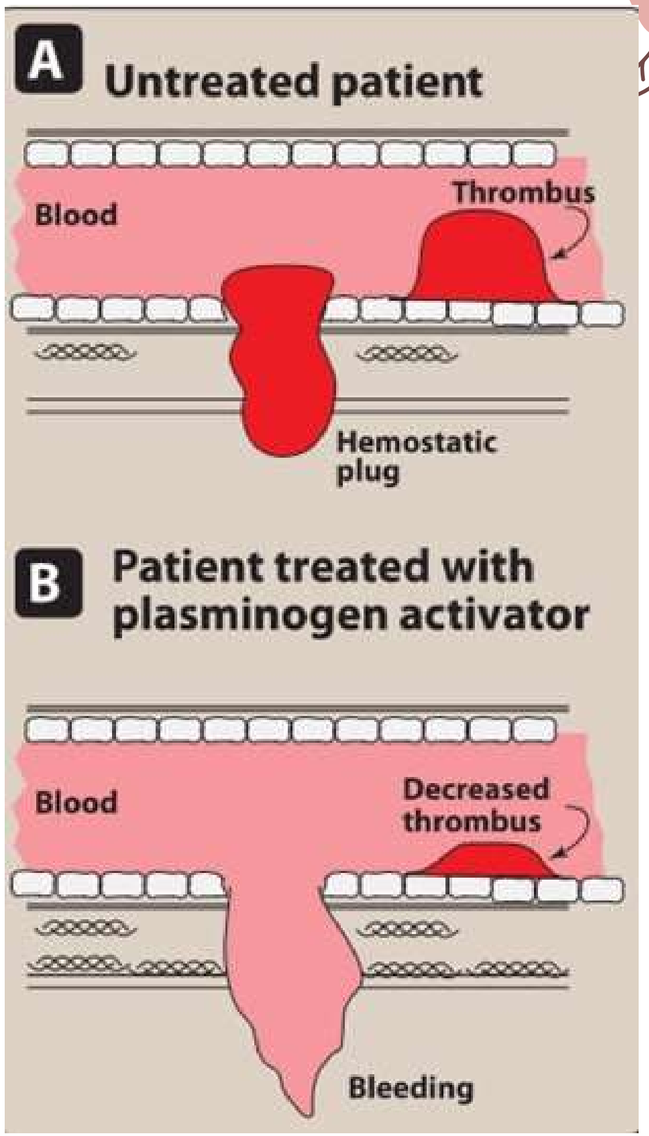
* These patients are at increased risk for hemorrhagic transformation

• Thrombolytic agents do not distinguish between the fibrin of an unwanted thrombus and the fibrin of a beneficial hemostatic plug. Thus, hemorrhage is a major adverse effect.

• excluded Hyper- and hypoglycemia before initiating t-PA because they may mimic the symptoms and signs of acute stroke.

• The patient is receiving t-PA—aggressive blood pressure control is necessary to reduce the likelihood of bleeding. (Keep BP < 185/110 mm Hg.)

* Medications: alteplase, reteplase, and tenecteplase.
- Alteplase has a very short half life (5-30 min), while tenecteplase has a longer half life



Stroke Prevention

By OTHMAN ALOTAIBI

prevention

Recurrence may be prevented by modifying risk factors, especially stopping smoking and, to a lesser extent, manipulating diet (low animal fat, low salt, avoiding excess alcohol) and prescribing cholesterol- lowering agents, i.e. statins. In the long term, control of blood pressure is also important.

For the first 2 weeks after an ischaemic stroke, how- ever, patients should not receive antihypertensive therapy beyond their pre- existing treatment unless there is evidence of malignant hypertension. This is because too rapid lowering of blood pressure may worsen ischaemia in a region where the cerebral circulation is already compromised

Treatment

- modifying risk factors
- treating hypertension
- stopping smoking
- reducing serum cholesterol by diet and drugs
- antiplatelet drugs (low-dose aspirin)
- anticoagulants (warfarin)

carotid endarterectomy

- surgical intervention to clear atheroma from the origin of the internal carotid artery is warranted for symptomatic severe carotid stenosis (greater than 70% stenosis), after TIAs or indeed minor stroke. Carotid angioplasty and stenting is a viable alternative to surgery

- The role of surgery for less severe or asymp-tomatic carotid stenoses is not so well established. At present, there is no surgical option for most vertebrobasilar TIAs (with the rare exception of the subclavian steal syndrome), though selected patients have been treated by vertebral angioplasty and stenting.

Complication associated with stroke

By Abdullah Al-Harbi

Complication associated with stroke .

The complications of stroke can vary widely depending on the location. Type and severity of the stroke as well as health of the individual. And it may occur immediately after the stroke or develop over time.

- Pneumonia:over 50% of stroke patients are acutely dysphagia. With a risk of aspiration.
- Septicaemia : pressures or UTI
- Deep vein thrombosis and pulmonary embolism
- Myocardialinfarction.Arrhythmias and heart failure
- Recurrent stroke: especially if the risk factors are not controlled like DM hypertension or atrial fibrillation

- Memory and cognitive impairment
- Hemiplegia and hemiparesis: when motor areas are affected
- Sensory deficitsLoss of sensation or altered sensations on one side of the body, especially when the parietal lobe is affected
- Dysphagia

Done by Group C11

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- **OTHMAN A SH ALOTAIBI**
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Thank You