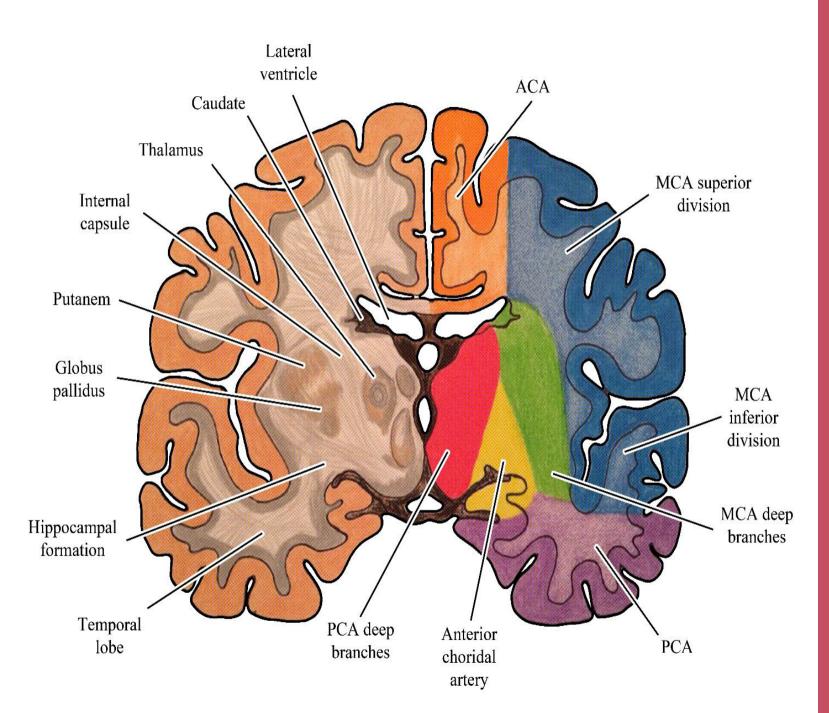
Cerebrovascular diseases

Neurology Rotation Fourth Year Medical Students

Vascular Anatomy

The <u>cerebral vasculature</u> is divided into the anterior and posterior circulation, with the <u>anterior</u> (<u>carotid</u>) circulation supplying the cerebral hemispheres except the medial temporal lobes and a portion of the occipital lobes, and the posterior (vertebrobasilar) circulation supplying the brainstem, thalami, cerebellum and the posterior portion of the cerebral hemispheres



A) Anterior circulation

The <u>right</u> common carotid artery (CCA) branches from the innominate artery. The <u>left</u> CCA arises directly from the aorta.

The CCA divides in the neck into the <u>internal carotid artery</u> (<u>ICA</u>) and the <u>external</u> <u>carotid artery</u>.

The <u>ICA</u> travels behind the pharynx, entering the skull where it forms an S-shaped curve –the <u>carotid siphon</u>.

This portion of the ICA gives rise to the <u>ophthalmic</u> <u>artery</u>.

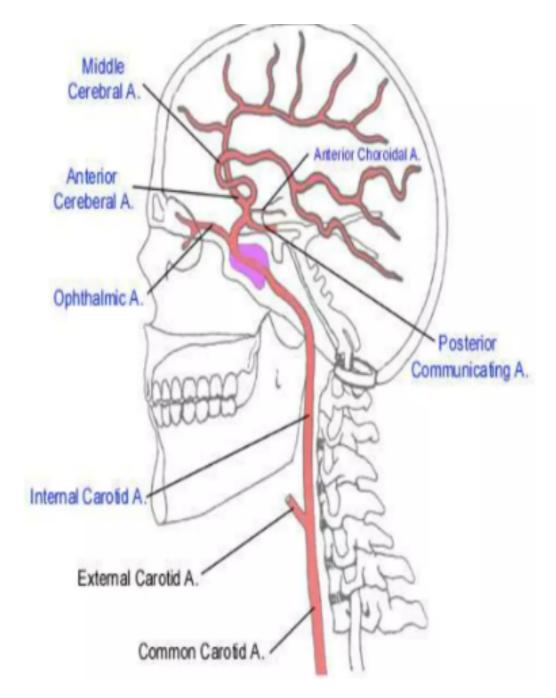
The ICA then penetrates the dura and gives off the anterior choroidal and posterior communicating arteries before <u>bifurcating into</u> the <u>anterior cerebral</u> (ACA) and <u>middle</u> <u>cerebral arteries</u> (MCA).

The <u>ACA</u> supplies the anterior medial cerebral hemispheres, the caudate nuclei and the basal frontal lobes.

The <u>anterior communicating</u> artery connects the two ACAs.

The <u>MCA</u> courses laterally, giving off <u>lenticulostriate artery branches</u> to the basal ganglia and internal capsule.

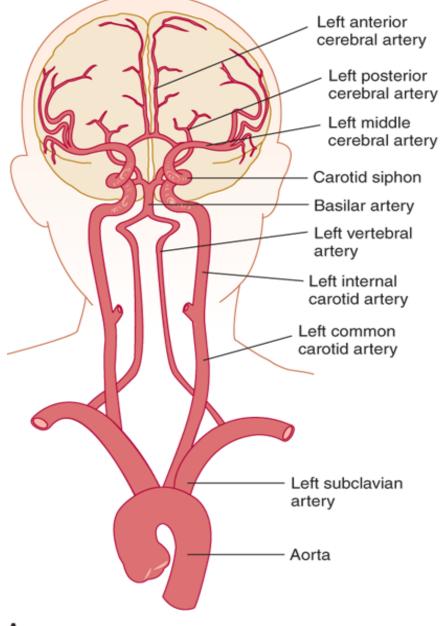
The <u>MCA</u> trifurcates into small anterior temporal branches and <u>large superior</u> and <u>inferior</u> divisions.

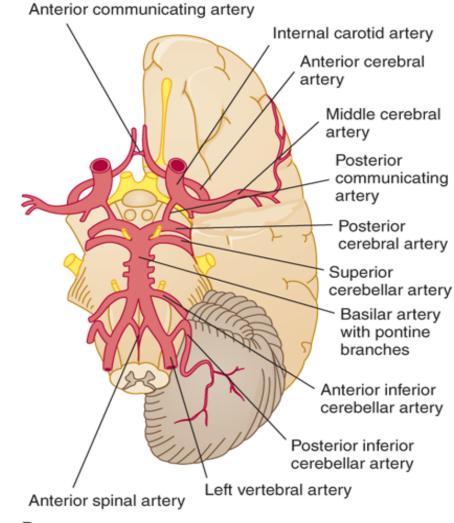


The first branch of each subclavian artery is the vertebral artery (VA).

The <u>VA</u> enters the <u>spinal column</u> via the transverse foramina of C5 or C6 and runs within the intervertebral foramina, <u>exiting to course behind</u> the <u>atlas</u> before piercing the dura mater to <u>enter</u> the <u>foramen magnum</u>.

The <u>intracranial VAs join</u> to form the <u>basilar artery</u> at the ponto-medullary junction.





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Source: Aaron L. Berkowitz: Clinical Neurology and Neuroanatomy: A Localization-Based Approach www.neurology.mhmedical.com Copyright © McGraw-Hill Education. All rights reserved. The <u>intracranial VA</u> gives off <u>posterior</u> and <u>anterior spinal artery</u> branches, penetrating arteries to the medulla and the posterior inferior cerebellar artery (<u>PICA</u>).

The <u>basilar artery</u> then runs in the midline along the clivus giving off bilateral <u>anterior inferior cerebellar artery</u> (<u>AICA</u>) and <u>superior cerebellar</u> <u>artery</u> (<u>SCA</u>) branches before dividing at the Ponto mesencephalic junction into the <u>posterior cerebral arteries</u> (PCA).

Small <u>penetrating arteries</u> arise at the basilar artery bifurcation to supply the <u>medial</u> portions of the <u>midbrain</u> and <u>thalami</u>.

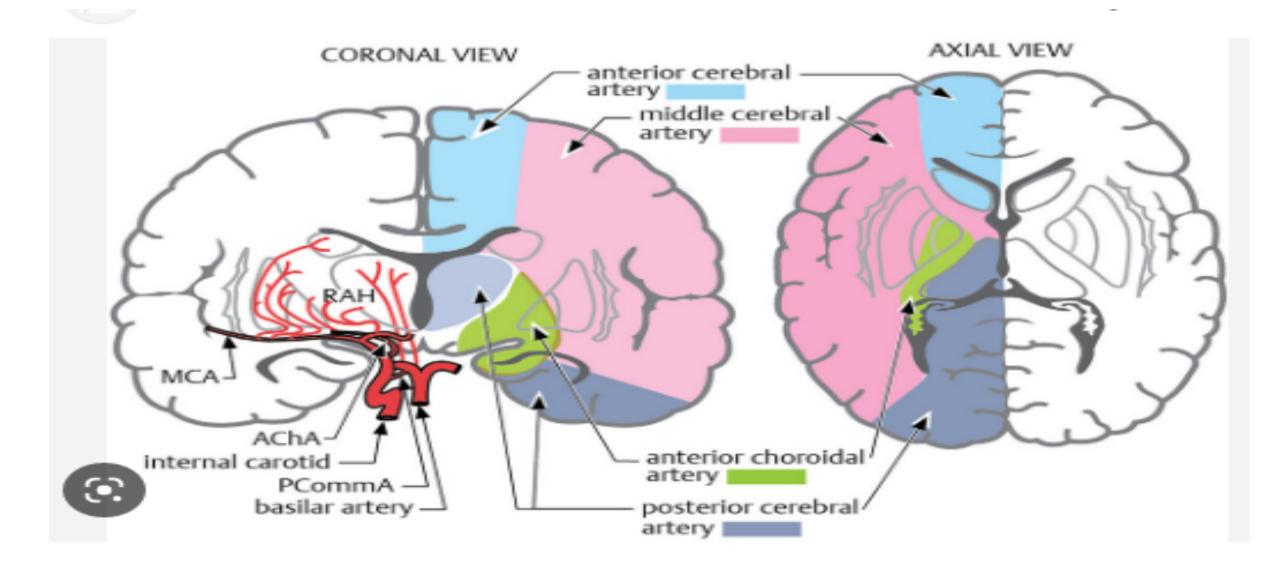
The <u>vascular supply</u> of the <u>brainstem</u> includes large <u>paramedian</u> <u>arteries</u> and smaller, <u>short circumferential</u> <u>arteries</u> that penetrate the basal portion of the brainstem into the tegmentum.

Long circumferential arteries course around the brainstem and give off branches to the lateral tegmentum.

The <u>PCA</u> gives off <u>penetrating arteries</u> to the <u>midbrain</u> and thalamus, courses around the cerebral peduncles and then supplies the <u>occipital</u> <u>lobe</u> and the <u>inferior surface</u> of the <u>temporal</u> lobe.

The <u>circle</u> of <u>Willis</u> connects the anterior circulation of each side through the <u>anterior communicating</u> artery, and the posterior and anterior circulation of each side through the <u>posterior communicating</u> artery.

Each <u>carotid</u> artery supplies <u>four-fifths</u> of the <u>brain</u>, the vertebrobasilar circulation, one-fifth



contical vascular territories

Anterior cerebral artery

Middle cerebral artery

Posterior cerebral artery

Definition of Stroke

- Strokes are a life-threatening emergency, and immediate medical attention is critical to prevent permanent damage or death.
- <u>The World Health Organization (WHO)</u> <u>defines stroke as "rapidly developing</u> <u>clinical signs of focal (or global)</u> <u>disturbance of cerebral function, with</u> <u>symptoms lasting 24 hours or longer or</u> <u>leading to death, with no apparent cause</u> <u>other than vascular origin"</u>

• There are three main types of ischemic stroke:

1) Thrombotic strokes: caused by a blood clot that forms in an artery that supplies blood to the brain, also called an atherosclerotic stroke. It affects mostly the large cervical and intracranial arteries. Less commonly a clot forms within the lumen due to a primary hematological disease (Polycythemia, Essential thrombocytosis or hypercoagulability).

<u>Vessel wall pathologies</u> leading to thrombosis include vasoconstriction, **fibromuscular dysplasia and arterial dissection**.

<u>Thrombosis of penetrating intracranial arteries is</u> most often the consequence of hypertension, with hypertrophy of the media and deposition of fibrinoid material(<u>lipohyalinosis</u>).

Types of ischemic stroke

2) Embolic strokes: When a clot forms somewhere else in the body and travels through the blood vessels to the brain. It gets stuck there and stops the flow of your blood. The <u>material</u> arises proximally, mostly from the <u>heart</u> or from <u>major arteries</u> such as the aorta, ICAs and VAs and from <u>systemic veins</u>. Atrial fibrillation increases the risk of clots forming in the heart that can then travels to the brain.

- <u>Cardiac sources</u> of embolism include the heart valves, endocardium and clots or tumors within the atrial or ventricular cavities.
- <u>Thrombi</u> originating in <u>systemic veins</u> travel to the brain through cardiac defects such as an atrial septal defect **or a patent foramen ovale, a process termed** <u>paradoxical embolism</u>.

Occasionally, air, fat, <u>cholesterol</u> <u>crystals</u>, bacteria and foreign bodies enter the vascular system and embolize to brain vessels Types of ischemic stroke

3) <u>Systemic hypoperfusion</u>

- Decreased blood flow to brain tissue may be caused by <u>low systemic perfusion pressure</u>.
- The <u>most common</u> causes are <u>cardiac pump failure(</u> most often due to myocardial infarction or arrhythmia) and <u>systemic hypoperfusion</u> (due to blood loss or hypovolemia).
- The <u>lack</u> of <u>perfusion</u> is more <u>generalized</u> than in localized thrombosis or embolism and <u>affects brain</u> <u>diffusely</u> and <u>bilaterally</u>.
- Poor perfusion is most critical in <u>border zone</u> or socalled <u>watershed regions</u> at the periphery of the major vascular supply territories, for example, between the ACA and MCA or between the MCA and

Types of ischemic stroke

Risk factors of ischemic stroke

- •Age over 60 years old
- History of <u>hypertension</u>, <u>heart</u> <u>diseases</u>, <u>hyperlipidemia</u>, <u>or diabetes</u>.
- •Arrythmia
- Smoking History
- Family history of strokes

Complications of ischemic strokes include Permanent weakness, Seizures and Vascular Dementia

Classification of Ischemic Stroke by Anatomy

| Site of the lesion | Associated effects |
|---------------------------|--|
| Anterior cerebral arten/ | Contralateral hemiparesis and sensory loss, lower extremity > upper |
| Middle cerebral artery | Contralateral hemiparesis and sensory loss, upper extremity > lower Contralateral homonymous hemianopia Aphasia |
| Posterior cerebral artery | Contralateral homonymous hemianopia with macular sparing Visual agnosia |

| Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain) | Ipsilateral CN III palsy Contralateral weakness of upper and lower extremity |
|--|---|
| Posterior inferior cerebellar artery (lateral medullary syndrome, Wallenberg syndrome) | Ipsilateral: facial pain and temperature loss Contralateral: limb/torso pain and temperature loss Ataxia, nystagmus |
| Anterior inferior cerebellar artery (lateral pontine syndrome) | Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness |
| Retinal/ophthalmic artery | Amaurosis fugax |
| Basilar artery | 'Locked-in' syndrome |

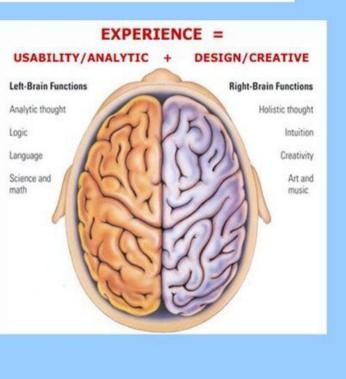
Common Ischemic Stroke Syndromes

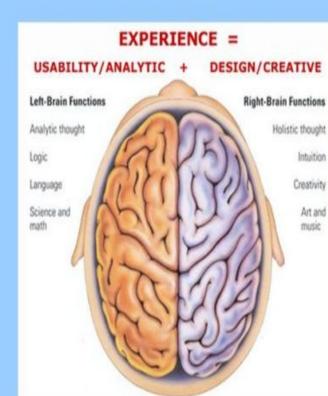
<u>Anterior Circulation:</u> Left/Right <u>cerebral hemisphere lesions</u> are most often <u>caused</u> by carotid artery occlusion, embolism to the MCA or its branches or basal ganglionic intracerebral hemorrhages.

Left Hemisphere Stroke

Effects right side of body

- Speech and language
- Aphasia
- Slow, cautious behavior
- Memory problems





 Effects left side of body

Right Hemisphere Stroke

- Spatial or perceptual abilities
- Impulsive, unaware
 of their impairments
- Left-sided neglect
- Short-term memory problems
- Often non-dominant

• **<u>Posterior circulation:</u>**

1. Lateral <u>medullary stroke</u> (<u>Wallenberg</u> syndrome, usually due o <u>intracranial VA</u> or <u>posterior inferior cerebellar</u> <u>artery</u>/ <u>PICA</u> <u>occlusion</u>)

- 2. <u>Bilateral pontine base</u> and often medial tegmentum <u>stroke(</u> usually due to <u>basilar artery occlusion</u> or <u>pontine hemorrhage</u>) causes:
- Quadriparesis

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- Unilateral or bilateral conjugate gaze paresis; sometimes internuclear ophthalmoplegia or 6th nerve palsy
- When the medial tegmentum is involved bilaterally, coma.
 - 3. <u>Cerebellar infarction</u> (<u>usually</u> due to <u>embolism</u> to the PICA or SCA, or cerebellar hemorrhage) causes:
- Gait ataxia
- Dysarthria
- Ipsilateral arm dysmetria



4. Left PCA territory stroke

causes:

Right homonymous hemianopia

b) At times, amnesia

c) Alexia (disorder of reading) without agraphia when the splenium of the corpus callosum is involved

5. Right PCA territory stroke causes:

Left homonymous

hemianopia

b) At times, left-sided visual neglect

PCA territory infarcts are most often caused by embolism arising



Lacunar syndromes

1.

Lacunar strokes are most often due to <u>occlusion</u> of a <u>penetrating artery</u>. Lacunar strokes may occur in either the <u>anterior</u> or the <u>posterior circulations</u>.

<u>Pure motor stroke</u>: Weakness of the contralateral arm, face and leg without sensory, visual or cognitive/behavioral signs. Common <u>locations</u> include **the corona radiata, posterior limb of the** <u>internal capsule</u> and <u>pons.</u>

2. <u>Pure sensory stroke</u>: Paresthesia of the contralateral body, limbs and face without motor, visual or cognitive abnormalities. The most common <u>location</u> is due to infarction in the <u>ventral posterior thalamus</u>.

3. Sensorimotor stroke: Combination of motor and sensory lacunes due to infarction in the <u>ventral</u> posterior <u>thalamus</u> and adjacent <u>posterior limb</u> of the <u>internal capsule</u>.

4<u>. Dysarthria-clumsy hand syndrome</u>: Slurred speech and clumsiness of the contralateral hand. The most common location is **in the <u>base</u> of the <u>pons.</u>**

5. <u>Ataxic hemiparesis:</u> Weakness and ataxia of the contralateral limbs, often greater in the leg and foot than in the arm and hand. The most common locations are the base of the <u>pons</u>, posterior limb of the <u>internal</u> <u>capsule</u> and <u>corona radiata</u>.

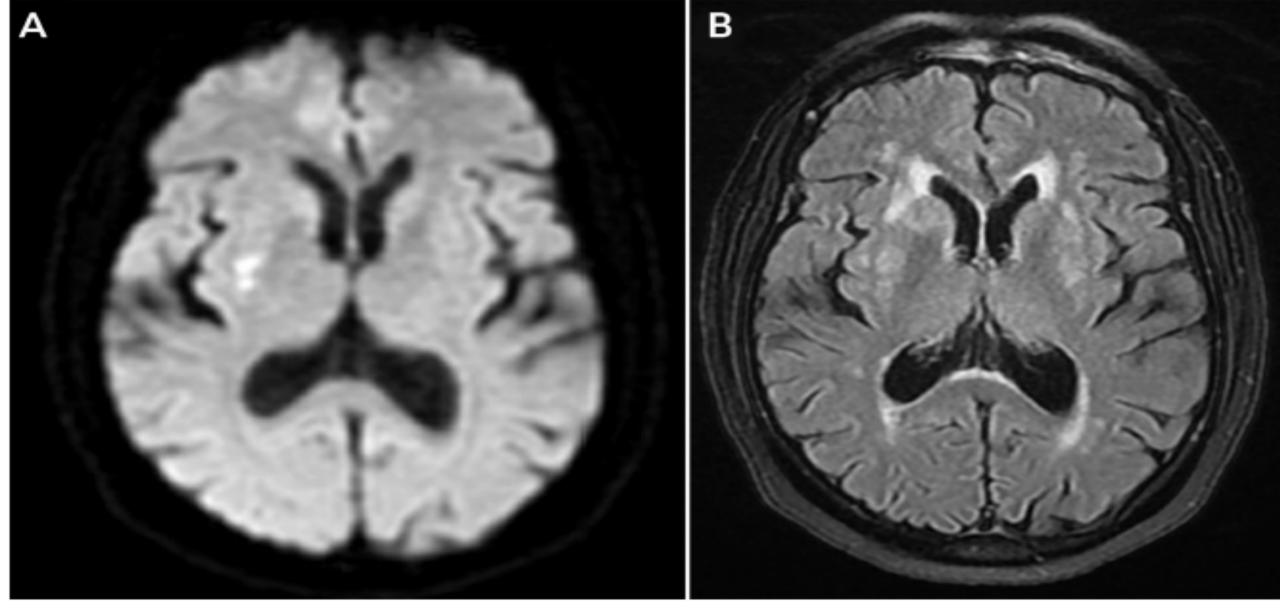


FIGURE 5-2

Imaging of the patient in CASE 5-1. Axial diffusion-weighted MRI (A) shows an acute infarct involving the right lentiform nucleus and internal capsule, and axial fluid-attenuated

<u>Arterial dissection:</u>

• Dissection of the carotid or vertebral arteries may lead to ischemic stroke

• a) Carotid dissection:

- - Typically presents with severe retro-orbital headache ipsilateral to the lesion
- <u>Strokes</u> involve the anterior circulation and occur either by thrombosis of the ICA or more commonly by an embolus arising from the dissection.
- <u>On examination</u>: patients may have an **ipsilateral <u>Horner's syndrome</u>** due to the involvement of the ascending oculosympathetic tract. <u>Perspiration</u> is <u>preserved</u> because those fibers ascend with the external carotid artery.
- b) <u>VA dissection may be produced by neck manipulation</u> or <u>trauma</u> and is commonly associated with ipsilateral neck pain and stroke in the posterior circulation.

Transient Ischemic Attack

• 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.

The mechanisms of TIA are identical to those of ischemic stroke.

Patients who have had a TIA have a <u>10% risk</u> of <u>stroke</u> in the <u>90</u> <u>days</u> following the event, and the <u>greatest risk</u> is <u>within</u> the <u>first 24</u> <u>hours</u> following a TIA.

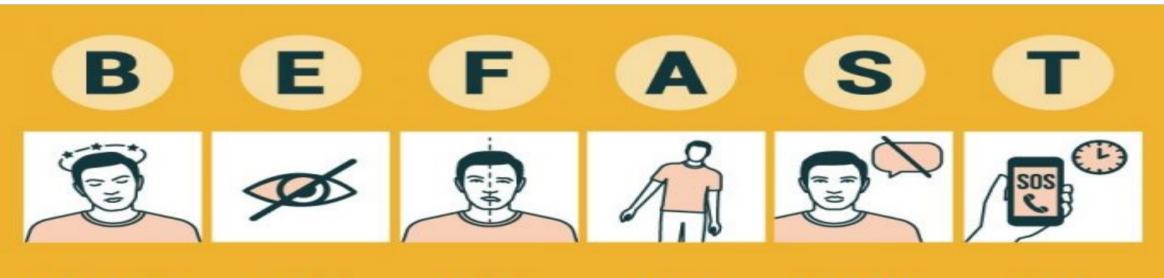
Therefore, the <u>evaluation</u> should be <u>identical</u> to that for a completed <u>stroke</u> and should be conducted just as <u>quickly</u>.



Assessment of Ischemic Strokes

Whilst the diagnosis of stroke may sometimes be obvious in many cases the presenting symptoms may be vague and accurate assessment difficult.

The BE FAST screening tool is widely known and has a positive predictive value of 78%. A variant of BE FAST called the ROSIER score is useful for medical professionals.





LOSS OF BALANCE, HEADACHE OR DIZZINESS EYES BLURRED VISION

ONE SIDE OF THE FACE IS DROOPING ARM OR LEG

WEAKNESS

SPEELA

SPEECH DIFFICULTY

TIME

TIME TO CALL FOR AMBULANCE IMMEDIATELY

ROSIER score

Exclude hypoglycaemia first, then assess the following:

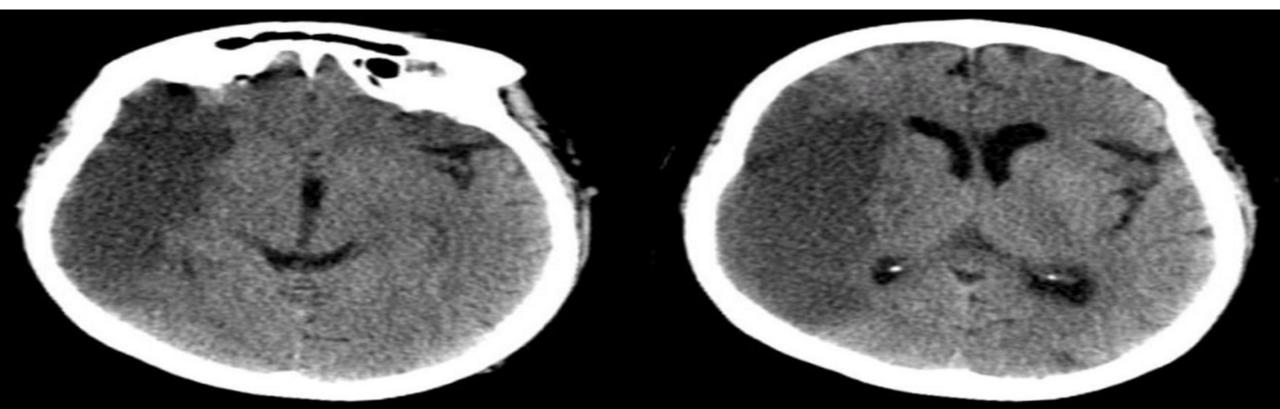
| Assessment | Scoring |
|--|-----------|
| Loss of consciousness or syncope | - 1 point |
| Seizure activity | - 1 point |
| New, acute onset of: | |
| asymmetric facial weakness | + 1 point |
| asymmetric arm weakness | + 1 point |
| asymmetric leg weakness | + 1 point |
| speech disturbance | + 1 point |
| visual field defect | + 1 point |

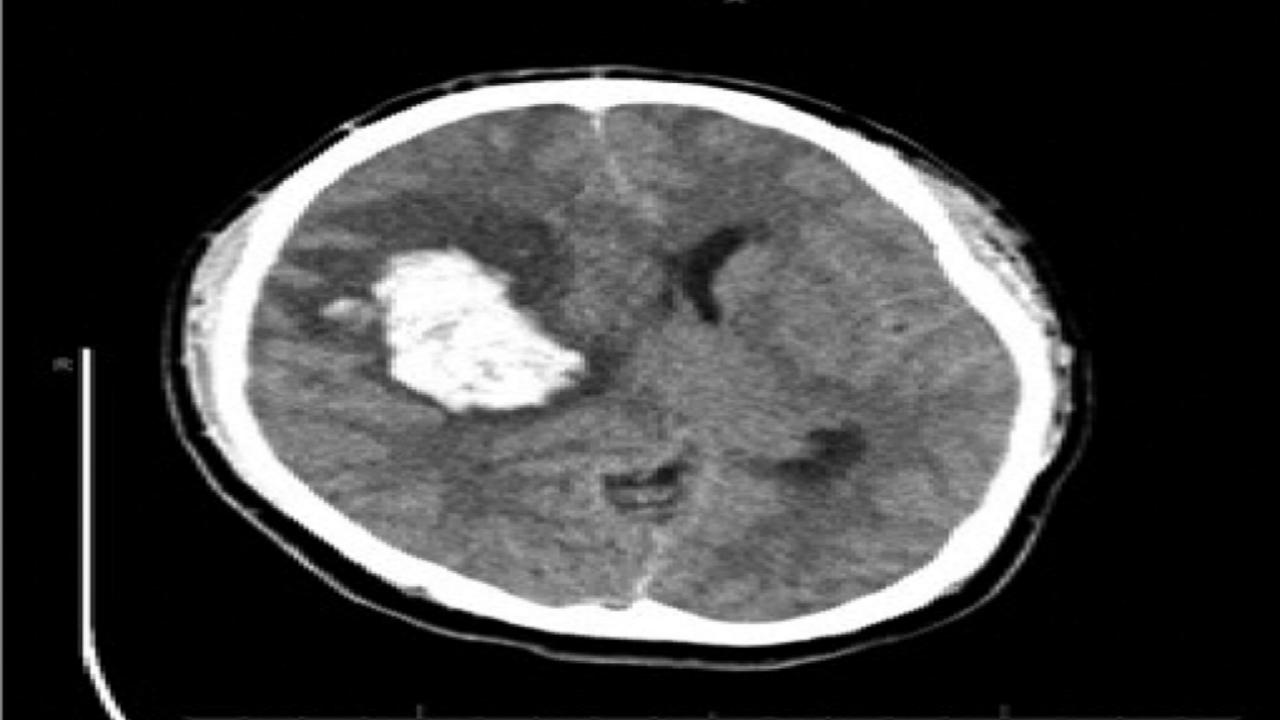
Assessment of Ischemic Strokes

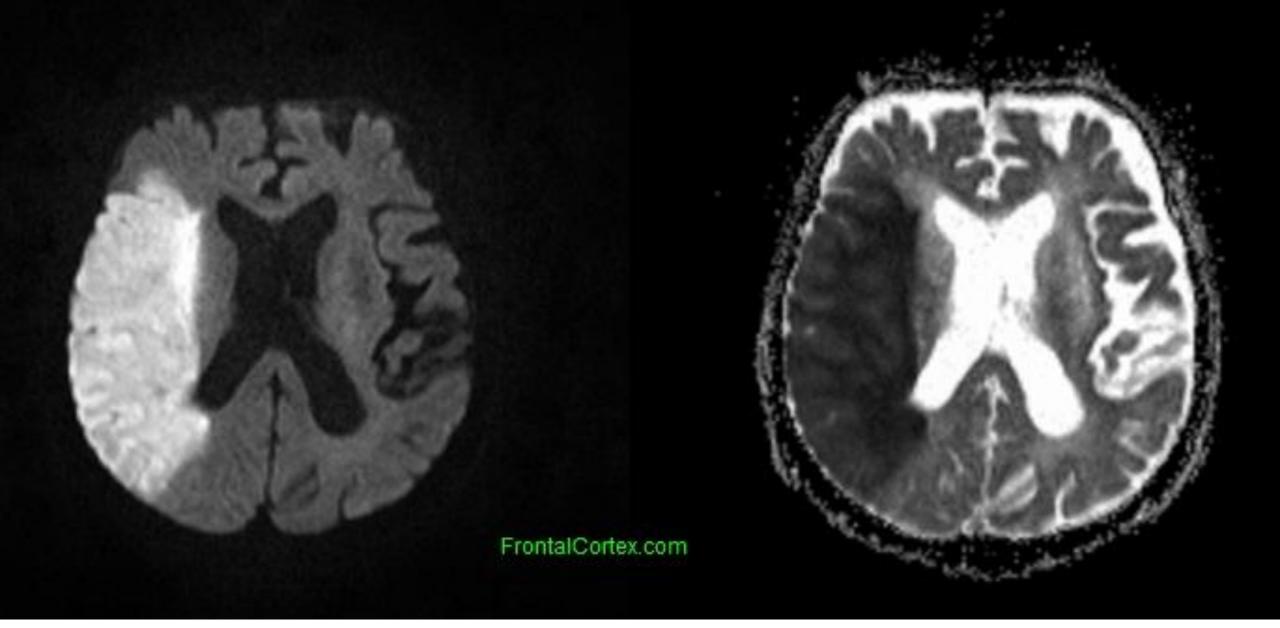
<u>A non-contrast CT brain scan is the first line radiological investigation for suspected stroke</u> as it helps to exclude hemorrhagic strokes or another pathology such as brain tumors before considering the usage of any antiplatelets or thrombolytics and thrombectomy in acute Stroke Management.

Acute ischemic strokes show areas of low density in the grey and white matter of the territory. Acute hemorrhagic strokes typically show areas of hyperdense material (blood) surrounded by low density (oedema).

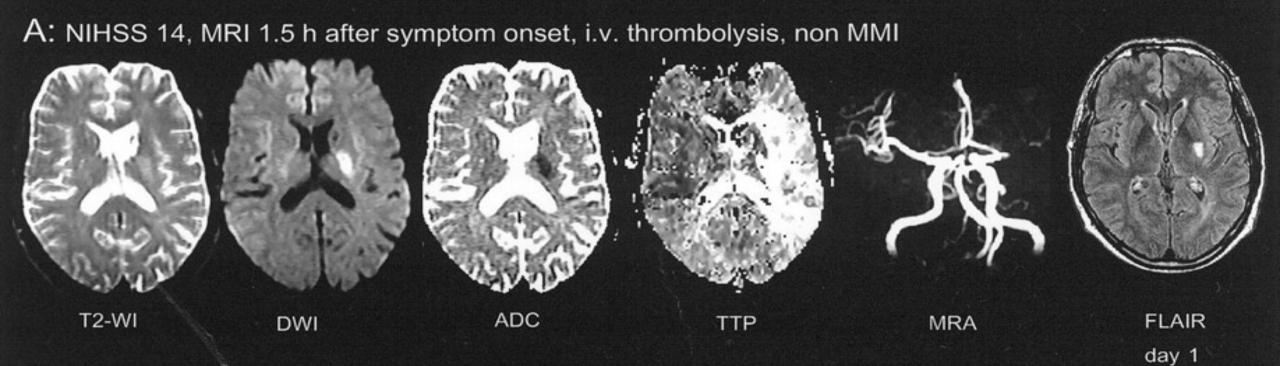
MRI Brain with diffusion weighted imaging is more sensitive to acute brain infarction than is CT.



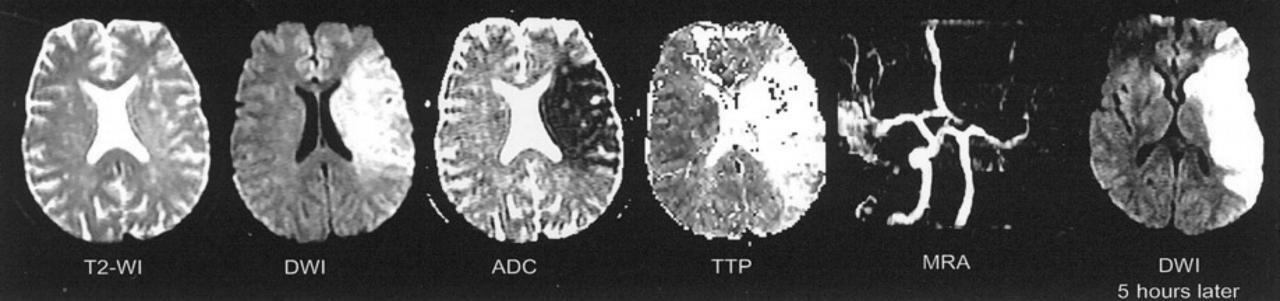




Acute Right MCA stroke on MRI (DWI on the left and ADC sequence on the right)



B: NIHSS 21, MRI 2h after symptom onset, MMI, hemicraniectomy



Diagnostic Work up for Ischemic Strokes

• Laboratory Work Up: Complete blood count, PT, PTT, Kidney function test, liver function test, Cardiac enzymes, Electrolytes, RBS level, HBA1C, Lipid Profile. Rheumatological work up is sometimes needed when history is significant for Antiphospholipid syndrome, Bechet disease or Vasculitis.

•Computerized tomography (CT) scan.

•Magnetic resonance imaging MRI/MRA/DW Images.

•Carotid ultrasound: shows buildup of fatty deposits called plaques and blood flow in the carotid arteries.

•Echocardiogram: can find a source of clots in the heart that may have traveled to the brain and caused a stroke.

•In cases of suspected <u>arterial dissection</u>, <u>CTA</u> or <u>MRA</u> with fat-suppressed imaging ("<u>fat sats</u>") to evaluate the cervical carotid and vertebral arteries should be obtained.

•Intensive investigation for coagulopathy may be required for some patients.

FIGURE 2-4. MRA of the circle of Willis. MRA, magnetic resonance angiography.

Management of Ischemic Strokes

- Blood glucose, hydration, oxygen saturation and temperature should be maintained within normal limits.
- Blood pressure should not be lowered in the acute phase unless there are complications such as Hypertensive Encephalopathy.
- Aspirin 300mg orally or rectally should be given as soon as possible if a hemorrhagic stroke has been excluded.
- Thrombolysis with alteplase should only be given if:
- it is administered within 4.5 hours of onset of stroke symptoms.
- Brain hemorrhage has been definitively excluded (Imaging has been performed)

| Absolute | Relative |
|--|---|
| Previous intracranial haemorrhage Seizure at onset of stroke Intracranial neoplasm Suspected subarachnoid haemorrhage Stroke or traumatic brain injury in preceding 3 months Lumbar puncture in preceding 7 days Gastrointestinal haemorrhage in preceding 3 weeks Active bleeding Pregnancy Oesophageal varices Uncontrolled hypertension >200/120mmHg | Concurrent anticoagulation (INR > 1.7) Haemorrhagic diathesis Active diabetic haemorrhagic retinopathy Suspected intracardiac thrombus Major surgery / trauma in the preceding 2 weeks |

Thrombectomy for acute ischemic strokes

- NICE recommend a pre-stroke functional status of less than 3 on the modified Rankin scale and a score of more than 5 on the National Institutes of Health Stroke Scale (NIHSS).
- Offer thrombectomy as soon as possible and <u>within 6 hours</u> of symptom onset, together with intravenous thrombolysis (if within 4.5 hours), to people who have: acute ischemic stroke and confirmed occlusion of the <u>proximal anterior circulation</u> demonstrated by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA).
- Offer thrombectomy as soon as possible to people who were last known to be well <u>between 6 hours</u> <u>and 24 hours</u> previously (including wake-up strokes): confirmed occlusion of the <u>proximal anterior</u> <u>circulation</u> demonstrated by CTA or MRA and if there is the potential to <u>salvage brain tissue</u>, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.
- Consider thrombectomy together with intravenous thrombolysis (if within 4.5 hours) as soon as possible for people last known to be well up to <u>24 hours</u> previously (including wake-up strokes): who have acute ischemic stroke and confirmed occlusion of the <u>proximal posterior circulation</u> (that is, basilar or posterior cerebral artery) demonstrated by CTA or MRA and if there is the potential to <u>salvage brain tissue</u>, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.

Secondary Prevention of ischemic strokes

- With regards to carotid artery endarterectomy: It is recommended if patient has suffered stroke or TIA in the carotid territory and are not severely disabled. It should only be considered if carotid stenosis > 70% according ECST (European Carotid Surgery Trialists' Collaborative Group) criteria or > 50% according to NASCET (North American Symptomatic Carotid Endarterectomy Trial) criteria.
- Patients with high-risk TIA and low NIHSS stroke should be treated with clopidogrel load in the first 24 hours followed by **DAPT for 21 days.**
- Statin should be prescribed for patients who have high lipid profile.
- Patients with ischemic stroke or transient ischemic attack and **atrial fibrillation should** receive oral anticoagulant therapy for secondary stroke prevention.
- In patients with stroke due to intracranial atherosclerosis ,dual antiplatelet therapy with aspirin and clopidogrel is favored.

Hemorrhagic Strokes

<u>Bleeding inside</u> the <u>skull</u> can be <u>divided</u> into **subarachnoid**, **intracerebral**, epidural and subdural hemorrhages.

The latter 2 types of hemorrhage are almost always traumatic.

Intracerebral hemorrhage (<u>ICH</u>) and subarachnoid hemorrhage (<u>SAH</u>) have <u>different causes</u>, <u>clinical</u> findings and <u>management</u>.

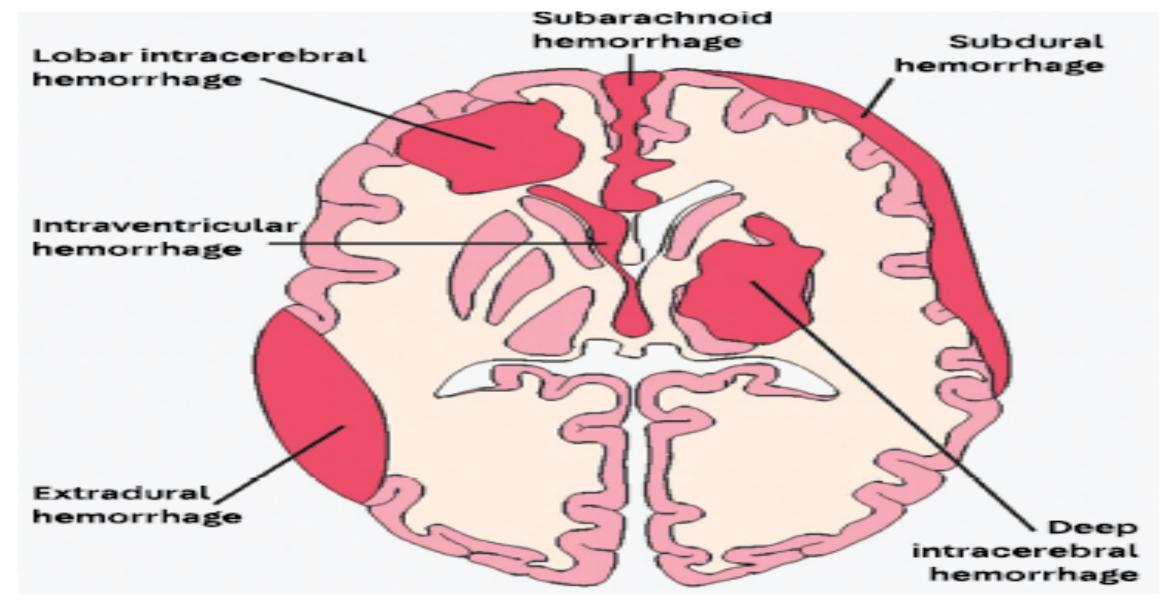


FIGURE 3-2

Locations and types of intracranial hemorrhage that may be seen on noncontrast CT.

- <u>Subarachnoid hemorrhage (SAH)</u>
- SAH is <u>often</u> due to <u>traumatic</u> injury. More <u>serious</u>, though, is SAH caused by <u>bleeding</u> from an <u>aneurysm</u> located along the circle of Willis.
- When <u>blood under arterial pressure</u> is suddenly released into the space around the brain, patients develop <u>sudden-onset</u>, <u>severe</u> <u>headache</u>.
- Often ,they <u>vomit</u> and cease what they are doing at the time of the hemorrhage.
- When the <u>intracranial pressure increases rapidly</u> or the insulae are

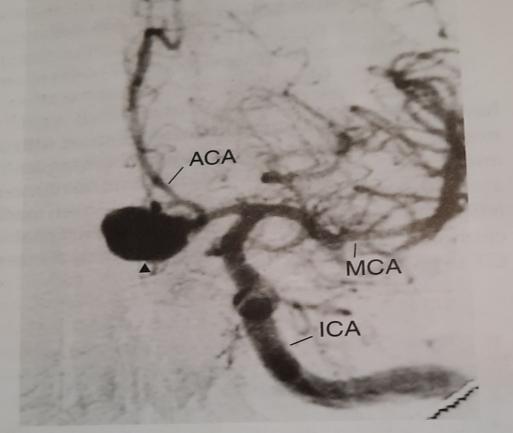


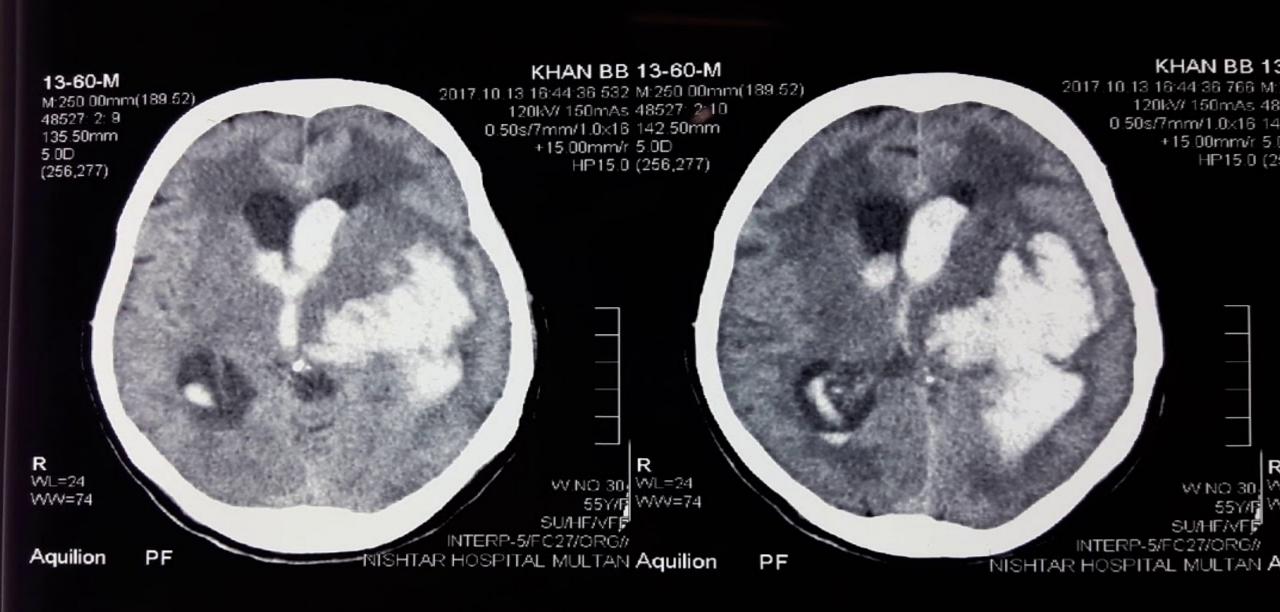
FIGURE 2-3. Conventional cerebral angiogram demonstrating aneurysm of the right middle cerebral artery (*arrow*). ACA, anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery. (Reproduced with permission from Yochum TR, Rowe LJ. Yochum and Rowe's Essentials of Skeletal Radiology. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.) <u>Treatment</u> is aimed at <u>preventing</u> the <u>rebleeding</u> and <u>vasoconstriction</u> that often follow SAH.

<u>Aneurysms</u> can be <u>clipped</u> surgically or " <u>coiled</u>" by interventional techniques.

The <u>calcium-channel blocker nimodipine</u> is used to minimize vasoconstriction and delayed brain ischemia.



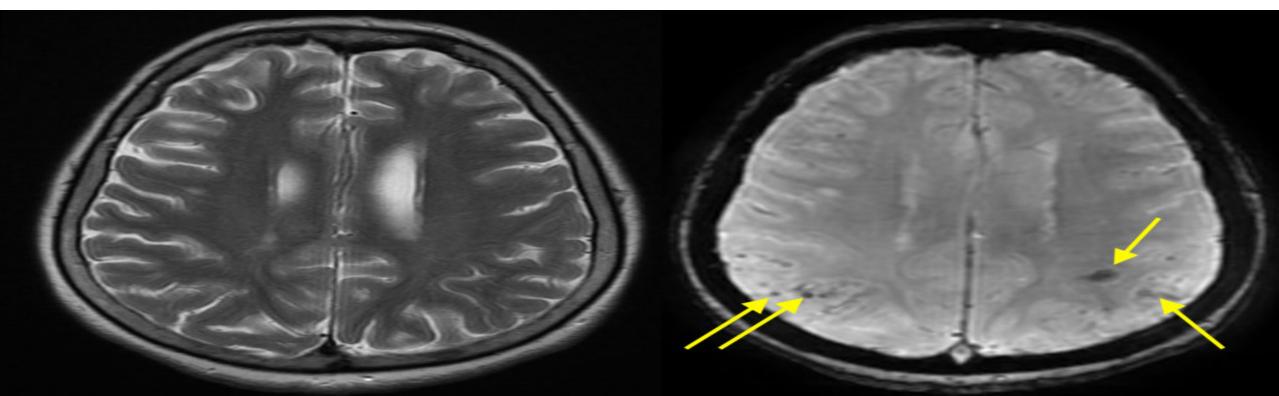
- Intracerebral hemorrhage
- ICH is <u>bleeding</u> directly into <u>brain</u> <u>parenchyma</u>.
- The <u>earliest symptoms</u> are <u>headache</u> and <u>neurologic signs</u> referable to the region in which the bleeding occurs.
- <u>Hypertension</u> (leading to <u>Charcot-</u> <u>Bouchard microaneurysms</u>) is the most common cause of ICH.
- The <u>most common locations</u> for <u>hypertensive</u> <u>ICH</u> are the **basal ganglia-internal capsule**, **caudate nucleus**, **thalamus**, **pons and**



<u>Cerebral amyloid angiopathy</u> is a cause of ICH that is more frequent in the <u>elderly</u>. and preferentially affects the <u>parietal</u> and <u>occipital lobes</u>.

<u>Trauma</u>, <u>vascular malformations</u> and <u>bleeding diatheses</u> (especially with patients who are taking <u>anticoagulants</u>) are other common causes.

<u>ICH</u> is often a <u>devastating condition</u>, and large hemorrhages are associated with <u>high</u> <u>mortality</u> rates.



Treatment involves <u>correcting</u> any <u>coagulopathy</u>.

In certain situations(particularly <u>cerebellar hemorrhages</u>), surgical <u>decompression</u> is necessary.

<u>Management</u> of risk factors for hemorrhage, specifically <u>hypertension</u>, is necessary to <u>prevent recurrence</u>.

Vascular malformations:

Variety of <u>congenital</u> and <u>acquired</u> vascular anomalies that have the <u>potential</u> to <u>bleed</u>, either within the brain (ICH) or around it.

* <u>Arteriovenous malformations</u> (<u>AVMs</u>) contain arteries that empty into arterialized veins.

These lesions contain <u>no</u> recognizable <u>normal capillary bed</u>, but <u>abnormal</u> <u>gliotic parenchyma</u> can be found between the component vessels.

In addition to causing <u>ICH</u>, AVMs may result in <u>seizures</u>.

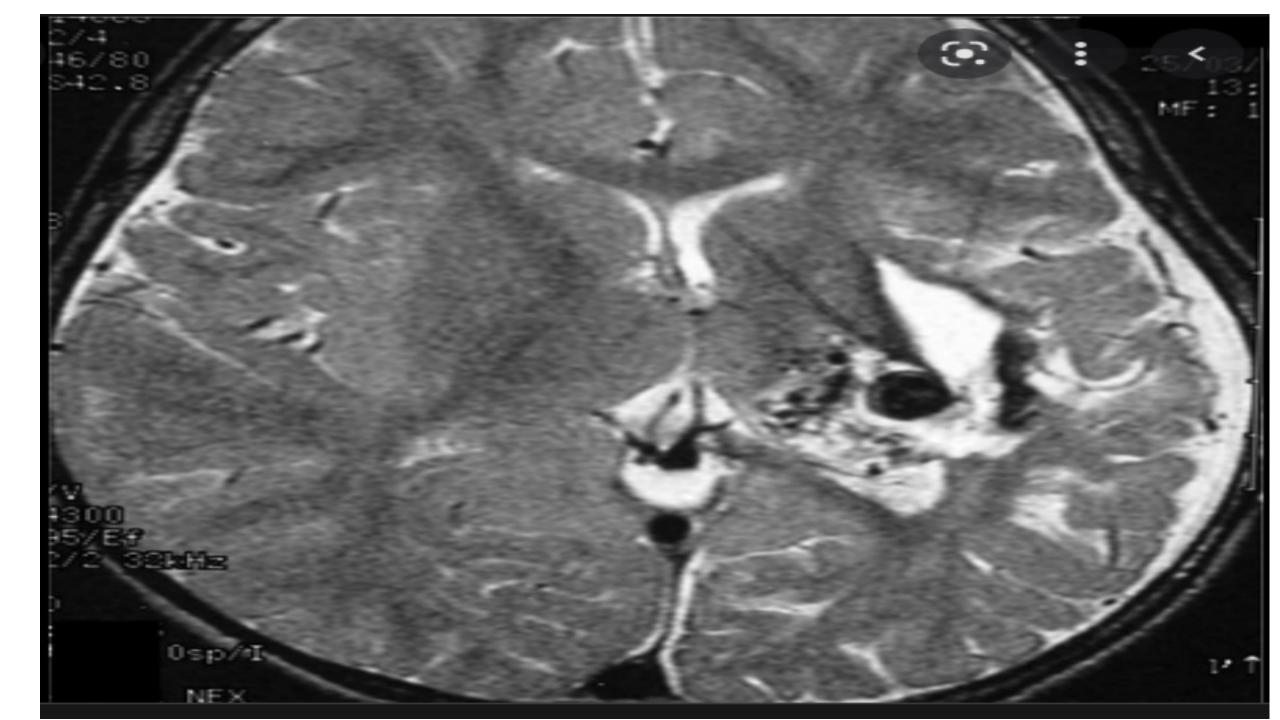
AVMs may be treated with **embolization or surgical resection**.





Medscape Reference

Arteriovenous Malformation Brain Imaging: Practice Essentials, Ultrasonography, Computed Tomography



<u>Cavernous angiomas:</u> consist of a relatively compact mass of sinusoidal vessels close together, without intervening brain parenchyma.

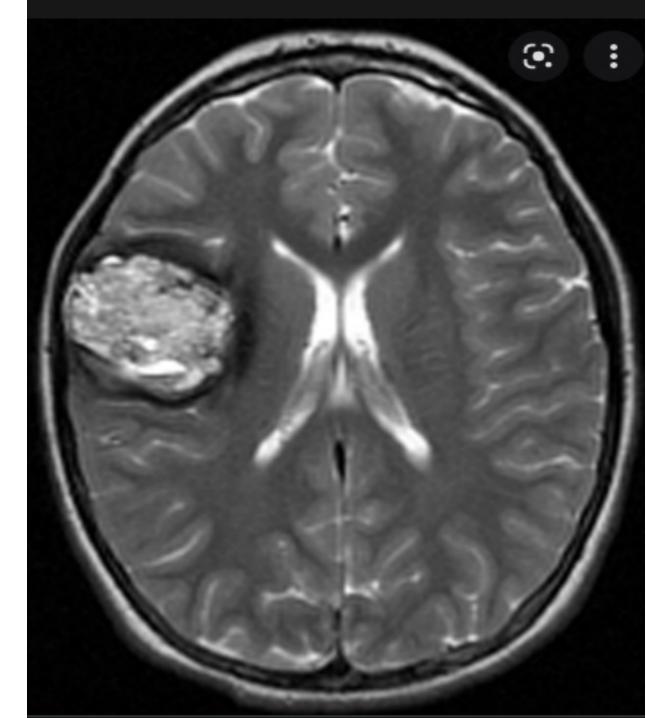
The lesions are well **<u>encapsulated.</u>**

Cavernous angiomas <u>bleed</u> or lead to <u>seizures</u>, occasionally, but are not threatening as AVMs are.

They may be followed with <u>serial</u> <u>neuroimaging</u> studies.

<u>Surgery</u> is required <u>rarely</u>.

They may require <u>antiseizure</u> drug <u>treatment</u> if recurrent seizures develop.

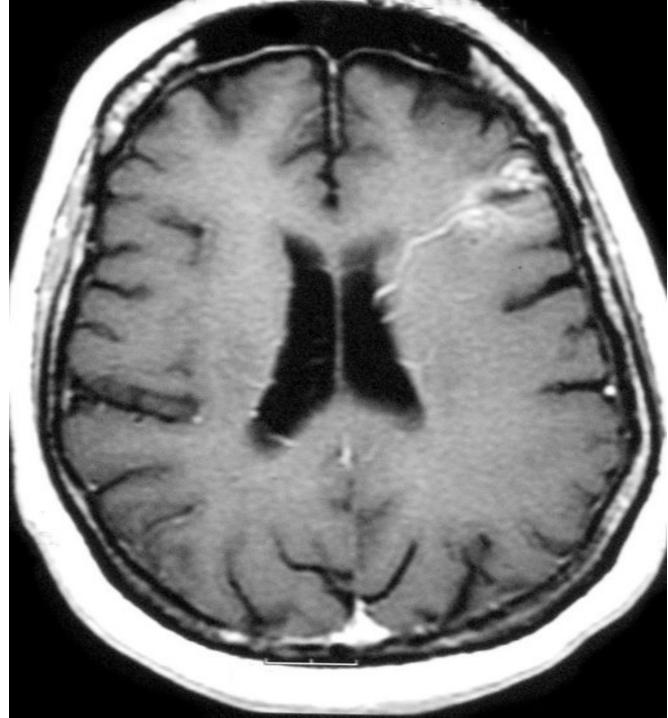


Developmental venous anomalies (**DVAs**): are composed of anomalous veins usually separated by morphologically normal brain parenchyma are the <u>most common</u> vascular malformations of the brain.

They <u>seldom hemorrhage</u> and are generally <u>not treated surgically</u> or followed with serial neuroimaging studies.

<u>Telangiectasias</u> are dilated capillaries with intervening brain parenchyma.





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