Reviewed By Tarfa Alsharidi

MED439 KING SAUD UNIVERSITY

Stroke

Objectives (regarding the Blueprint):

1. Recognize the clinical presentation of acute ischemic stroke and intracerebral hemorrhage
2. Recognize common imaging findings of ischemic and hemorrhagic stroke
3. Manage patient presenting with hyperacute and acute ischemic stroke
4. Address risk factors for ischemic and hemorrhagic stroke

439 Team Leader:
Nourah Alklaib

439 Team Member:
Afnan Almohsen

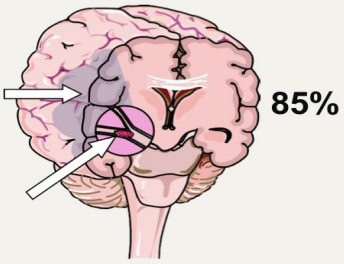
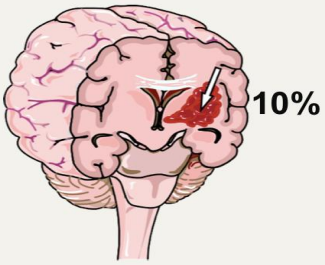
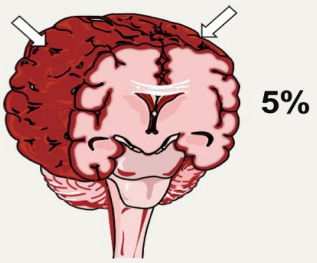
Editing File

Color Index

- Slides / Reference Book
- Doctor notes
- OnlineMeded / Amboss
- Important
- Extra

stroke

Types of stroke

Ischemic stroke	Intracerebral hemorrhage	Subarachnoid hemorrhage
 85%	 10%	 5%

Morbidity and Mortality

Ischemic Stroke

- The leading cause of long-term physical disability.
- The increase in life expectancy will increase the incidence of stroke.
- A second to only heart disease in causing death world-wide.
- According to the WHO 15 million people worldwide suffer a stroke each year.
- 30-day mortality is 12%.

High Socio-Economic

For survivors aged > 65 years:

- 50% have hemiparesis
- 30% are unable to ambulate
- 19% are aphasic
- 35% are depressed
- 26% resides in nursing home.

Ischemia Mechanism:

TOAST classification:

1) Large-artery atherosclerosis
2) Cardio-embolism
3) Small-vessel occlusion
4) Stroke of other determined aetiology*
5) Stroke of undetermined aetiology.

* For example: Hypercoagulable state, dissection, hypoperfusion, etc.

History taking (symptoms)

Onset: Acute focal neurologic deficit

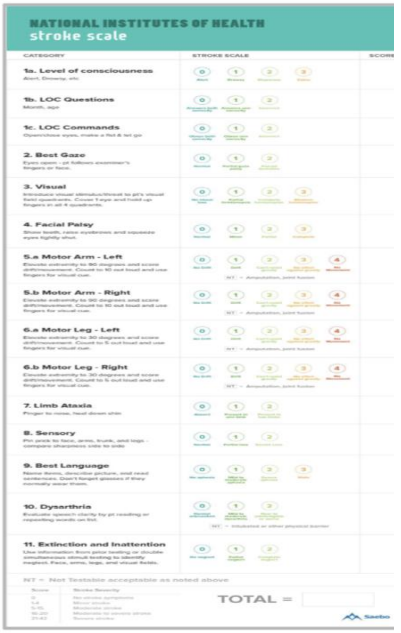
- Unilateral weakness(whole or in part)
- Unilateral sensory symptoms
- Slurred speech
- Language difficulty
- Visual symptoms (monocular, homonymous hemianopsia,double vision)
- LOC
- Difficulty swallowing
- Simultaneous bilateral weakness
- Imbalance
- Vertigo
- Crossed motor or sensory loss
- Difficult dressing, combing hair
- Visuospatial neglect

Past medical history:

- HTN
- Diabetes
- Dyslipidemia
- Smoking/ Illicit drugs
- Ischemic/ Valvular Heart Disease
- Previous TIA/ Stroke
- Any thrombo-embolism

Physical examination in stroke patient

➔ keep it neurological (focused) and quick, use **National Institution of Health Stroke Scale (NIHSS)**, the higher the score, the more severe the stroke is.



(Make sure you assess the NEW symptoms. Not residual symptoms from old strokes)

Treat if NIH score is 4+

ABC (Sometimes they add D for dextrose)

General examination

- e.g. in Pain, distressed, jaundiced, pale or febrile (suspect IE)

Pulse

- (A.fib. ⟶ irregular)

BP

- Will be high (in both ischemic and hemorrhagic strokes)

Hand

Listen for heart murmur, carotid bruits.

Cortical infarcts

are suspect based on the presence of:

- language impairment (If dominant hemisphere is affected)
- neglect or anosognosia (If non-dominant hemisphere is affected)
- graphesthesia or stereognosis
- visual field impairment

Neurological Examination

Cognitive assessment: Orientation (time, place and person),Attention (serial 7s), Language,Memory (recall and retrograde), Executive,Praxis (wave, salute), Visuospatial (clock drawing)

Tone

- **decreased** on side of weakness early on, **later on increased**

- Cranial nerves examination
- Motor examination
- Sensory examination
- Coordination(fingertonoseandheeltochin)
- Gait

Note: Spasticity and hyperreflexia take time to develop

Reflexes

hyperreflexia on side of weakness, with upgoing toe.

Differential diagnosis :

- Migraine aura
- Seizure
- Multiple sclerosis
- Metabolic (hypoglycemia)
- Brain tumor
- Syncope Conversion/somatization

Investigations

1- CBC

2- coagulation profile

- PT
- PTT
- INR

3- chemistry

- fasting glucose
- HbA1c
- Lipids

4-imaging (immediate investigation)

- **CT scan**
  - **CT scanning is the mainstay of emergency stroke imaging. It allows the rapid identification of intracerebral bleeding and stroke 'mimics' (i.e. pathologies other than stroke that have similar presentations), such as tumors. it is done to exclude hemorrhage and early infarct**
  - **non-contrast CT is the only way to differentiate between ischemic and hemorrhagic strokes**
- MRI

5- vascular imaging (later investigation)

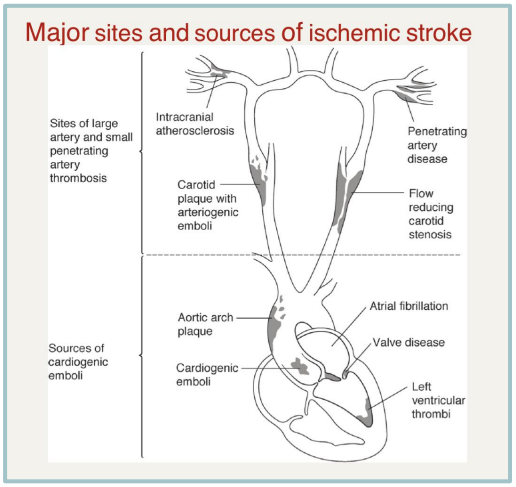
- carotid US
- CTA
- MRI
- Cerebral angio

6- cardiac workup

- ECG
- Echo (TTE or TEE)
- Holter

7- in specific cases

- Hb
- Electrophoresis
- Hypercoagulable workup
- CTD screen
- HIV and syphilis



Further Investigations:Look for the source

Recognize the clinical presentation of acute ischemic stroke and intracerebral hemorrhage

The Clinical presentation of ischemic stroke (Depending on the site of occlusion) :

Clinically we can't differentiate between ischemic and hemorrhagic strokes.

- **Middle Cerebral Artery (MCA) occlusion (Total anterior circulation syndrome (TACS)**

Common cause:

- Embolism from heart or major vessels

Symptoms:

Combination of:

- Hemiparesis: Arm + face (UE) more than leg weakness (LE)
- Hemisensory loss
- Higher cerebral dysfunction:
  - **Aphasia** if affecting the dominant (left) hemisphere.
  - Neglect if affecting the non-dominant hemisphere.
- Contralateral homonymous hemianopia

Types of aphasia	
1. Broca's (expressive, anterior) aphasia:	• **Damage in the left inferior frontal lobe causes reduced speech fluency with relatively preserved comprehension.** • **The patient makes great efforts to initiate language.** • **Patients who recover say they knew what they wanted to say, but could not get the words out.**
2. Wernicke's (receptive, posterior) aphasia:	• **Left temporo-parietal damage leaves fluency of language but words are muddled.** • **This varies from insertion of a few incorrect or non-existent words into speech to a profuse outpouring of jargon (i.e. rubbish with wholly non-existent words).** • **Severe jargon aphasia is bizarre and often mistaken for psychotic behaviour.** • **Patients could neither stop speaking nor understand speech.**
3. Nominal (anomic, amnestic) aphasia:	• **difficulty naming familiar objects (early feature in all types of aphasia)**
4. Global (central) aphasia:	• **combination of the expressive problems of Broca's aphasia and the loss of comprehension of Wernicke's with loss of both language production and understanding.** • **Due to widespread damage to speech areas, the commonest aphasia after a severe left hemisphere infarct. Writing and reading are also affected.**

Recognize the clinical presentation of acute ischemic stroke and intracerebral hemorrhage

The Clinical presentation of ischemic stroke (Depending on the site of occlusion) :

- **Anterior Cerebral Artery (ACA) occlusion**

  Symptoms:

  - **Weakness LE more than UE (Opposite to MCA)**
  - **Emotional disturbance.**
  - **Visual field is spared**

- **Branch of MCA or ACA occlusion (Partial anterior circulation syndrome (PACS))**

  Common cause:

  - **Embolism from heart or major vessels Symptoms Could be:**
  - **Isolated motor loss (e.g. leg only, arm only, face)**
  - **Isolated higher cerebral dysfunction (e.g. aphasia, neglect)**
  - **Mixture of higher cerebral dysfunction and motor loss (e.g. aphasia with right hemiparesis)**

- **Internal Carotid occlusion**

  Symptoms

  - **Above and ophthalmic.**

- **Posterior Cerebral Artery (PCA) occlusion**

  Symptoms

  - **Vision - visual field (homonymous hemianopia sparing the center)**
  - **memory**

- **Vertebrobasilar (posterior circulation stroke)**

  Common cause:

  - **Embolism from heart or thrombosis in situ leading to occlusion of vertebral, basilar, or PCA**

  Symptoms

  - **Cranial nerve syndrome with crossed motor**
  - **Crossing weakness or numbness (L arm and R leg for example)**
  - **cerebellum (cerebellar syndrome): Ataxia, vertigo, vomiting**
  - **altered LOC. (often misdiagnosed as seizures or intoxication)**
  - **homonymous hemianopia**

- **Midbrain**

  Symptoms

  - **CN III: signs of complete CN III palsy:**
    - **dilated pupil (if the left midbrain is affected → dilated and abducted left eye)**
    - **Unilateral complete ptosis (levator weakness)**
    - **Eye deviated down and out (unopposed lateral rectus and superior oblique**
  - **Weber's syndrome: Ipsilateral IIIrd nerve palsy with contralateral hemiplegia is due to a unilateral infarct in the midbrain. Paralysis of upward gaze is usually present.**

Recognize the clinical presentation of acute ischemic stroke and intracerebral hemorrhage

The Clinical presentation of ischemic stroke (Depending on the site of occlusion) :

- **Pons**

  Symptoms
  - **CN V → Sensory: facial numbness, anterior ⅔ of the tongue sensory loss. Motor: weakness of jaw movements.**
  - **CN VI → lateral rectus palsy (horizontal diplopia when looking into the distance, maximal when looking to the side of the lesion.)**
  - **CN VII → facial weakness.**
- **Medulla**

  Symptoms
  - **CN VIII → vertigo, hearing loss.**
  - **CN IX, X → dysphagia.**
  - **CN XII → tongue weakness.**
- **Small penetrating arteries (Lacunar syndrome)**

  Common cause:
  - **thrombosis in situ of small penetrating arteries.**

Symptoms: unlike MCA and ACA ischemia, in lacunar syndrome legs, arms and face will be affected to the same degree, could be:
  - **pure motor stroke affecting two limbs**
  - **Pure sensory stroke**
  - **Sensory motor stroke**
- **Note: no higher cerebral dysfunction (no cortical involvement, that leads to peripheral weakness when present) or hemianopia**

Four Major Stroke Syndromes:

- for Rapid Recognition in the ED
- All Occur Suddenly in Stroke Patients

Left (Dominant) Cerebral Hemisphere	Right (non-dominant) Cerebral hemisphere	brainstem	Cerebellum
- Aphasia - L gaze preference - R visual field deficit - R hemiparesis - R hemisensory loss	- Neglect (Lt hemi-inattention) - R gaze preference - L visual field deficit - L hemiparesis - L hemi-sensory loss	- Neglect (Lt hemi-inattention) - R gaze preference - L visual field deficit - L hemiparesis - L hemi-sensory loss	- Truncal = gait ataxia - Limb ataxia

Note: The dominant cerebral hemisphere is the side that controls language function.

Recognize the clinical presentation of acute ischemic stroke and intracerebral hemorrhage

The Clinical presentation of hemorrhagic stroke :

At the bedside, there is no entirely reliable way of distinguishing between haemorrhage and thromboembolic infarction. Both produce stroke. Intracerebral haemorrhage tends to be dramatic with severe headache. It is more likely to lead to coma than thromboembolism.

- Alteration in level of consciousness (approximately 50%).
- Nausea and vomiting due to increase ICP (approximately 40-50%).
- Headache (approximately 40%)
- Seizures (approximately 6-7%)

Focal neurological deficits:

➔ **depending on the location:**

Putamen	Contralateral hemiparesis, contralateral sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, aphasia, neglect, or apraxia.
Thalamus	Contralateral sensory loss, contralateral hemiparesis, gaze paresis, homonymous hemianopia, miosis, aphasia, confusion Any focal sensory loss, think of the thalamus
Lobar	Contralateral hemiparesis or sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, abulia, aphasia (if affecting the left side), neglect (if affecting the right side), or apraxia.
Caudate nucleus	Contralateral hemiparesis, contralateral conjugate gaze paresis, or confusion.
Cerebellum	Ataxia on the same side

Transient ischemic attack (TIA):

- Sudden transient focal neurological deficit
- Symptoms lasting less than 24 hours (less than one hour) —-> complete resolution
- Symptoms maximal at onset
- Normal CT/MRI of brain

Risk for subsequent Stroke:

Among TIA pts who go to ED:

- 5% have stroke in next 2 days
- 25% have recurrent event in next 3 months

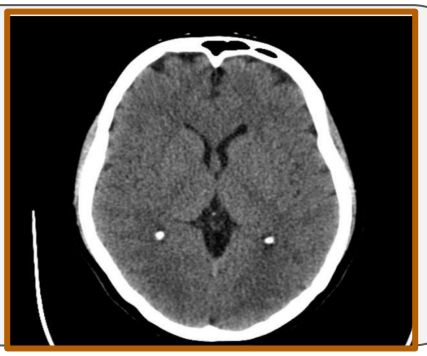
Stroke risk decreased significantly with proper medical therapy

Recognize common imaging findings of ischemic and hemorrhagic stroke

ischemic stroke:

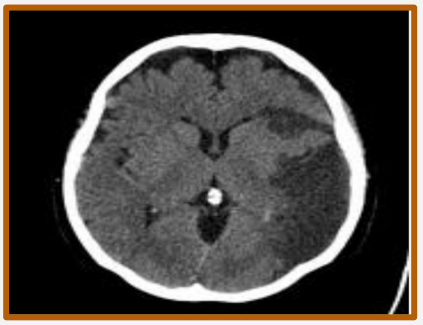
➔ **CT brain at ER**

Normal



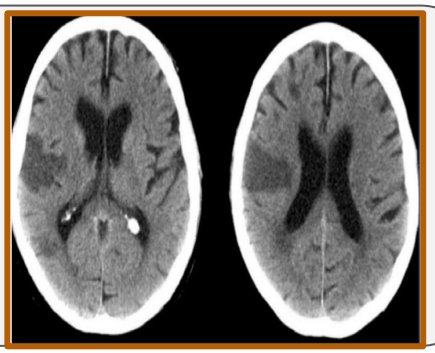
➔ **CT brain of subacute stroke**

Left hemisphere



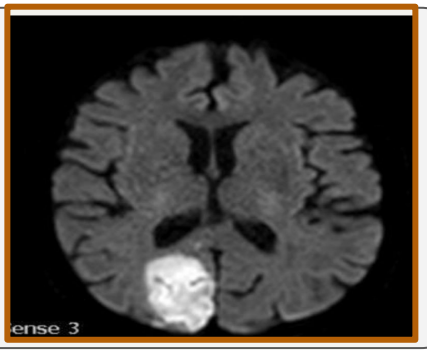
➔ **Acute wedge-shaped embolic stroke**

Wedge infarcts are typical of cardioembolic strokes



➔ **DWI (MRI) for acute Rt PCA**

Reach central line posteriorly = PCA

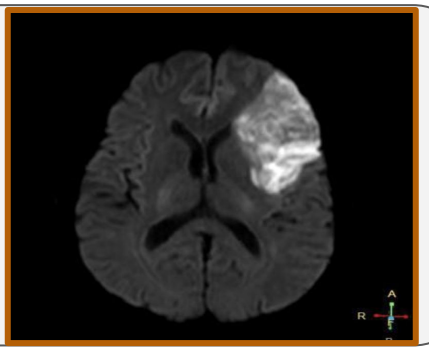


Recognize common imaging findings of ischemic and hemorrhagic stroke

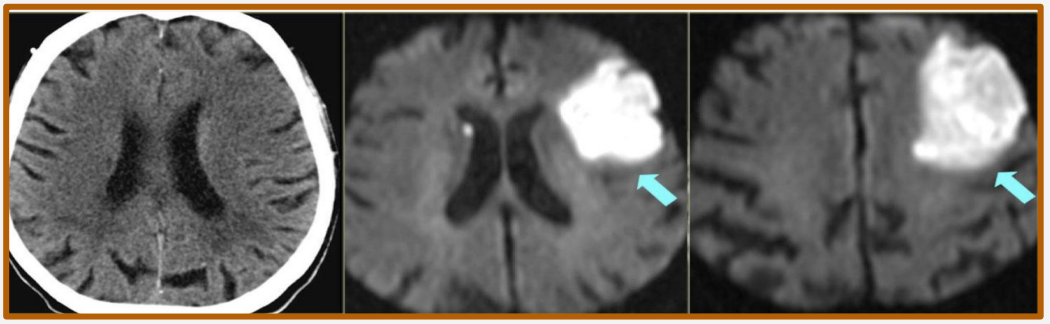
ischemic stroke:

➔ Frontal lobe ≠ ACA !!

- Left
- Incompe MCA

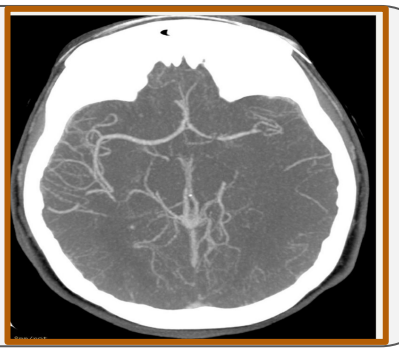


➔ MRI vs CT for same pt:



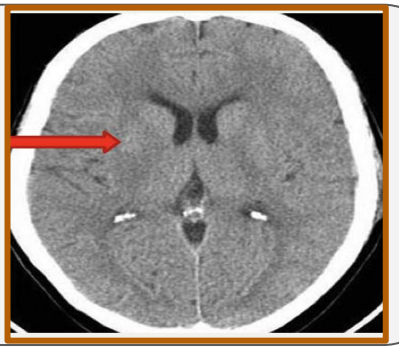
➔ CT Angio:

Left MCA occlusion



➔ Acute ischemic change in CT

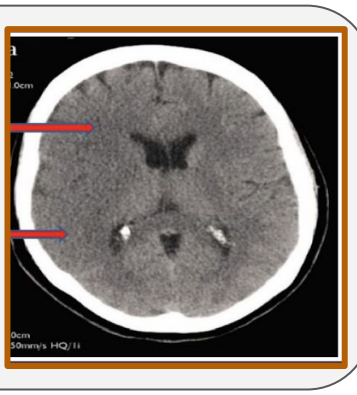
Obscuration of lentiform nucleus



Recognize common imaging findings of ischemic and hemorrhagic stroke

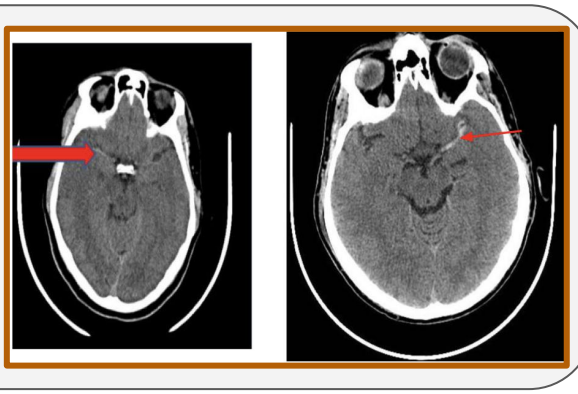
➔ Acute Ischemic changes in CT

- Loss of gray-white matter differentiation: You can't Identify the border between gray and white matter
- Sulcal effacement: The brain sulci are pushed into the skull because of cerebral edema causing them to flatten



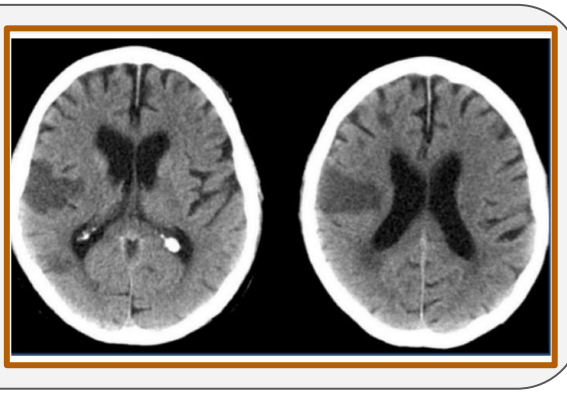
➔ Hyperdense MCA sign

- The affected MCA appears hyper dense because of the thrombus

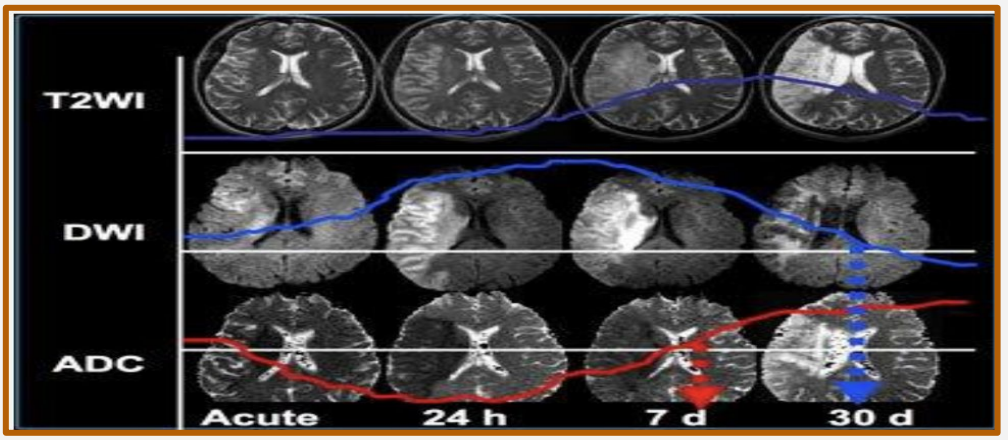


➔ Acute wedge-shaped embolic stroke

- Wedge infarcts are typical of cardioembolic strokes



➔ Acute ischemic changes in MRI

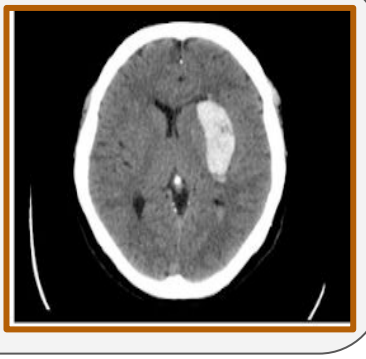


Recognize common imaging findings of ischemic and hemorrhagic stroke

Hypertensive hemorrhage

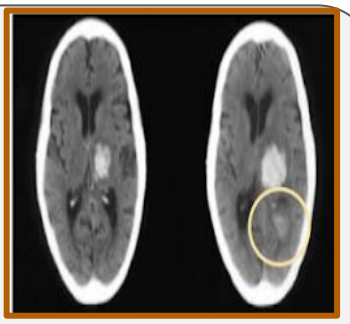
➔ Putamen hemorrhage

- hemorrhage is hyperdense in CT scan.
- A typical location for hypertensive hemorrhage.
- Hemorrhage is compressing the ventricles (Mass effect)
- Symptoms will be weakness in the contralateral side



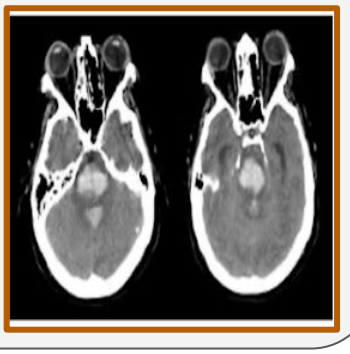
➔ Thalamic hemorrhage

- picture shows a left thalamic haemorrhage with ventricular expansion
- Patient presents with numbness and decreased sensation on the right side



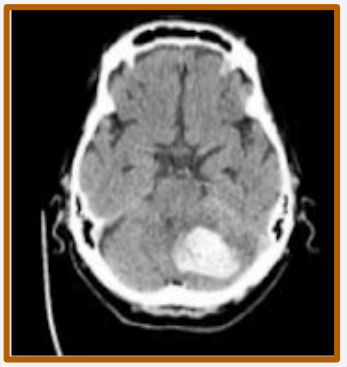
➔ Pontine

- hemorrhage in the bilateral pontine area.
- very poor prognosis (brainstem hemorrhage)



➔ Cerebellar hemorrhage

- Easy surgery, good prognosis
- left cerebellar hemorrhage which is very close to the brainstem so neurosurgeon must interfere and evacuate→ good outcome. if we didn't interfere it will push the brainstem → herniation → death.
- patient will present with ataxia on the left side.



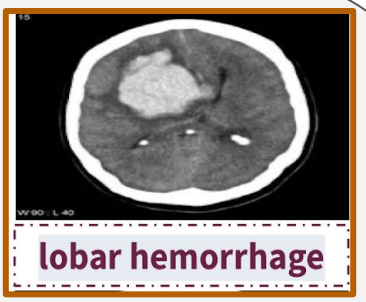
Recognize common imaging findings of ischemic and hemorrhagic stroke

Hypertensive hemorrhage

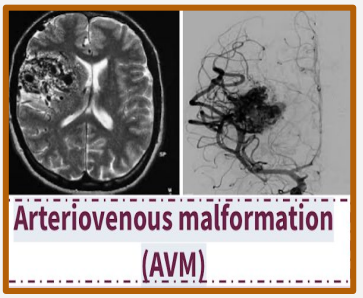
➔ Lobar hemorrhage

It is the 5th most common cause of hypertinstive haemorrhage, but it could also be caused by:

- **Tumor with ICH:**
  - lobar hemorrhage
  - Hemorrhage in a tumor:
  - Very hyperdense area (haemorrhage) next to little hyper-dense area (tumor).
- **AVM** :
  - The artery will drain directly to the vein without passing into venuoles and capillaries which will cause dilatation and hemorrhage, Treatment is by embolization of artery
  - MRI showing collection of worms → typical appearance of AVM


lobar hemorrhage


Tumor with ICH

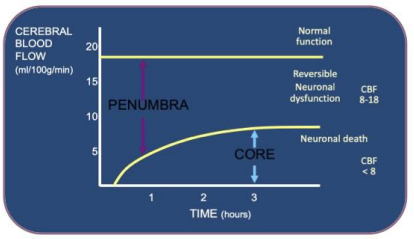

Arteriovenous malformation (AVM)

Manage patient presenting with hyperacute and acute ischemic stroke

Management of ischemic stroke

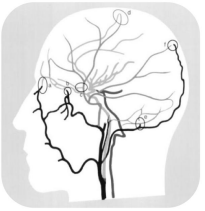
Stroke treatment:

- primary stroke prevention
- Acute stroke treatment
- Secondary stroke prevention
- Stroke rehabilitation

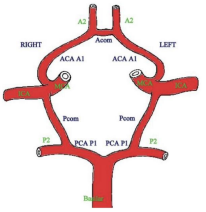


Stroke penumbra:
The target of acute stroke treatment

Penumbra suppliers:



External



Internal

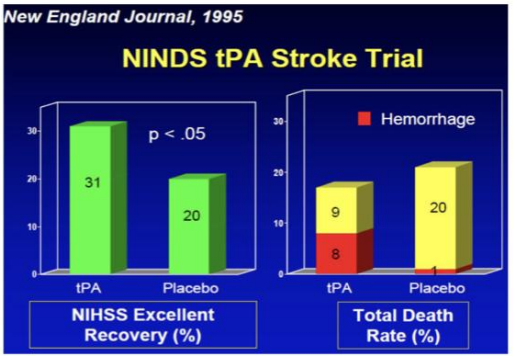
Acute ischemic Stroke Management

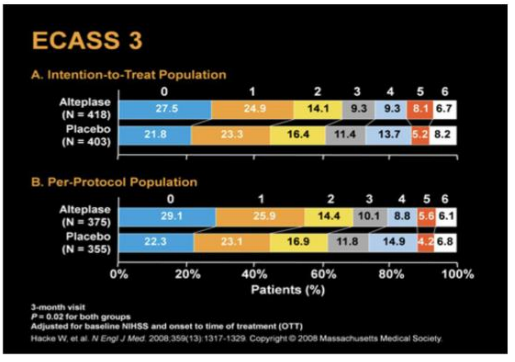
Modalities of Acute Stroke Treatment:

1. IV t-PA (standard)

Inclusion criteria	Exclusion criteria
• Clinical Dx of stroke • Stroke onset < 270 minutes • Age is > or = 18	• Intracranial Hge in imaging or clinical presentation suggests SAH • Active/ recent internal bleeding or on warfarin with INR > 1.7 or platelets < 100K • Serum Glucose <50 or > 400 • Systolic BP > 185 or diastolic >110 • Recent MI (3/52) • Recent (2/52) major surgery or trauma • Recent arterial puncture at noncompressible site • Others (see NINDS protocol)

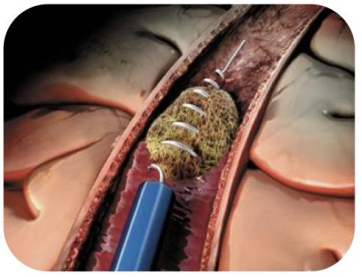
- Stroke onset: timing of first neurological deficit OR last time pt was seen well
- TIA: has to end with complete neurological recovery
- IV t-PA (alteplase)
  - 0.9mg/kg to a maximum of 90mg
  - 10% bolus over 1 minute then infuse rest over 60 minutes
  - Hold infusion and re-evaluate the pt in case of HTN (S>185,D>110), sudden headache, or sudden reduction in LOC
- outcome with IV t-PA :





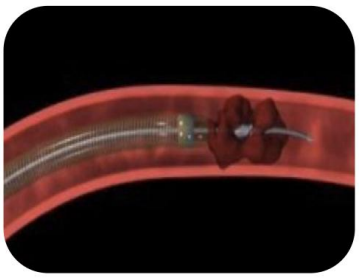
Acute ischemic Stroke Management

Modalities of Acute Stroke Treatment:



2- Endoarterial Mechanical Disruption

- Merci Retriever
  - first FDA approved device
  - Increased recanalization rate and secondary clinical outcome
  - when used for large cerebral arteries
- Penumbra system:
  - FDA approved
  - It does: clot suctioning
  - Similar rates of recanalization and clinical outcomes to Merci retriever
- 3rd Generation of devices:
  - Solitaire Device: Solitaire was superior to Merci in Swift trial
  - Trevo retriever: Trevo was superior to Merci in Trevo II trial



3- Endoarterial thrombolysis:

- Combined IA and Mechanical disruption
- General recommendation:
  - For M1 (MCA) clot
  - For Basilar artery clot
  - In certain cases where IV t-PA can not be given e.g. patient is on warfarin or recent MI
- Limitations:
  - Time (should not delay IV t-PA initiation)
  - Expertise
  - Costs ?

Key time intervals

- Perform an initial patient evaluation within 10 minutes of arrival in the ER
- Notify the stroke team within 15 minutes of arrival
- Initiate a CT scan within 25 minutes of arrival
- Interpret the CT scan within 45 minutes of arrival
- Ensure a door-to-needle time for IV rt-PA within 60 minutes from arrival

Recommendation strategies

- Advance hospital notification by EMS
- Rapid triage and stroke team notification
- Single call activation system
- Rapid access to CT and rapid interpretation
- Rapid laboratory testing (point of care)
- Mix t-PA a head of time
- Team-based approach

Barriers for acute stroke therapy

- Late patient presentation to ER (In USA; only 30% present within t-PA window)
- Poor stroke recognition and delay ed triage at ER (mainly for unusual stroke presentations)
- Lack of appropriate infrastructure
- Presence of a contraindication
- Difficulty in getting patient's or family's verbal consent

Workup for secondary stroke prevention:

- Vascular Imaging: carotids, vert's, COW
  - (US – CT angio – Conventional Angiogram)
- 72 hour Holter Monitor (can be repeated or extended)
- Echocardiogram: TTE vs TEE
- Thrombophilia: Young pts with Rt – Lt shunt

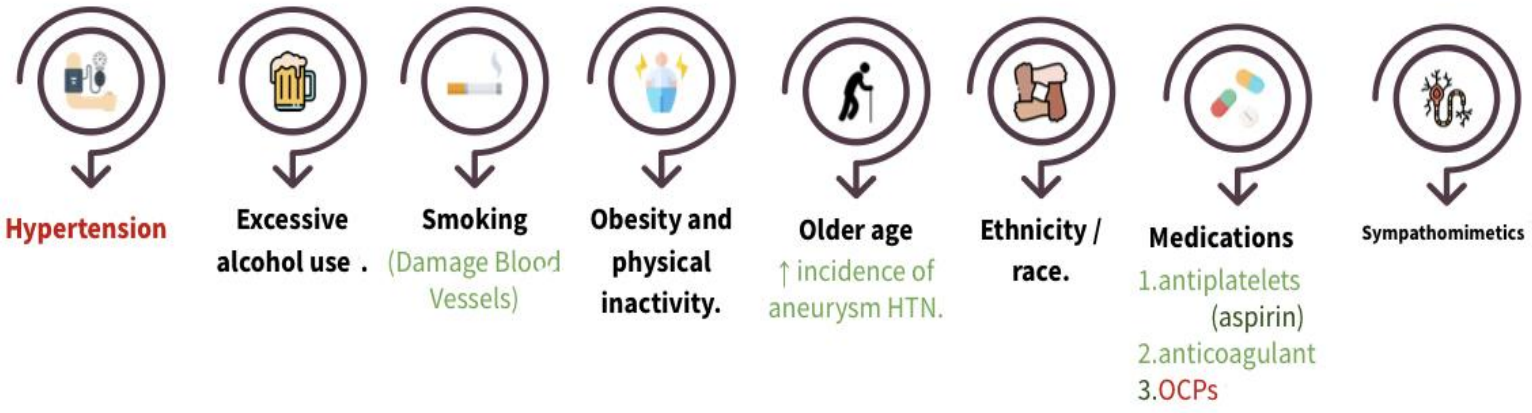
- Compare work up for lacunar stroke vs. embolic stroke.

Address risk factors for ischemic and hemorrhagic stroke

1- Risk Factors for ischemic stroke

Modifiable	Non Modifiable
- Hypertension. (Most important one) - Diabetes mellitus. - Hyperlipidemia. - Cardiac diseases (particularly Atrial fibrillation, CHF, IE). - Stroke, TIA, and carotid artery stenosis. - Smoking/Illicit drugs. - Sedentary lifestyle. - Stress, type A personality. - Air pollution - Obstructive sleep apnea - Polycythemia - Any thrombo-embolism	- Age, risk after 60 double with each decade. - Sex, generally men are more prone to stroke. However, young women are at higher risk than men due to pregnancy, hormonal changes. - Ethnicity (African americans) - Genetic determinants (e.g. sickle cell disease) - Previous vascular events (MI, stroke, PVD)

2- Risk Factors for Hemorrhagic stroke

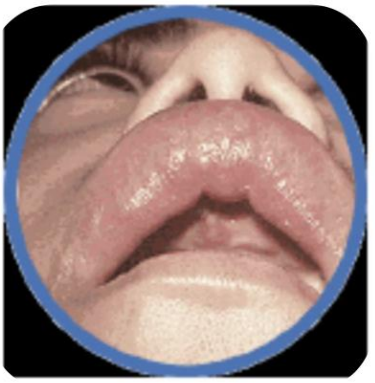


Cases by the doctor

➔ Case 1

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

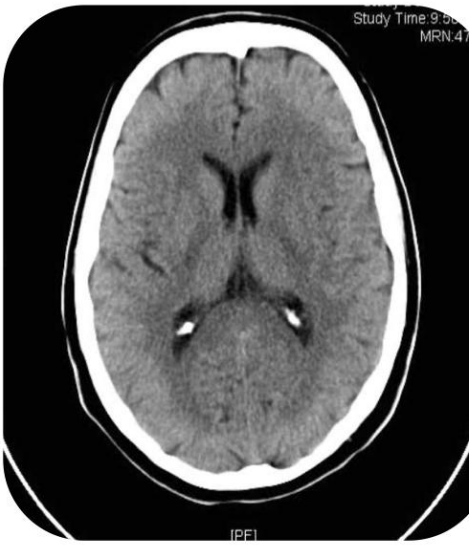
Oro-lingual angioedema



➔ Case 2

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

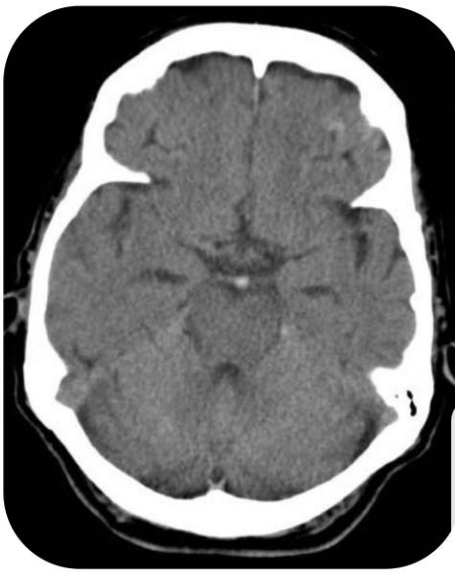
Acute Left MCA (upper division) ischemic stroke with (N) CT brain



➔ Case 3

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

Acute Basilar artery stroke



MedEd notes

Neurology [STROKE]

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Intro

Stroke is high impact; it's the **3rd leading Cause of Death** and a leader in lingering **morbidity**. Not only that, but it's a **preventable** and largely **time-sensitive** disease.

Etiologies

Stroke is a **brain attack** - that is, an ischemic injury to the brain parenchyma. This can happen in three ways. 1) **Emboli** may form on diseased valves, in the left atrial appendage during Afib, or on a carotid dissection/stenosis. The emboli will then travel to a smaller vessel where it gets lodged in the lungs and occludes flow. 2) **Thrombi** may form in a vessel. This is the same pathogenesis as atherosclerotic plaque in the heart, predisposed by CAD/PVD/HTN/Atherosclerosis. Of the etiologies, 3) **Hemorrhage** has the worst prognosis. Both **subarachnoid** and **intraparenchymal hemorrhage** (discussed elsewhere) are considered "brain bleeds" - usually a product of hypertension. In this case blood does flow, just into the parenchyma. It's an irritant, decreases perfusion to the distal brain, and is a potential mass effect.

Presentation

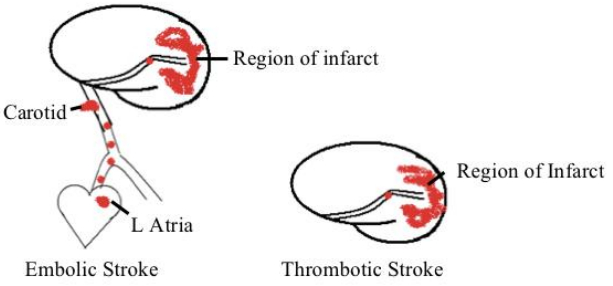
The clinical presentation doesn't correlate with etiology. The tell-tale sign of any stroke is **Focal Neurologic Deficit** of **Acute Onset**. The location of infarct correlates to the defect. Thus a sudden onset loss of **motor**, **speech**, **sensation**, or **level of consciousness** prompts investigation. It becomes important to revisit arterial supply and vascular distribution. The **posterior circulation** is made of the **vertebral arteries** that come to form the **basilar artery**. Lesions here cause **cerebella dysfunction**, **Δ in Mental Status**, and **blindness**. The **anterior circulation** is comprised of the **anterior** and **middle cerebral arteries**. These feed the speech centers, motor strips, and sensory strips. Recall the homunculus.

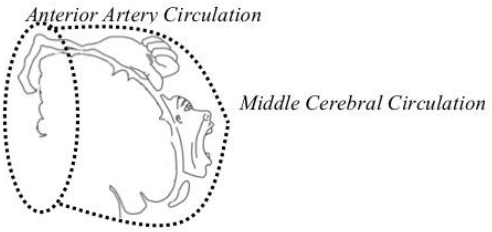
Certain elements of presentation may help beyond simply location. For example, the patient with **Afib** has an increased likelihood of embolism. **Painful neck pulsations** and a patient who **grabs her neck** are indicative of a carotid dissection. Patients who've experience **TIAs** (FND < 24 hrs) in the past are highly likely to have a thrombotic event.

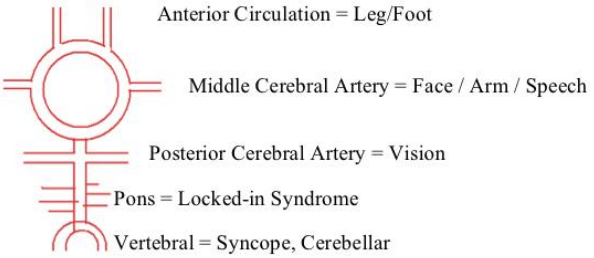
Diagnosis and Workup

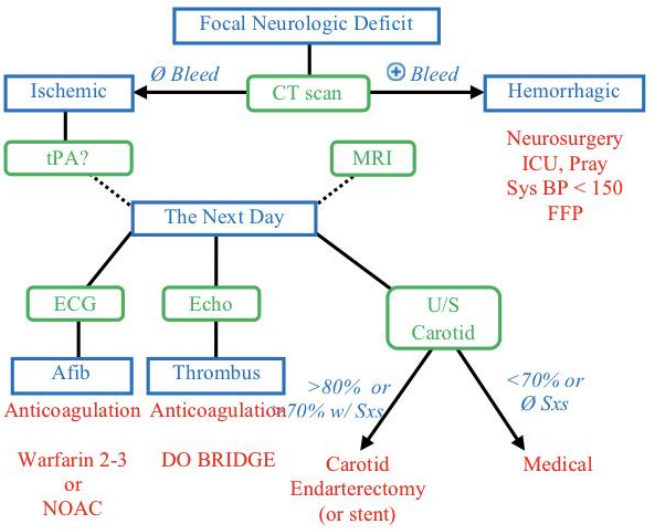
Regardless of presentation, in the **acute phase** of a stroke (**within 30 minutes** of presentation and **within 6 hrs of symptoms**) the goal is **rapid identification** and intervention if possible. The first thing to do is a **CT scan without contrast** to rule out hemorrhage. At this point therapeutic intervention is considered. After the initial presentation (usually on day 2), additional testing may be done. **Transesophageal Echo** assesses the cardiac valves, **carotid duplex** for carotid stenosis, **MRI** to look at areas of ischemia (CT scan is not to diagnose CVA, but to rule out hemorrhage), and **CT angiogram** for blood vessels of the brain.

Etiology	*Type*	*Examples*
Ischemic	Embolus	Afib, Carotid Stenosis
	Thrombotic	CAD/PVD, Atherosclerosis, HTN
Hemorrhagic	Hemorrhagic	SAH, Intraparenchymal, HTN









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Treatment Options

Treatment is broken into the "treat right now" and "secondary prevention."

If they present **within 4.5 hours** of symptom onset (**3 hours of diabetic**) they can be considered for tPA. It's the LAST TIME SEEN NORMAL. Which means someone who went to sleep normal and woke up with a stroke starts at the time they went to sleep. The big to-do for stroke is **tPA**, the **clot buster**. It has greatly restricted use but can actually rescue ischemic tissue and preserve the **penumbra**. The risk of transforming an ischemic stroke into a hemorrhagic one is high so caution must be used. Even if tPA isn't used the stroked brain will die. However, optimally controlling **oxygen >95%**, **tight glucose control 60-100**, and **Blood Pressure** (permissive hypertension) will allow the at-risk penumbra to recover.

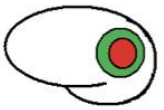
Most strokes occur and it's too late - nothing can be done for them - so preventing the next one becomes crucial. **Aspirin** is the mainstay treatment unless the patient gets/has an allergy. If there is an aspirin allergy, use **clopidogrel**. If there's a stroke on aspirin, add a second agent - usually **aspirin + dipyridamole.** Newer literature is suggesting the use of aspirin + clopidogrel, but it hasn't hit the boards yet.

For future stroke prevention, risk factor control is essential. This is what **medical management** means. Risk factor to medical management is as follows:

- Dyslipidemia: High-potency statin
- Diabetes: A1c < 7% (orals or insulin)
- HTN: Ace-I and other agents
- Smoking: cessation counseling
- Antiplatelet: ASA first stroke, Add Second agent repeat on ASA (Dipyridamole vs Clopidogrel controversial)

In the condition where there's **carotid stenosis 80%** or **70% and symptoms**, a carotid endarterectomy may be performed. While a **carotid stenting** can be performed, it should be reserved only for those patients who CANNOT undergo surgery. Stenting and endarterectomy should **never** be performed in the acute setting; wait 2 weeks or more (going during a stroke worsens the stroke).

If there's **Afib** they need to be on **anticoagulation**. It doesn't matter whether you use **warfarin** (with an INR goal of 2-3) or the **novel oral anticoagulants** except that NOACs can't be used on valvular afib. **Neither needs a bridge**. Often, initiation of warfarin requires a heparin bridge. Afib is the one time where you definitely do **NOT NEED TO BRIDGE** (unless there is another indication such as a mechanical valve).



Penumbra (can be saved)
Infarct (can't be saved)

tPA	*ASA*
Thrombotic / Embolic only Never if Brain Bleed Ever Sxs onset < 3 hrs Ø if surgery in 21 days Ø if head trauma	Primary med for secondary prevention
Afib – Warfarin/NOAC	***Clopidogrel***
Embolic Stroke Prevention CHF HTN Age > 65 DM Stroke (worth 2)	Used when the patient can't tolerate ASA or when ASA fails. **probably going to be ASA + Clopidogrel in the future*

Test	*Notes*	*Timing*
CT scan NO contrast	Do it at presentation, rule out hemorrhage but is Ø useful to diagnosis CVA until days later	<30 min
ECG	Afib or not	Immediately
U/S Carotid	Carotid Visualization to rule out or rule in carotid artery stenosis and dissection	2nd Day
2D Echo (TEE +/-)	Visualize heart valves, especially in Afib, r/o source of embolism. TTE ↓Se, Easy TEE ↑Se, Difficult	2nd Day
CTA/MRA (not needed)	Visualize blood vessels, Ø require angiogram	2nd Day
MRI	Visualize areas of ischemia, track resolution, confirm diagnosis if unsure	24 hrs

Treatment	*Notes*	*Chronic*
tPA	Ischemic stroke < 3 hrs + DM Ischemic stroke < 4.5 hrs + not DM Contraindicated with ICH, Bleeding, recent surgery or trauma	N/A
Heparin	Never	Never
Antiplatelet	1st Stroke: ASA Repeat Stroke: ASA + Dipyridamole ASA Allergy: Clopidogrel	Forever
Warfarin (NOAC)	Acute: Never Chronic: Afib with CHADS2 score 2+	Forever
BP	Ischemic no tPA <220/<120 Ischemic with tPA <180/<105 Hemorrhagic Stroke < 150 / 80	<140/<80 Ace-i
Diabetes	<140	A1c<7
Statin	High-POtency	High-potency

Lecture Quiz

1:D / 2:A / 3:A / 4:C

Q1: A 73-year-old patient with a stroke experiences facial drooping on the right side and right-sided arm and leg paralysis. When admitting the patient, which clinical manifestation will the nurse expect to find?

A. Impulsive behavior
B. Right-sided neglect
C. Hyperactive left-sided tendon reflexes
D. Difficulty comprehending instructions

Q2: A 35-year-old previously healthy woman suddenly develops a severe headache while lifting weights. A minute later she has transient loss of consciousness. She awakes with vomiting and a continued headache. She describes the headache as "the worst headache of my life." She appears uncomfortable and vomits during the physical examination. Blood pressure is 140/85, pulse rate is 100/min, respirations are 18/min, and temperature is 36.8° C (98.2°F). There is neck stiffness. Physical examination, including careful cranial nerve and deep tendon reflex testing, is otherwise normal. Which of the following is the best next step in evaluation?

A. CT scan without contrast
B. CT scan with contrast
C. Cerebral angiogram
D. Holter monitor

Q3: A 79-year-old man is admitted with left hemiparesis. CT reveals a middle cerebral artery infarct. What is his most significant risk factor for stroke?

A. Hypertension
B. Smoking
C. Family history
D. Diabetes
E. Cholesterol

Q4: A 71-year-old right-handed male is brought in by ambulance at 17:50 having suffered a collapse. His wife came home to find him on the floor unable to move his right arm or leg and unable to speak. Her call to the ambulance was logged at 17:30. He has a past medical history of well-controlled hypertension, ischaemic heart disease and atrial fibrillation for which he is on warfarin. He had a hernia repair three months ago and his brother had a 'bleed in the brain' at the age of 67. What is the absolute contraindication to thrombolysis in this male?

A. Family history of haemorrhagic stroke
B. History of recent surgery
C. Time of onset
D. Current haemorrhagic stroke
E. Warfarin treatment