Diagnosis and Management of Acute Stroke

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14th RTC -EAN
Dar es Salam, Tanzania
30th October 2023

Outlines of this Talk

- Definitions
  - Stroke
  - TIA
- Types of stroke:
  - Ischaemic, Haemorrhagic(ICH, SAH), Venous strokes, Spinal Cord Stroke
- Daignosis of Acute stroke (FAST. AVVV)
  - History – Clinical examinations - Imaging
- Identification of Stroke Risk Factors:
  - Modifiable : Hypertension High (BP) , AF, DM ,Dylip,)
  - Imaging in acute stroke
- Management of Ischaemic Stroke
  - Monitoring (? Stroke Unit, ICU , A &E ward)
  - BP management during General Care (IP or OPD)
  - Thrombolytic therapy (tPA)
  - Endovascular Clot retrieval techniques
- Management of Haemorrhagic stroke
  - ICH/ SAH
- Neurosurgical Interventions in stroke:
  - Decompressive Craniectomy / open aneurysmal clipping/ EVD for complications
- Rehabilitation in Acute Stoke
- Stroke Mimics and Stroke Chameleons






Stroke :definition

"Rapidly developing clinical signs of focal (or global) disturbance of cerebral , spinal or retinal function with symptoms **lasting 24 hours** or longer or leading to death with no apparent cause other than that of vascular origin".

Silent stroke — radiological or pathological evidence of an infarction or haemorrhage not caused by trauma without an attributable history of acute neurological dysfunction attributable to the lesion.






Global Burden of Disease Study

- Stroke is the second leading cause of mortality worldwide
- 3 million of the 4.5 million deaths occur in developing countries
- The most uncertain estimates are those for large parts of sub-Saharan Africa where “even the exact levels of mortality rates are not known”
- A leading cause of adult disability
- **Up to 80% of all strokes are preventable through risk factor management**
- On average, someone suffers a stroke every 40 seconds in America





Stroke Mortality

- Stroke mortality rises rapidly with age
- In industrialised countries stroke is the third leading cause of death (10-12% of all deaths) - 88% are over 65 years.
- Stroke mortality in the US is significantly higher in African Americans than in caucasians.






Aetiology

- Cerebral infarction (CI) – 75- 85%
- Primary intracerebral haemorrhage (PICH) – 10-15 %
- Subarachnoid haemorrhage (SAH) - 5%
- Cerebral venous thrombosis - < 1%
- Spinal Cord Stroke ? epidemiology

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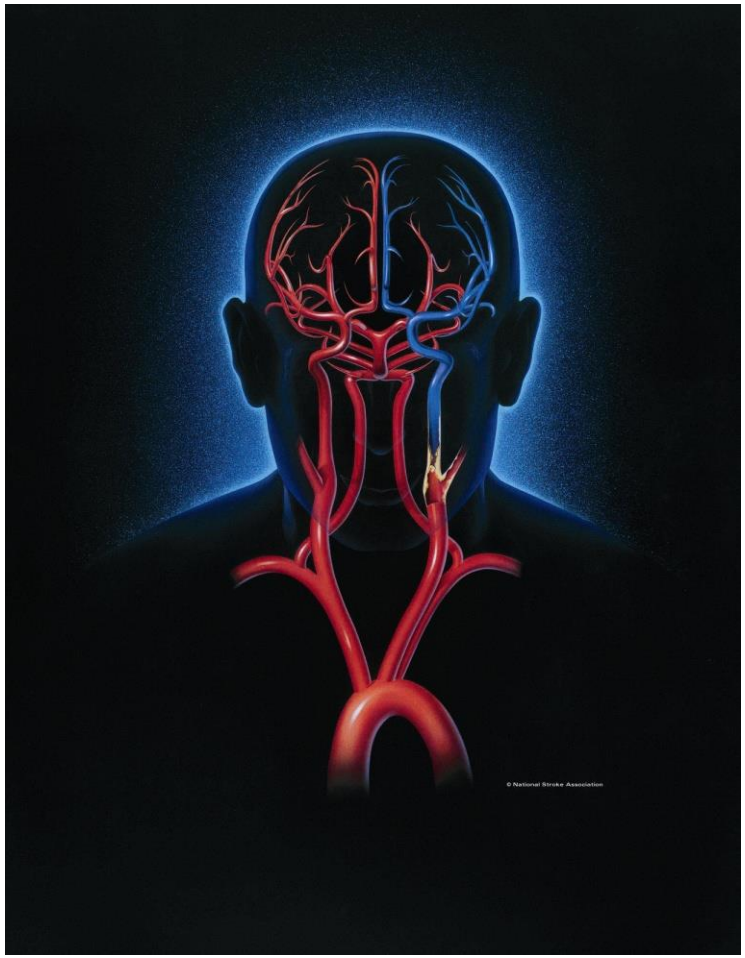
Diagnosis and Acute Management

Stroke:

- Acute Stroke
- Recurrent
- Vascular Dementia

TIA

Brain Attack! Time is Brain



Stroke is a "Brain Attack."

Stroke happens in the brain not the heart

Stroke is an emergency

Stroke Strikes F.A.S.T.
You Should, To Act Fast

- **F =** Face: ask the person to smile
- **A =** Arm: ask the person to raise both arms
- **S =** Speech: ask the person to speak a simple sentence
- **T =** Time: to act

• **FAST is Not Good for Posterior Circulation**

USE (AVVV = Ataxia. Vertigo, vomiting, Visual disturbance)

Every minute matters!

Time is BRAIN

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Transient Ischaemic Attacks (TIA)

- Symptoms and signs resolve **within 24 hours** (most within 30 minutes)
- As many as 20% may sustain a small infarct visible on CT/MRI (DWI)
- 5-10 times risk of subsequent stroke
- Only 15% of strokes are preceded by a TIA

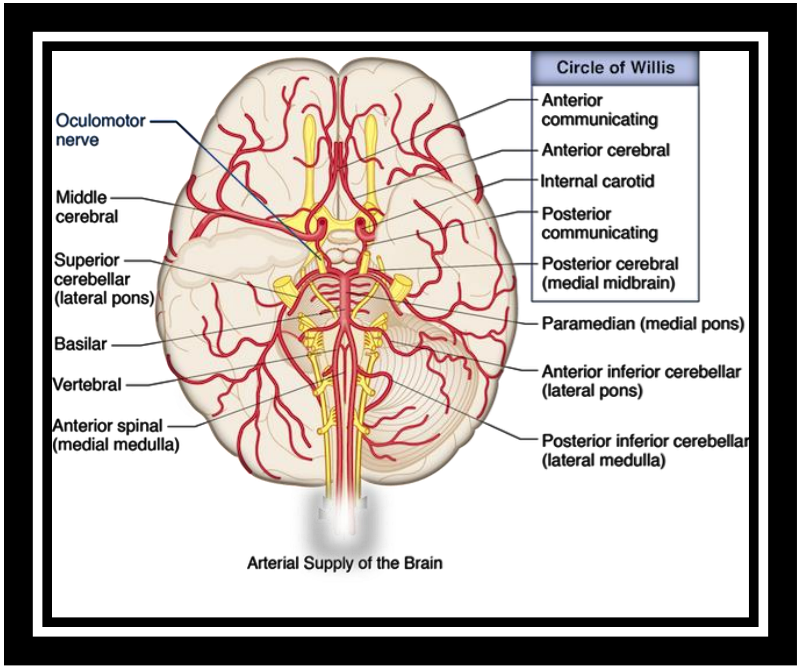


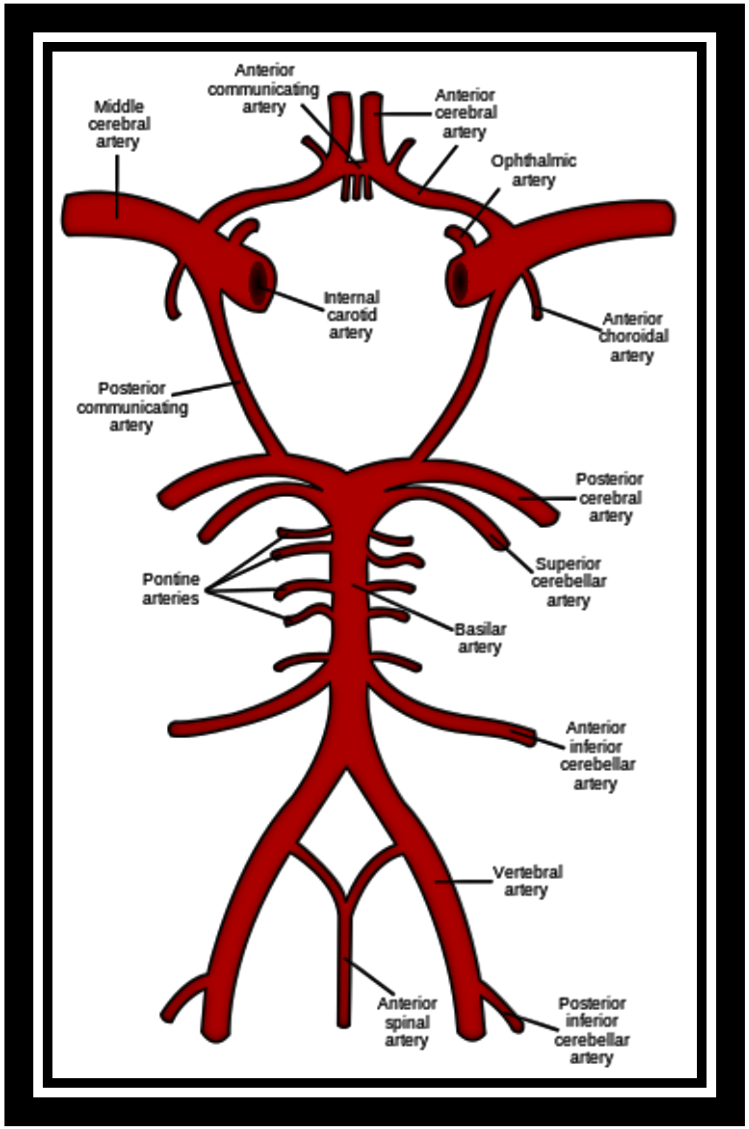



Why People Don't Recognize and Respond to Symptoms

- Don't recognize symptoms
- Denial
- Think nothing can be done
- Worry about cost
- Think symptoms will go away
- Fear or don't trust hospitals

Blood Supply





- Anterior Cerebral Artery
- Middle Cerebral Artery
- Posterior Cerebral Artery
- Vertebral & Basilar Arteries

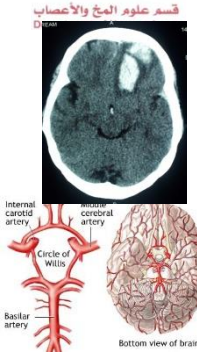
Classification of Stroke

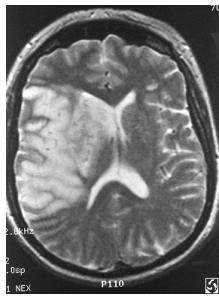
Where is the Lesion?

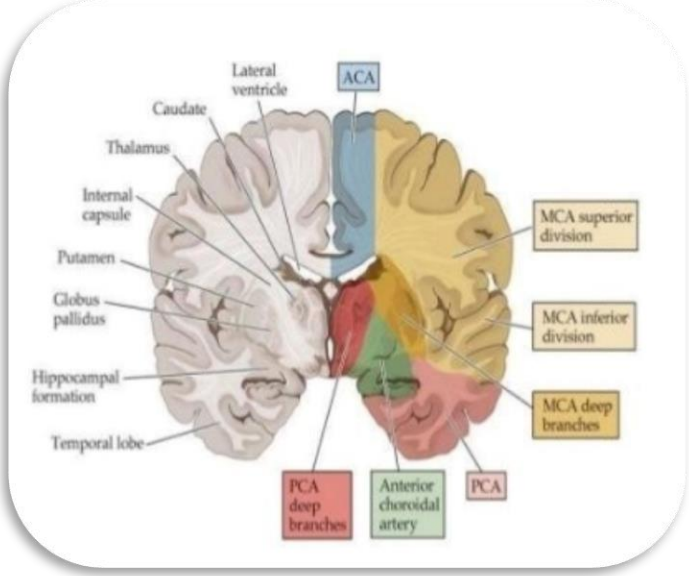
Bamford Classification System (Oxford Classification System) –

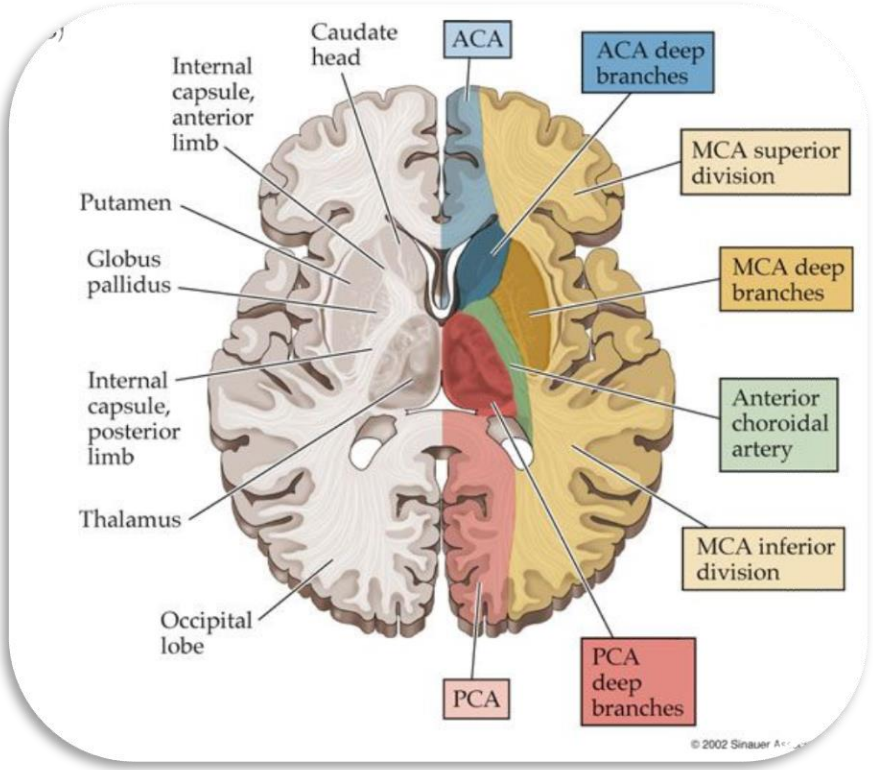
- **TACS – Total Anterior Circulation Stroke** - All 3 – Unilateral motor +- sensory face arm leg, Homonymous hemianopia, Higher cortical function (Dysphasia, Visuospatial)

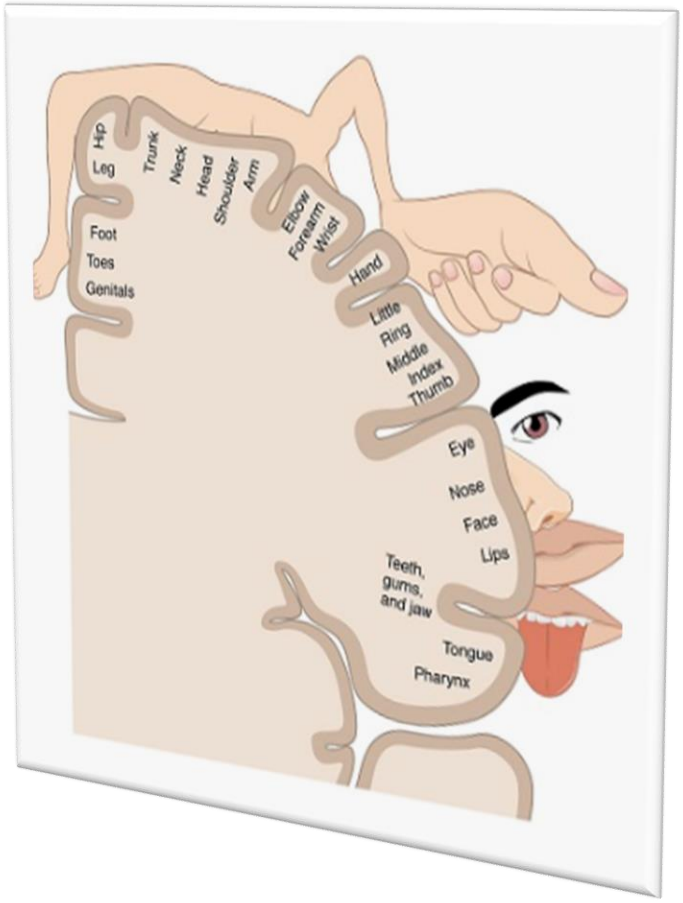
- PACS – Partial Anterior Circulation Stroke – 2 out 3 Unilateral motor +- sensory face, arm, leg, Homonymous hemianopia, Higher cortical function (Dysphasia, Visuospatial)

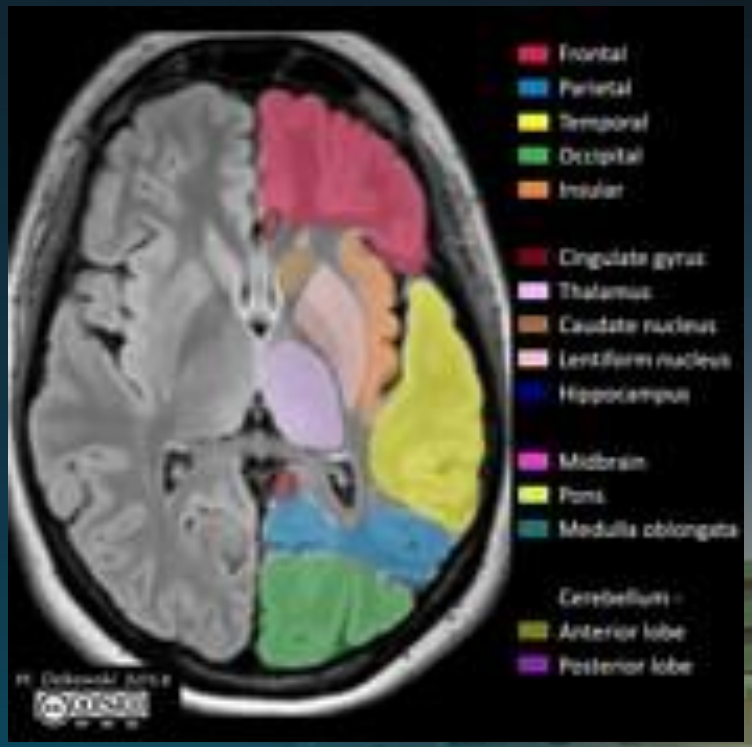


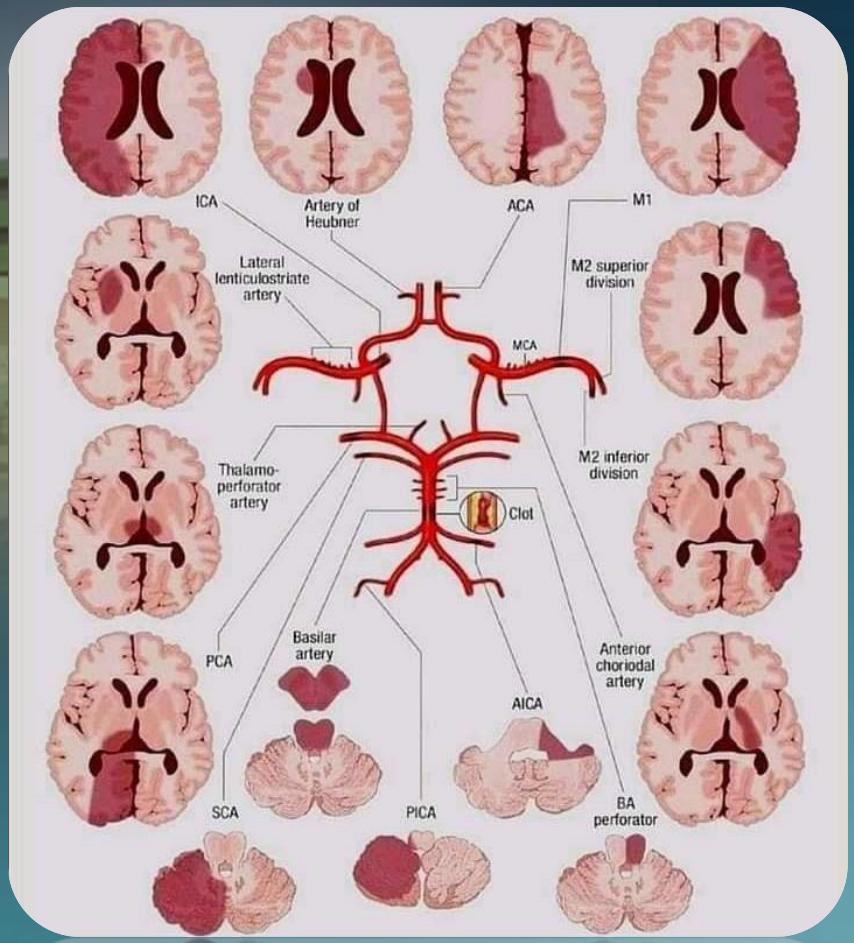



Lateral ventricle
ACA
Caudate
Thalamus
Internal capsule
Putamen
Globus pallidus
Hippocampal formation
Temporal lobe
MCA superior division
MCA inferior division
MCA deep branches
PCA deep branches
Anterior choroidal artery
PCA


Caudate head
ACA
ACA deep branches
Internal capsule, anterior limb
MCA superior division
Putamen
Globus pallidus
MCA deep branches
Internal capsule, posterior limb
Anterior choroidal artery
Thalamus
MCA inferior division
Occipital lobe
PCA deep branches
PCA
© 2002 Sinauer


Hip
Leg
Trunk
Neck
Head
Shoulder
Arm
Elbow
Forearm
Wrist
Hand
Foot
Toes
Genitals
Little
Ring
Middle
Index
Thumb
Eye
Nose
Face
Lips
Teeth, gums, and jaw
Tongue
Pharynx

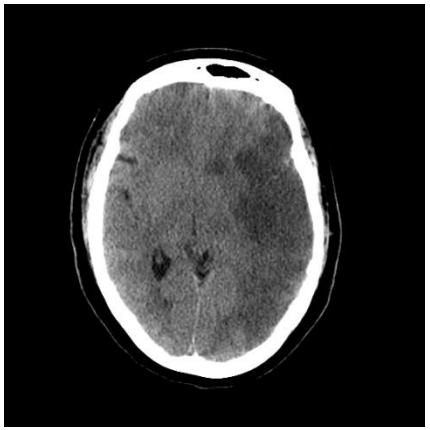

Frontal
Parietal
Temporal
Occipital
Insular
Cingulate gyrus
Thalamus
Caudate nucleus
Lentiform nucleus
Hippocampus
Midbrain
Pons
Medulla oblongata
Cerebellum -
Anterior lobe
Posterior lobe


ICA
Artery of Heubner
ACA
M1
Lateral lenticulostriate artery
M2 superior division
MCA
Thalamo-perforator artery
M2 inferior division
Clot
PCA
Basilar artery
Anterior choriodal artery
AICA
SCA
PICA
BA perforator

ACS

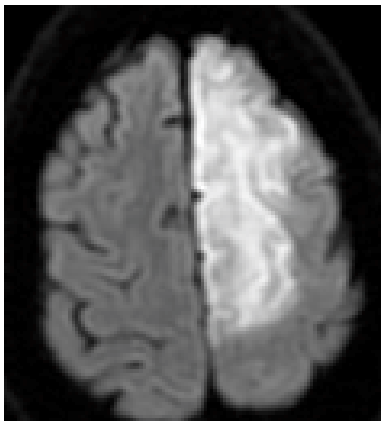
TACS

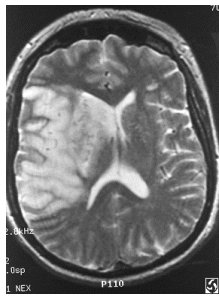
- Hemiparesis
- Hemianopia
- HCD: dysphasia, VS dysfunction



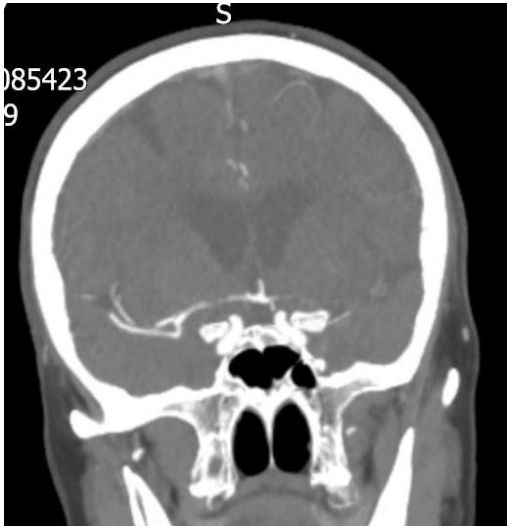
PACS: MCA or ACA

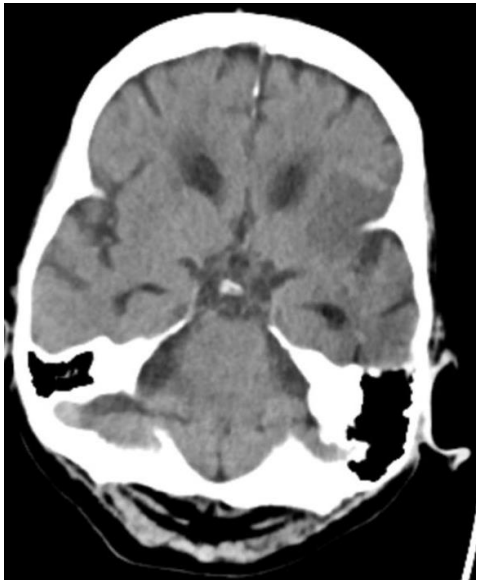
- Any 2 of:
- Hemiparesis
- Hemianopia
- dysphasia, VS dysfunction
- HCD alone





Hyperdense MCA Sign





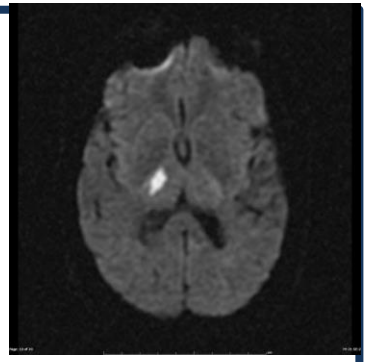
LACS

Pure motor

Pure sensory

Sensorimotor

Ataxic hemiparesis



POCS

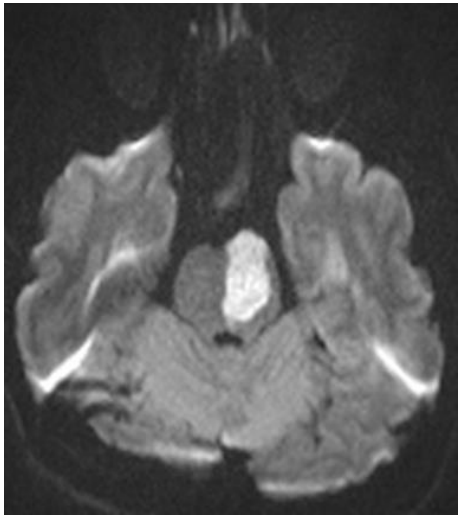
MANY SYNDROMES

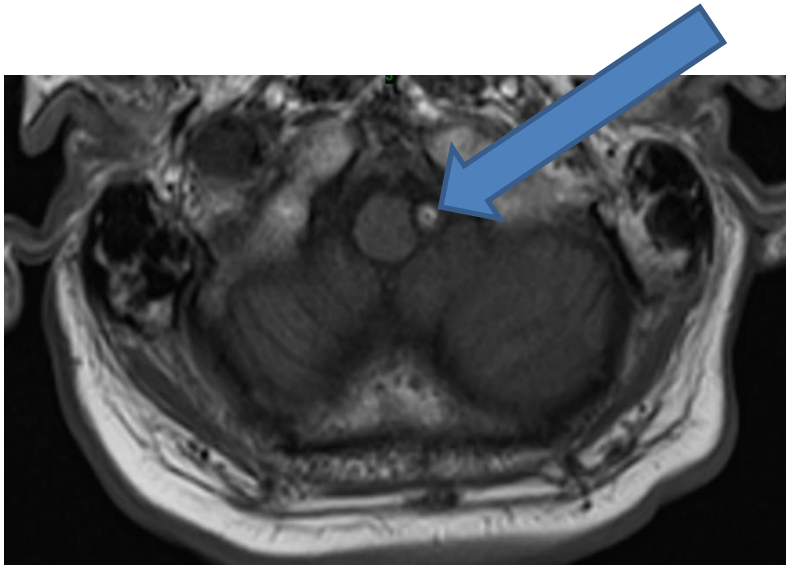
HEMI/QUADRIPARESIS, ATAXIA, CN PALSIES, CONJUGATE EYE MOVEMENT DISORDER, NYSTAGMUS, COMA, VF DEFECTS

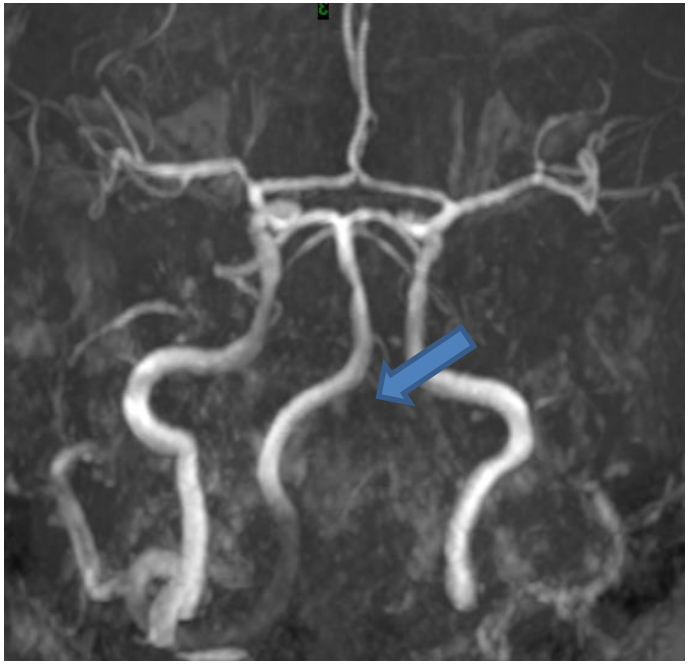
Posterior Circulation Infarctions

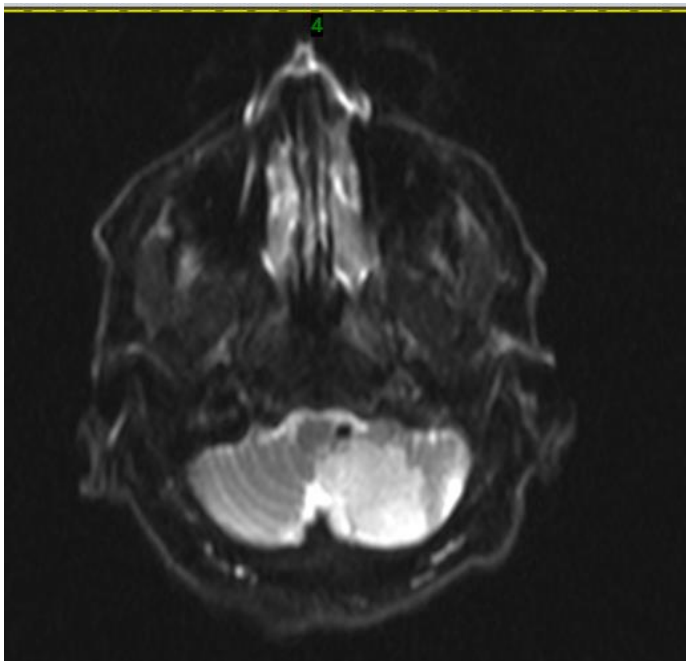


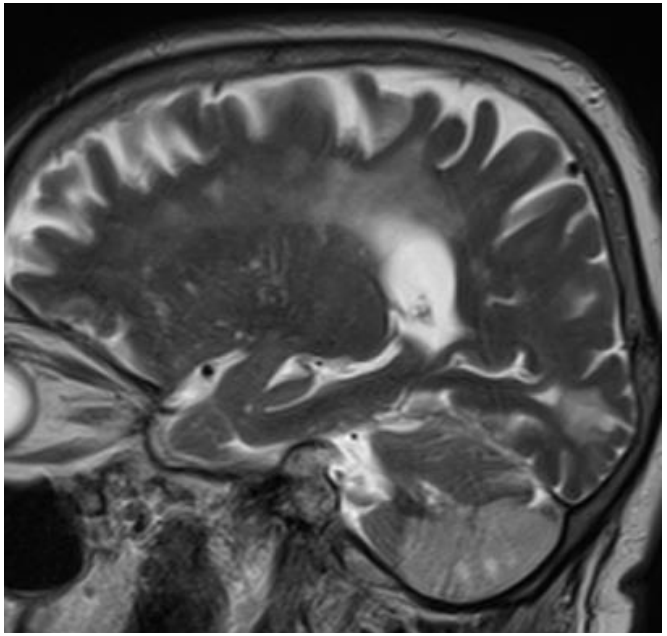










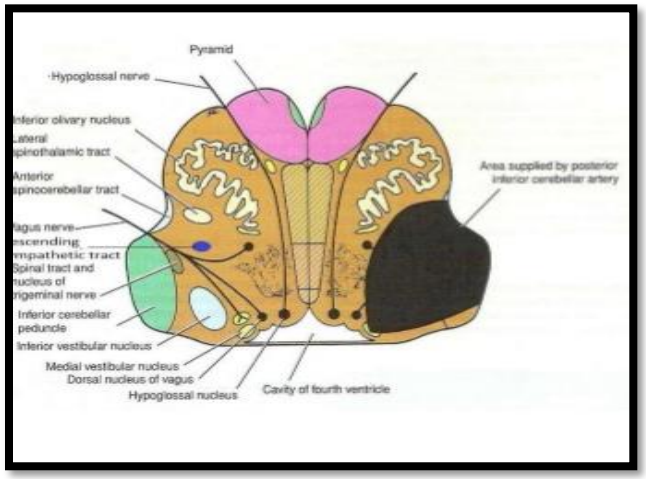


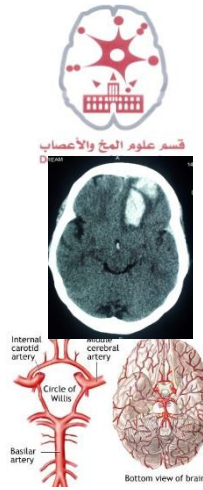
Lateral Medullary Syndrome

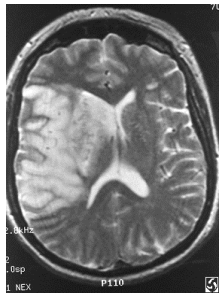
(Wallenburg Syndrome)

Characterised by:

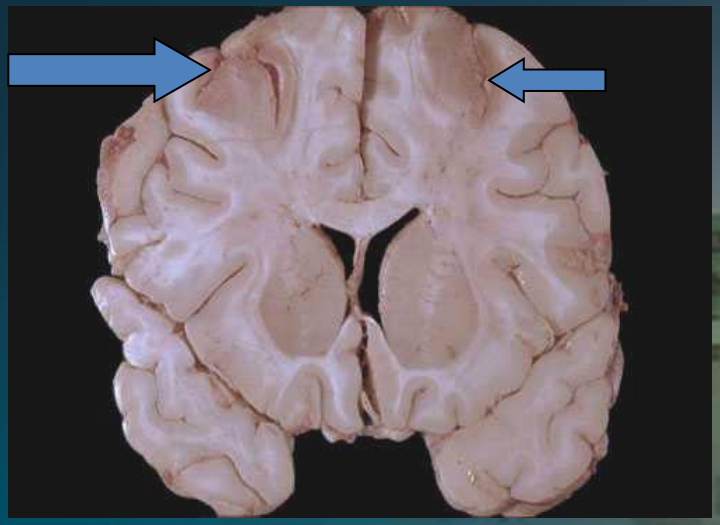
- **vestibulocerebellar symptoms:**
  - **vertigo, falling towards the side of lesion, diplopia, and multidirectional nystagmus**
  - **Autonomic dysfunction: ipsilateral Horner syndrome, hiccups**
- **sensory symptoms:**
  - **initially abnormal stabbing pain over the ipsilateral face then loss of pain and temperature sensation over the contralateral side of body (spinal trigeminal nucleus involvement)**
- **Ipsilateral bulbar muscle weakness: hoarseness, dysphonia, dysphagia, and dysarthria, decreased gag reflex (nucleus ambiguus)**

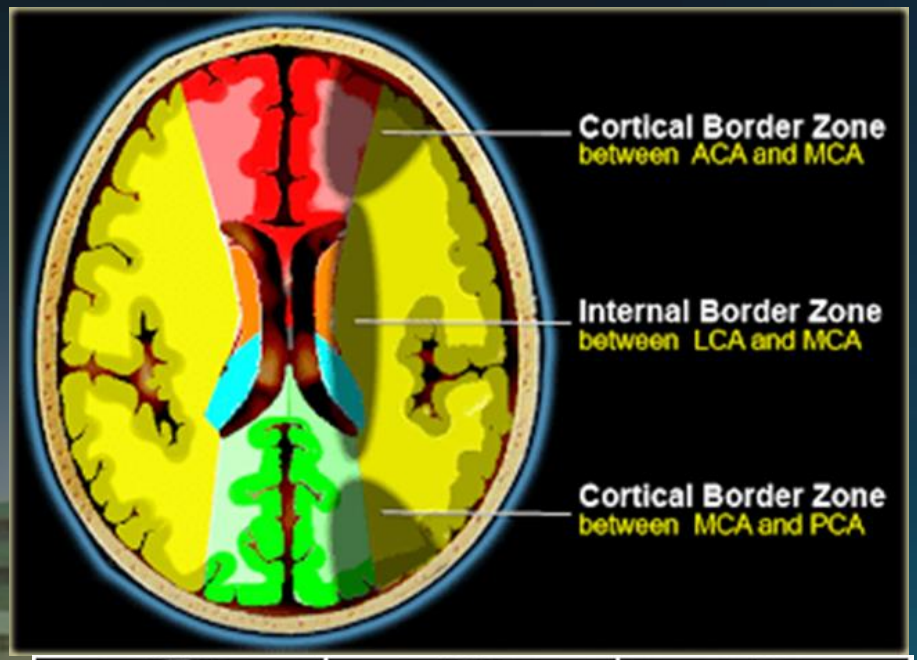


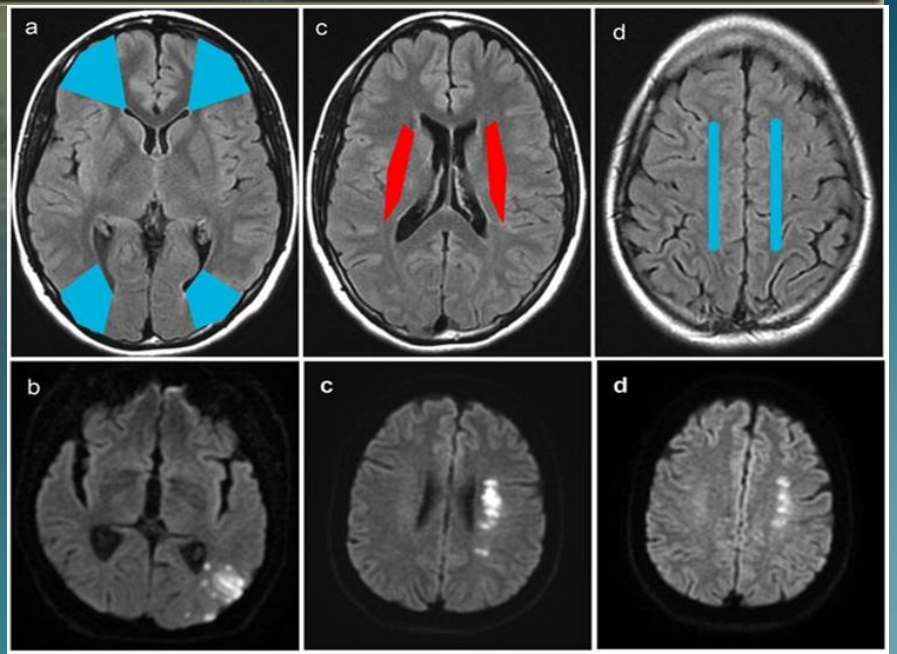


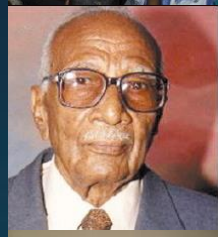


Watershed/Boundary zone infarcts

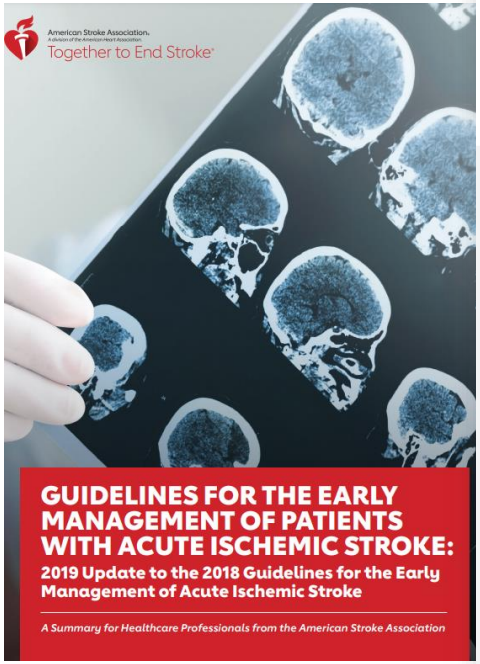








Management of Stroke



World Stroke Organization

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Summary of systematic review and synthesis of global stroke guidelines on behalf of WSO

12 May 2023

Gillian E Mead, Luciano A Sposato, Gisele Sampaio Silva, Laetitia Yperzeele, Simiao Wu, Mansur Kutlubaev, Joshua Cheyne, Kolawole Wahab, Victor C Urrutia, Vijay K Sharma, PN Sylaja, Kelvin Hill, Thorsten Steiner, Mayowa Owolabi, David S Liebeskind and Alejandro A Rabinstein

Management Acute Ischaemic Stroke

- **General Care:**
  - Reassure, O2, Vascular access, blood samples –Basic tests
  - Brief History and neurological examination (Grading)
  - Cardiovascular check
    - BP
    - Rhythm: AF
    - Carotid bruit
- **Imaging:**

  CT brain CT angio. / perfusion scan / MRI (MRA/ MRV)
- **Reperfusion therapy**
  - Thrombolysis – tPA
  - 4.5 Hr exact time ? Wake up time ? Extended time issue (MRI guided – DWI)
- **Outside Time Limit:**
  - Full general and Neurological assessment
  - Medical therapy
  - Mechanical Thrombectomy:
    - Wake up stroke – MRI needed (DWI)
    - Large artery occlusion/ Basilar Artery





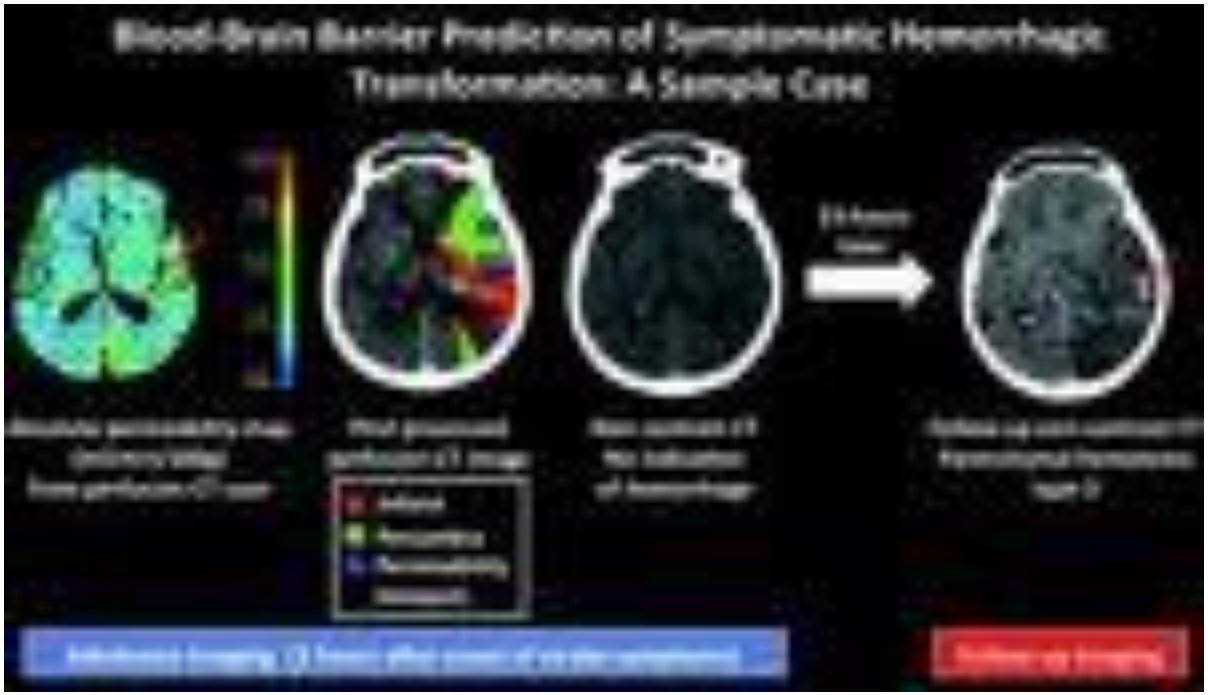

Medical Therapy of Acute Stroke

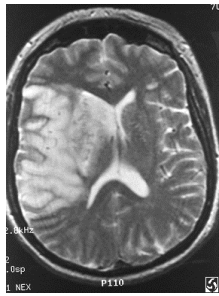
- **BP control**
- **Antiplatelets**
  - **Aspirin 300mg od – 14/7 if no CI or Clopidogrel 300mg stat then 75mg OD**
  - **Dipyridamole can be used in secondary prevention but not in conjunction with Clopidogrel**
- **Lipid lowering drugs**
- **Swallowing assessment/ nutrition:**
  - **NGT as needed**
- **Fluid balance –**
  - **Adequate hydration**
  - **need for bladder catheterization if needed**
- **DVT prophylaxis**
- **Identification and management of Co- morbidities:**
  - **Euoglycaemia – DM –prognosis**
  - **Cardiovascular Problems: Arrhtyjmia, Carotid stenosis, cardia lesion)**
  - **Infections (complication / cause)**
  - **Complications**
- **Transfer to ICU/ Stroke Unit/ stroke ward/ medical ward**
- **Consider discharge in minor stroke : lacunar/ recurrent minor**






Thrombolytic Therapy





Hypertension High (BP) *****

Management of BP in Ischaemic Stroke

- Monitoring (? Stroke Unit, ICU , A &E ward)
- BP management during General Care (IP or OPD)
- BP management in thrombolytic therapy (tPA)
- BP management with Clot retrieval techniques

Management of BP with ICH/ SAH

BP control with Neurosurgical Interventions in stroke:

- Decompressive Craniectomy / open aneurysmal clipping/ EVD for complications

Rehabilitation in Stoke:

- BP care with Physiotherapy / treat Spasticity including Botox
- BP of the Carers!!!

Secondary Prevention --





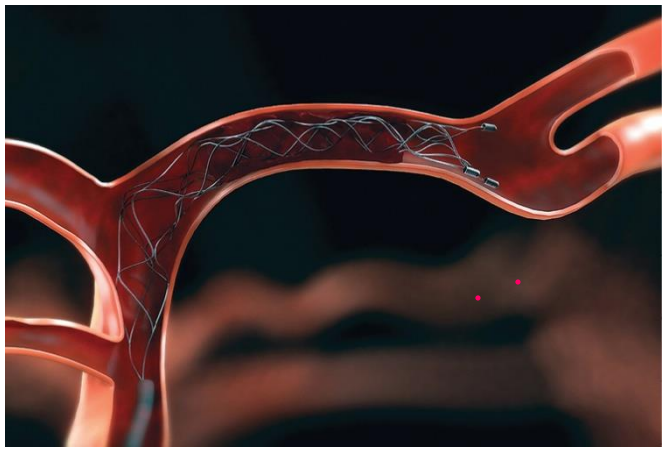

Haemorrhagic Stroke

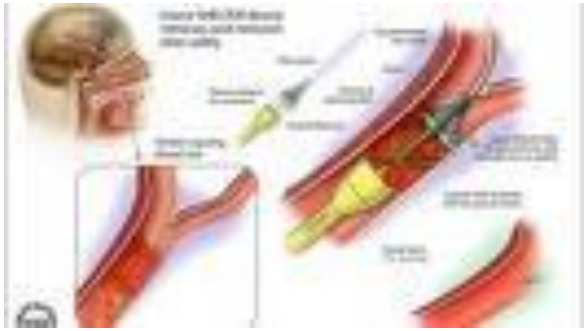
- **Hypertension**
- Cerebral Amyloid Angiopathy
- Drugs – Anticoagulation
- AVM
- Tumors: primary / mets.
- Cavernoma / venous angiona
- Drugs: Cocaine , Cannabis
- Scorpions/ Snakes envenomation

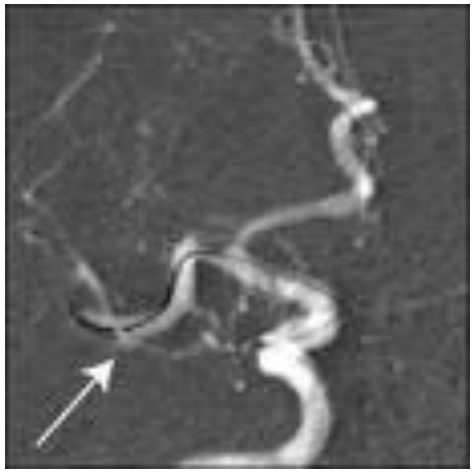


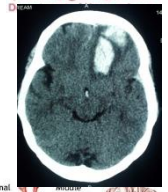


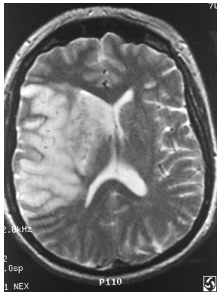

Endovascular Clot Removal



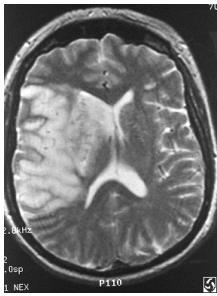






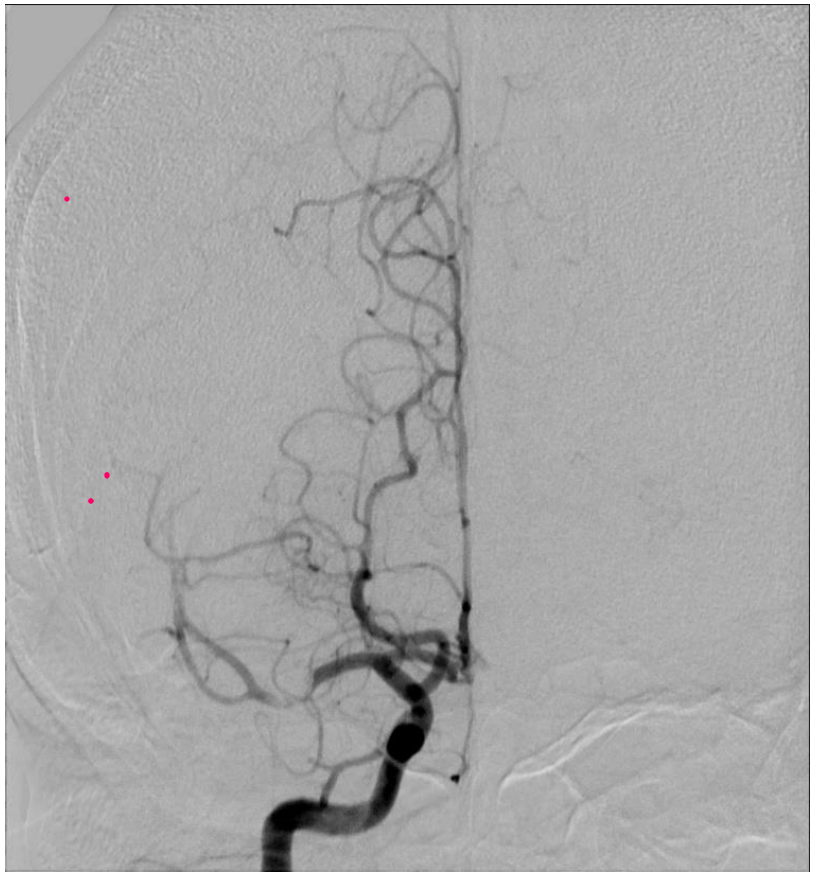


Endovascular Thrombectomy

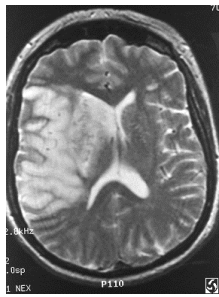


Clot Retrieval

Pre-clot retrieval

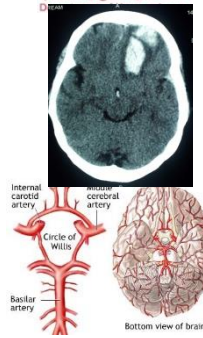


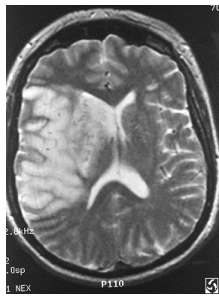
Post-clot retrieval



RCP Guidelines- Clot Retrieval 2016

- **Beyond an onset-to-arterial puncture time of 5 hours if:**
- **The large artery occlusion is in the posterior circulation, in which case treatment up to 24 hours after onset may be appropriate;**
- **A favourable profile on salvageable brain tissue imaging has been proven, in which case treatment up to 12 hours after onset may be appropriate.**



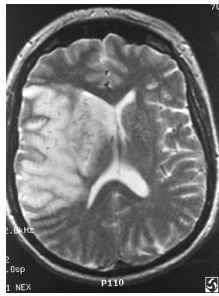


The ENCHANTED2/MT trial is the largest multicentre randomised controlled trial comparing the safety and efficacy of blood pressure (BP)–lowering treatment targets in patients after endovascular therapy for acute ischaemic stroke.

Among 821 patients, intensive BP lowering to a systolic target less than 120 mm Hg, compared with a systolic target of 140 to 180 mm Hg, was associated with worse functional outcomes (OR, 1.37), greater early neurological deterioration at 7 days (OR, 1.53), and major disability at 90 days (OR, 2.07). There was no significant difference in the risk of symptomatic intracerebral haemorrhage or mortality between the target groups.

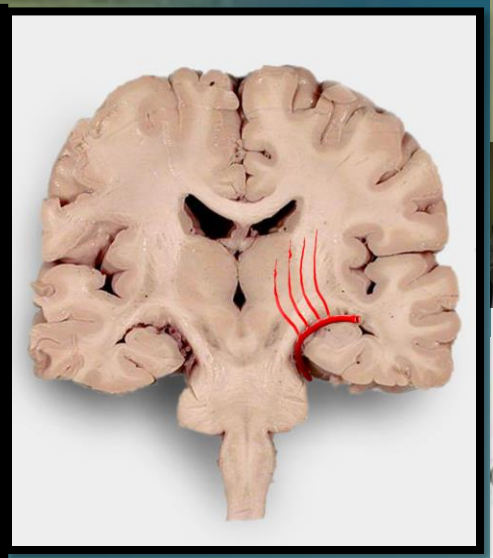
After endovascular thrombectomy for acute ischaemic stroke owing to intracranial large-vessel occlusion, intensive control of systolic BP to lower than 120 mm Hg should be avoided, given the risk of neurological deterioration and worse functional outcomes.

– Giselle A. Suero-Abreu, MD, PhD, MSc



Hypertensive Bleeds

- Most common bleed
- Secondary to microaneurysms of perforating arteries – Charcot-Bouchard aneurysms – Chronic Hypertension
  - 80% Lenticulostriate – BG (most common - Putamen), Thalamus
  - 10% Pons
  - 10% Cerebellum



RCP Guidelines 2016

Patients with primary intracerebral haemorrhage who present within 6 hours of onset with a systolic BP above 150mmHg should be treated urgently using a locally agreed protocol for blood pressure lowering to a systolic blood pressure of 140 mmHg for at least 7 days, unless:

– the Glasgow Coma Scale score is 5 or less;

– the haematoma is very large and death is expected

– a structural cause for the haematoma is identified;

– immediate surgery to evacuate the haematoma is planned (Premorbid fit- progressive/ rebleeding)

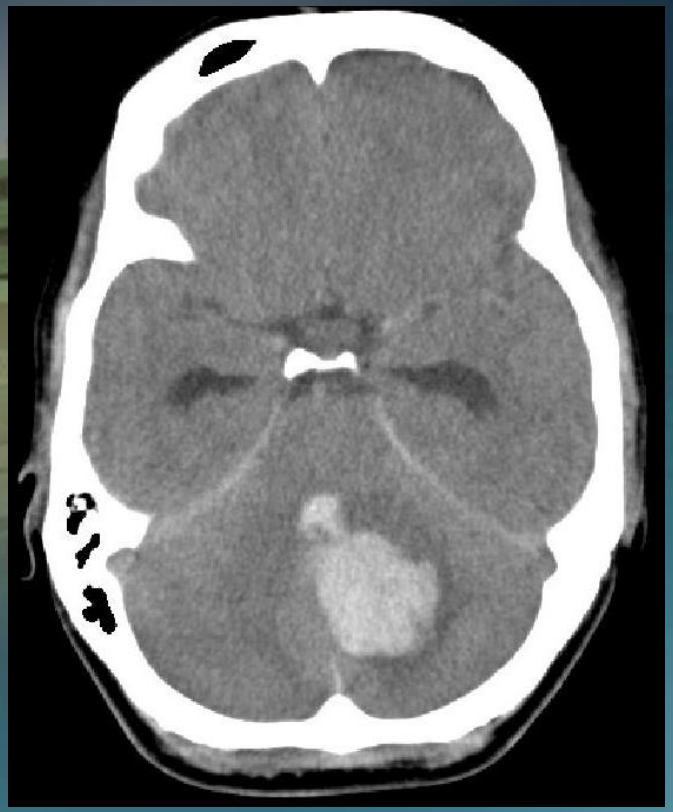




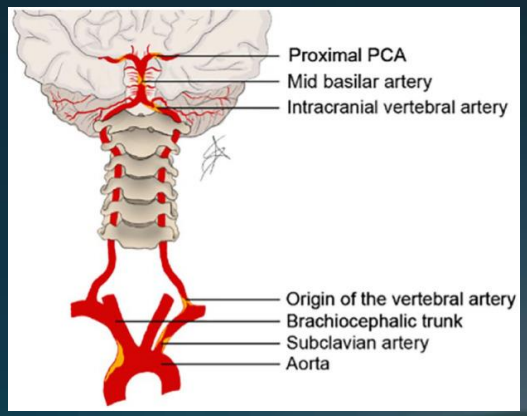

Hypertensive Bleeds Pons

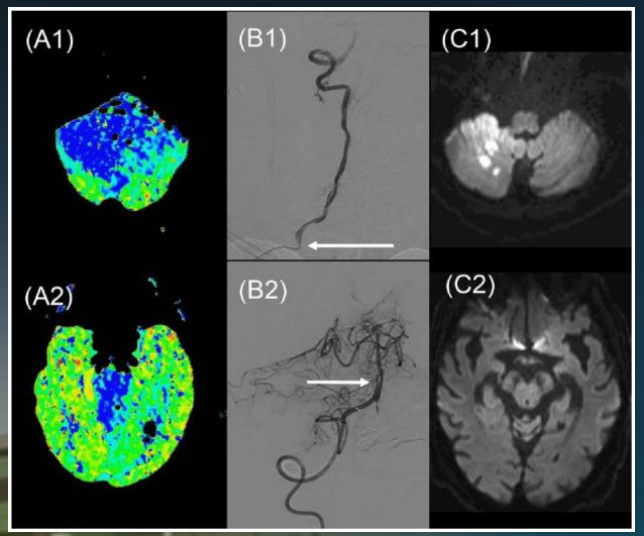
Pons

Cerebellum



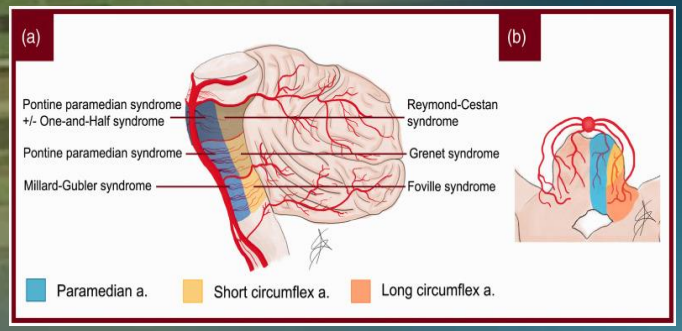
Posterior Circulation Stroke 2





The 5 Ds

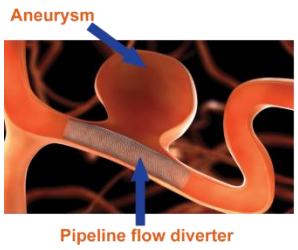
- Dizziness.
- Diplopia (double vision)
- Dysarthria (slurred speech)
- Dysphagia (difficulty swallowing)
- Dystaxia (abnormal gait, balance, motor movements)

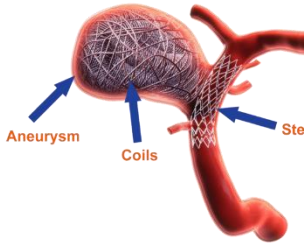


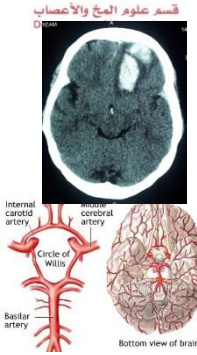
Posterior fossa craniotomy and evacuation of the haemorrhage may be necessary for patients with worsening clinical condition.
With surgical intervention some comatose patients still may have a good clinical outcome

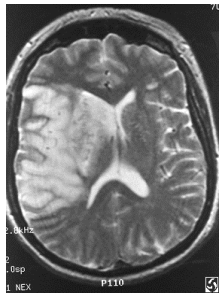
Subarachnoid Haemorrh (SAH)

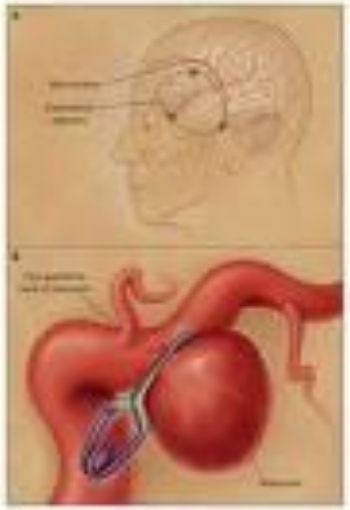
- Rupture of aneurysm in the circle of Willis
- Occasionally due to leak from an arteriovenous malformation (AVM) - spontaneous or secondary to trauma
- Cerebral vasospasm may lead to delayed cerebral infarction (4-14 days) in up to a third of patients
- Non-Aneurysmal SAH- peri-mesencephalic
- Extension from ICH or Intra-ventricular haemorrhage



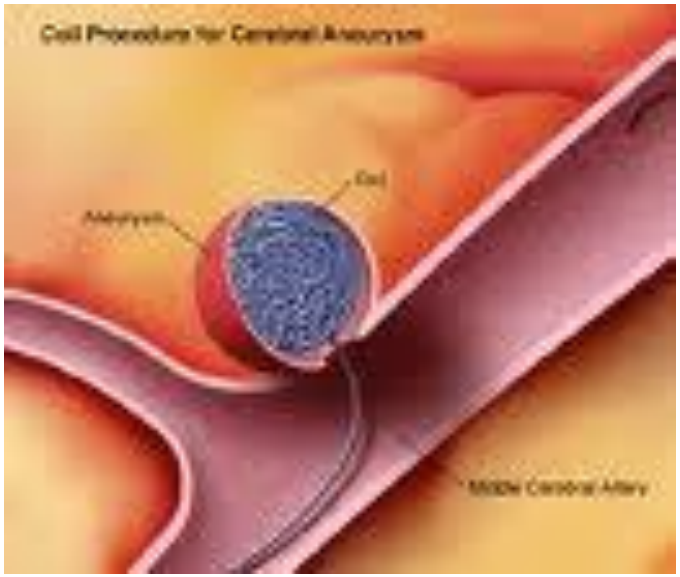




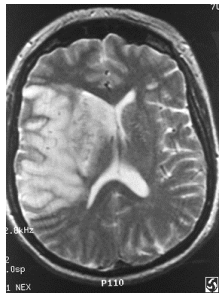





Types of Aneurysms


Coil Procedure for Cerebral Aneurysm
Middle Cerebral Artery


Internal carotid artery
Middle cerebral artery
Circle of Willis
Basilar artery
Bottom view of brain



BP Management in SAH

AHA/ASA Guideline

Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists.

Endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons; and by the Society of NeuroInterventional Surgery

E. Sander Connolly, Jr, MD, FAHA, Chair; Alejandro A. Rabinstein, MD, Vice Chair; J. Ricardo Carhuapoma, MD, FAHA; Colin P. Derdeyn, MD, FAHA; Jacques Dion, MD, FRCPC; Randall T. Higashida, MD, FAHA; Brian L. Hoh, MD, FAHA; Catherine J. Kirkness, PhD, RN; Andrew M. Naidech, MD, MSPH; Christopher S. Ogilvy, MD; Aman B. Patel, MD; B. Gregory Thompson, MD; Paul Vespa, MD, FAAN; on behalf of the American Heart Association Stroke Council, Council on Cardiovascular Radiology and Intervention, Council on Cardiovascular Nursing, Council on Cardiovascular Surgery and Anesthesia, and Council on Clinical Cardiology

Purpose—The aim of this guideline is to present current and comprehensive recommendations for the diagnosis and

NICE National Institute for Health and Care Excellence

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Home › NICE Guidance › Conditions and diseases › Cardiovascular conditions › Cranial aneurysms

Subarachnoid haemorrhage caused by a ruptured aneurysm: diagnosis and management

NICE guideline [NG228] Published: 23 November 2022

Guidance | Tools and resources | Information for the public | Evidence | History

- Overview
- Recommendations
- Recommendations for research
- Rationale and impact
- Context

Guidance

Download guidance (PDF)

Next

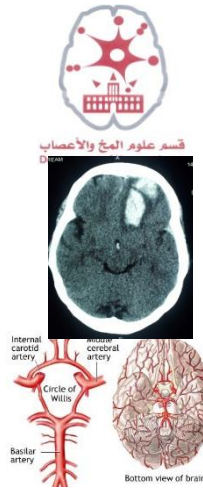
Recommendations

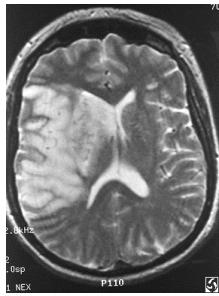
1.1 Assessment and diagnosis

1.2 Managing a confirmed aneurysmal subarachnoid haemorrhage

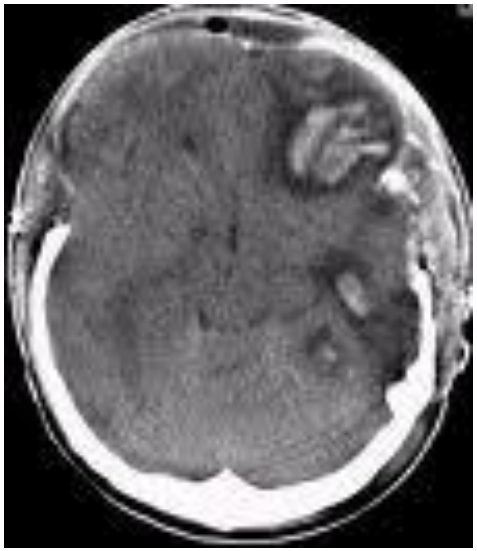






قسم علوم المخ والأعصاب
Internal carotid artery
Middle cerebral artery
Circle of Willis
Basilar artery
Bottom view of brain


P110

Neurosurgery in Stroke



Decompressive craniotomy
Clot removal
Carotid Endarterectomy/ stenting
Vascular bypasses
SAH- management (ligation, embolization)

SPECIAL ARTICLE Level of Recommendation

Stroke Prevention in Symptomatic Large Artery Intracranial Atherosclerosis Practice Advisory

Report of the AAN Guideline Subcommittee

Tanya N. Turan, MD, MSCR, Osama O. Zaidat, MD, Gary S. Gronseth, MD, Marc I. Chimowitz, MBChB, Antonio Culebras, MD, Anthony J. Furlan, MD, Larry B. Goldstein, MD, Nestor R. Gonzalez, MD, Julius G. Latorre, MD, MPH, Steven R. Messé, MD, Thanh N. Nguyen, MD, Rajbeer S. Sangha, MD, Michael J. Schneck, MD, MBA, Aneesh B. Singhal, MD, Lawrence R. Wechsler, MD, Alejandro A. Rabinstein, MD, Mary Dolan O'Brien, MLIS, Heather Silsbee, and Jeffrey J. Fletcher, MD, MSc

Correspondence
American Academy of Neurology
guidelines@aan.com

Neurology® 2022;98:486-498. doi:10.1212/WNL.0000000000200030

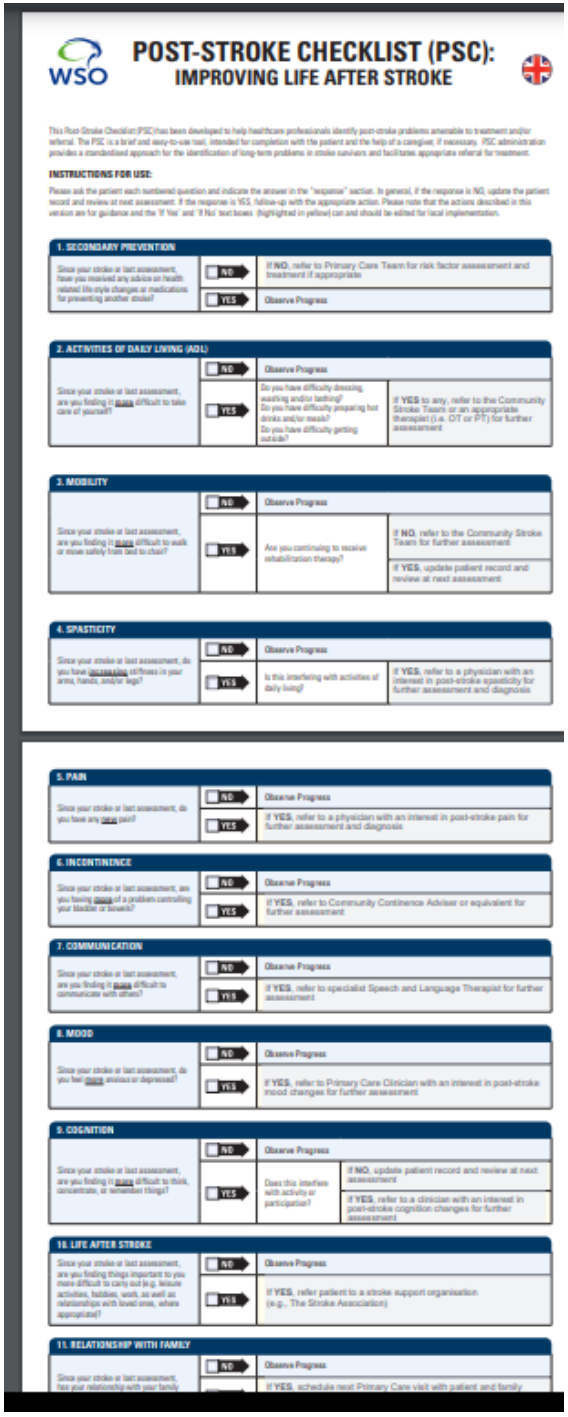
Abstract

Background and Objectives

To review treatments for reducing the risk of recurrent stroke or death in patients with symptomatic intracranial atherosclerotic arterial stenosis (sICAS).

Methods

The development of this practice advisory followed the process outlined in the American Academy of Neurology *Clinical Practice Guideline Process Manual, 2011 Edition,* as amended. The systematic review included studies through November 2020. Recommendations were based on evidence, related evidence, principles of care, and inferences.



WSO

POST-STROKE CHECKLIST (PSC):
IMPROVING LIFE AFTER STROKE

This Post-Stroke Checklist (PSC) has been developed to help healthcare professionals identify post-stroke problems amenable to treatment and/or referral. The PSC is a brief and easy-to-use tool, intended for completion with the patient and the help of a caregiver, if necessary. PSC administration provides a standardised approach for the identification of long-term problems in stroke survivors and facilitates appropriate referral for treatment.

INSTRUCTIONS FOR USE:

Please ask the patient each numbered question and indicate the answer in the "response" section. In general, if the response is NO, update the patient record and review at next assessment. If the response is YES, follow-up with the appropriate action. Please note that the actions described in this version are for guidance and the 'If Yes' and 'If No' text boxes (highlighted in yellow) can and should be edited for local implementation.

1. SECONDARY PREVENTION

Since your stroke or last assessment, have you received any advice on health related life-style changes or medications for preventing another stroke?

- NO → If NO, refer to Primary Care Team for risk factor assessment and treatment if appropriate
- YES → Observe Progress

2. ACTIVITIES OF DAILY LIVING (ADL)

Since your stroke or last assessment, are you finding it more difficult to take care of yourself?

- NO → Observe Progress
- YES → Do you have difficulty dressing, washing and/or bathing? Do you have difficulty preparing hot drinks and/or meals? Do you have difficulty getting outside? → If YES to any, refer to the Community Stroke Team or an appropriate therapist (i.e. OT or PT) for further assessment

3. MOBILITY

Since your stroke or last assessment, are you finding it more difficult to walk or move safely from bed to chair?

- NO → Observe Progress
- YES → Are you continuing to receive rehabilitation therapy?
  - If NO, refer to the Community Stroke Team for further assessment
  - If YES, update patient record and review at next assessment

4. SPASTICITY

Since your stroke or last assessment, do you have increasing stiffness in your arms, hands, and/or legs?

- NO → Observe Progress
- YES → Is this interfering with activities of daily living? → If YES, refer to a physician with an interest in post-stroke spasticity for further assessment and diagnosis

5. PAIN

Since your stroke or last assessment, do you have any new pain?

- NO → Observe Progress
- YES → If YES, refer to a physician with an interest in post-stroke pain for further assessment and diagnosis

6. INCONTINENCE

Since your stroke or last assessment, are you having more of a problem controlling your bladder or bowels?

- NO → Observe Progress
- YES → If YES, refer to Community Continence Adviser or equivalent for further assessment

7. COMMUNICATION

Since your stroke or last assessment, are you finding it more difficult to communicate with others?

- NO → Observe Progress
- YES → If YES, refer to specialist Speech and Language Therapist for further assessment

8. MOOD

Since your stroke or last assessment, do you feel more anxious or depressed?

- NO → Observe Progress
- YES → If YES, refer to Primary Care Clinician with an interest in post-stroke mood changes for further assessment

9. COGNITION

Since your stroke or last assessment, are you finding it more difficult to think, concentrate, or remember things?

- NO → Observe Progress
- YES → Does this interfere with activity or participation?
  - If NO, update patient record and review at next assessment
  - If YES, refer to a clinician with an interest in post-stroke cognition changes for further assessment

10. LIFE AFTER STROKE

Since your stroke or last assessment, are you finding things important to you more difficult to carry out (e.g. leisure activities, hobbies, work, as well as relationships with loved ones, where appropriate)?

- NO → Observe Progress
- YES → If YES, refer patient to a stroke support organisation (e.g., The Stroke Association)

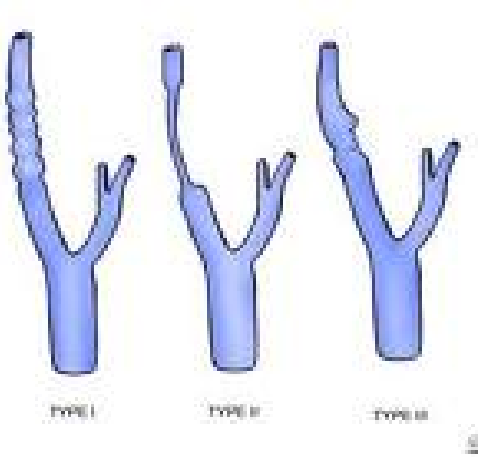
11. RELATIONSHIP WITH FAMILY

Since your stroke or last assessment, has your relationship with your family

- NO → Observe Progress
- YES → If YES, schedule next Primary Care visit with patient and family

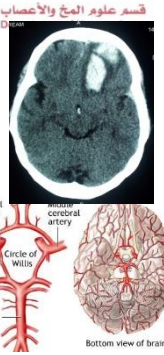
Fibromuscular Dysplasia FMD

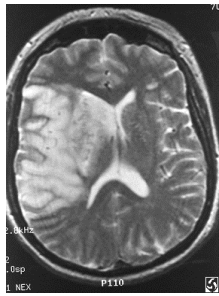
Severe Hypertension in Young patients



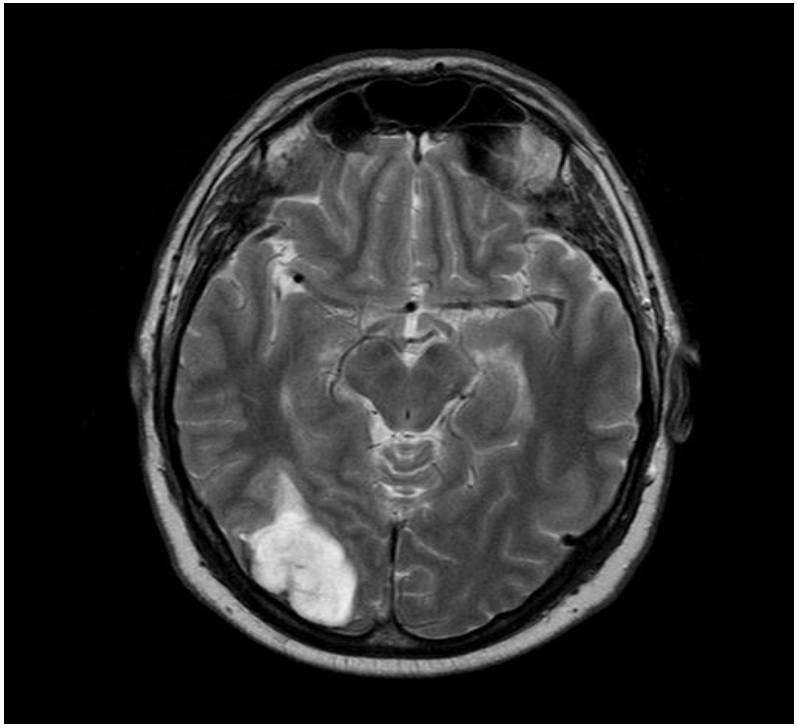
Bil Renal artery stenosis

Avoid ACE inhibitors

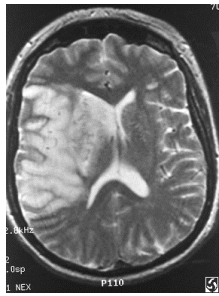




Mild headache with Sudden Loss of part of Visual Field !



Right Occipital Haemorrhage



Osheik A. Seidi
Feb 2004

Neurology and renal disorders

Osheik Seidi MBBS ABIM MRCP(UK), Consultant Neurologist, *Sunderland Royal Hospital and the Regional Neuroscience Centre, Newcastle upon Tyne*

Clin Med 2007;7:165–70

Following the introduction of haemodialysis and renal transplantation it became particularly clear that renal disease may adversely affect the nervous system. A range of neurological manifestations of acute (ARF) and chronic renal failure (CRF) have been recognised.

- There are well-known interactions when systemic disorders such as diabetes or hypertension affect both the kidney and the nervous system.
- Systemic lupus erythematosus (SLE), other vasculitides and granulomatous disorders commonly show neurological and renal complications.
- Electrolyte disturbances in patients with renal and systemic disorders can manifest with specific and non-specific neurological features. These should be recognised early to avoid permanent sequelae which may occur if not treated promptly, as in the osmotic demyelination syndromes following rapid correction of hypo- or hypernatraemia.
- Rare, but potentially treatable, serious neurorenal conditions include thrombotic thrombocytopenic purpura (TTP) and cocaine-related vasculitis.[1]
- Neurological features may be the first indication of renal disease, for example peripheral neuropathy with CRF, aneurysmal subarachnoid haemorrhage in polycystic kidney

Neurological problems associated with renal replacement therapies

Dialysis dysequilibrium syndrome

The dialysis dysequilibrium syndrome is a complication of haemodialysis caused by the creation of an osmotic gradient between the brain cells and the plasma. Rapid clearance of urea and other solutes leads to a shift of water into the brain parenchyma with resultant cerebral oedema. It presents with headache, nausea, vomiting, restlessness, muscle cramps and confusion. It usually resolves in a few hours after dialysis and is prevented by slower dialysis.

Dialysis dementia

Dialysis dementia is now seen infrequently (if at all) compared with the early years of haemodialysis. It was linked to an increased level of aluminium in the soft water used in the dialysate and presented with dysarthria, dysphasia and dysgraphia, progressing to gait apraxia, myoclonic jerks and seizures, leading in extreme cases to immobility and mutism followed by death. Dialysis dementia is treated with the chelating agent desferrioxamine.[2]

Uraemic encephalopathy

Either acute or chronic, uraemic encephalopathy is usually more severe in the context of ARF. The initial symptoms are fatigue, poor concentration and clumsiness, but as the renal function deteriorates there is progression to asterixis, multifocal mycoclonic jerks, generalised seizures, confusion and coma. In the chronic form, patients show emotional lability, sluggishness and inversion of sleep pattern as well as renal replacement therapies (RRTs) and may resolve completely after successful renal transplantation.[3]

Osmotic demyelination syndromes

The osmotic demyelination syndromes (ODS) complicate treatment of hyponatraemia in which serum sodium is usually less than 120 mmol/l. The commonly recognised type is central pontine myelinolysis (CPM), although extrapontine myelinolysis (EPM) is increasingly reported; the pathogenesis is the same in both types. ODS should be considered in patients who deteriorate neurologically after an illness associated with hyponatraemia or have received a large volume of intravenous fluids even if the imaging is not supportive initially. To prevent this serious complication the sodium should not be corrected by more than 8 mmol/l/day, particularly in chronic hyponatraemia (serum sodium <136 mmol/l for >48 hours).

CPM presents with brainstem dysfunction, including flaccid tetraparesis and occasionally the locked-in syndrome. EPM has variable presentations which depend on the affected area

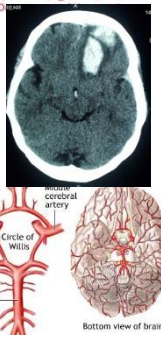
Key Points

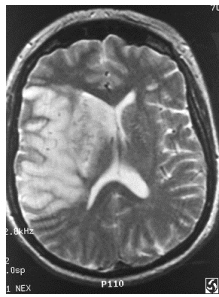
The kidney and the nervous system have close interactions under both physiological and pathological states

Systemic disorders like diabetes, hypertension, vasculitudes and genetic disorders can affect both the nervous system and the kidney

Awareness and early recognition of conditions such as thrombocytopenic purpura and osmotic demyelination syndromes should lead to prompt treatment and prevention of serious sequelae

Neurological features can be the first manifestations of a renal disease or





Stroke in Infections

1- Viral
- HIV
- HZV
- CMV
- PML (JC virus)

Protozoal:
- Malaria
- Toxoplasmosis

2- Bacterial – meningitis including TB

3- Spirochaetal
- Syphilis

4- Helminthic
- Cysticercosis
- Hydatic Disease

5- Post infectious Angiitis






Stroke in HIV

OPEN

Prevalence and incidence of stroke among people with HIV

Min Du[a], Yaping Wang[a], Chenyuan Qin[a], Donghua Mi[b], Min Liu[a] and Jue Liu[a,c,d,e]

Objective: We aimed to obtain more precise estimates of stroke to address the wide variation of stroke burden among people with HIV (PWH) in different clinical settings.

Design: Systematic review and meta-analysis.

Methods: We systematically searched PubMed, EMBASE, and Web of Science for original articles reporting the prevalence and incidence of stroke among PWH up to November 23, 2022. Der Simonian-Laird random effects were used to obtain pooled estimates and 95% confidence intervals (CIs).

Results: We included 17 observational studies covering 1 749 003 PWH on estimation

- *Ischaemic*
- *Haemorrhagic*
- *Venous strokes – C dural Sinus thrombosis*






HIV and Ischaemic Stroke

- Clinical, radiological, and pathological series, there is an increased risk of IS in AIDS patients

- South Africa (2000–2006) 67 HIV- infected with Stroke
  - 96% pts. Ischemic strokes
  - 91% were younger than 46 years
  - opportunistic infections- 37%, most common infection was tuberculosis (15%)
  - HIV-associated vasculopathy-20%
  - Cardioembolism- (14%) patients
  - At the time of their stroke, 46% of these patients had CD4 counts < 200 cells/mm3
  - Traditional vascular risk factors were uncommon in these HIV-infected patients with stroke

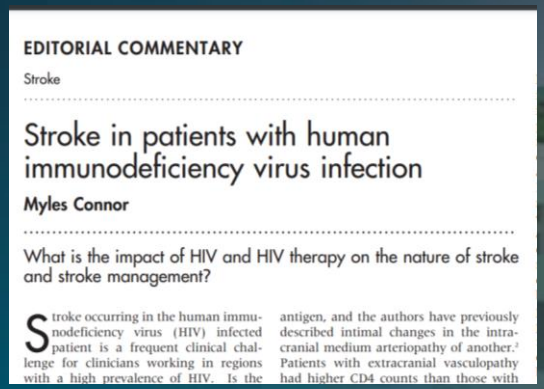
Tipping B et.al. J Neurol Neurosurg Psychiatry 2007;78:1320–1324






Stroke in Infections

1- Viral
- HIV
- HZV



EDITORIAL COMMENTARY

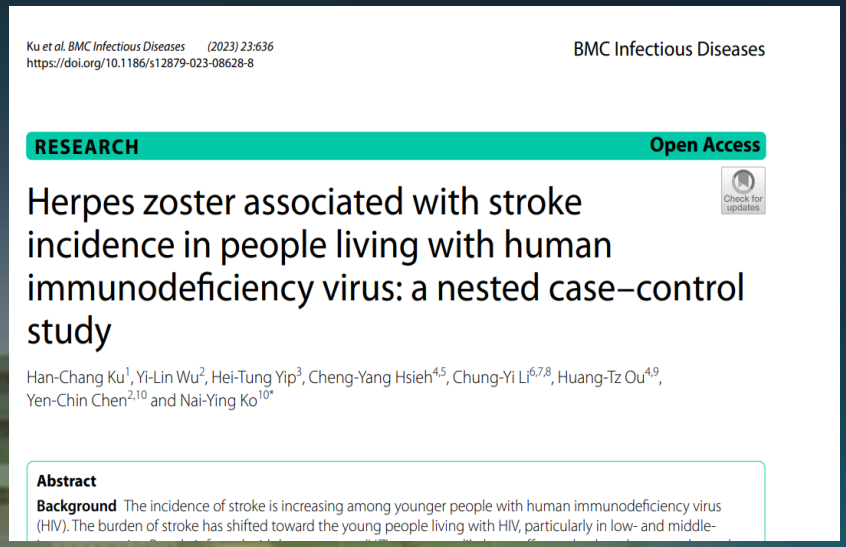
Stroke

Stroke in patients with human immunodeficiency virus infection

Myles Connor

What is the impact of HIV and HIV therapy on the nature of stroke and stroke management?

Stroke occurring in the human immunodeficiency virus (HIV) infected patient is a frequent clinical challenge for clinicians working in regions with a high prevalence of HIV. Is the antigen, and the authors have previously described intimal changes in the intracranial medium arteriopathy of another.[1] Patients with extracranial vasculopathy had higher CD4 counts than those with



Ku *et al. BMC Infectious Diseases* (2023) 23:636
https://doi.org/10.1186/s12879-023-08628-8

BMC Infectious Diseases

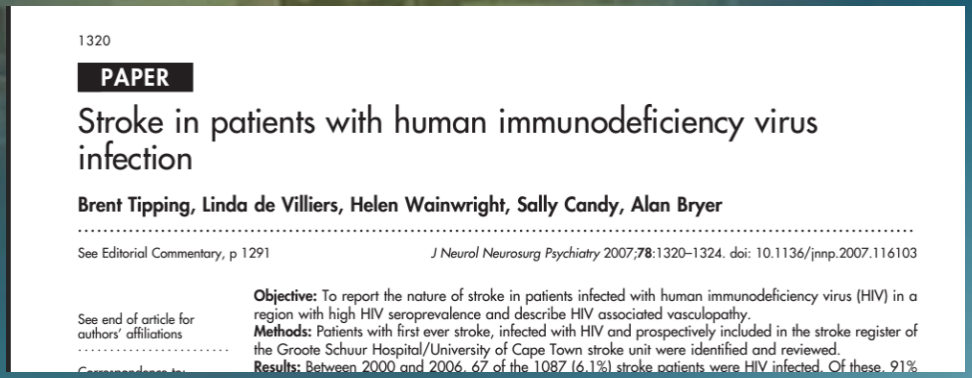
RESEARCH Open Access

Herpes zoster associated with stroke incidence in people living with human immunodeficiency virus: a nested case–control study

Han-Chang Ku[1], Yi-Lin Wu[2], Hei-Tung Yip[3], Cheng-Yang Hsieh[4,5], Chung-Yi Li[6,7,8], Huang-Tz Ou[4,9], Yen-Chin Chen[2,10] and Nai-Ying Ko[10*]

Abstract

Background The incidence of stroke is increasing among younger people with human immunodeficiency virus (HIV). The burden of stroke has shifted toward the young people living with HIV, particularly in low- and middle-



1320

PAPER

Stroke in patients with human immunodeficiency virus infection

Brent Tipping, Linda de Villiers, Helen Wainwright, Sally Candy, Alan Bryer

See Editorial Commentary, p 1291

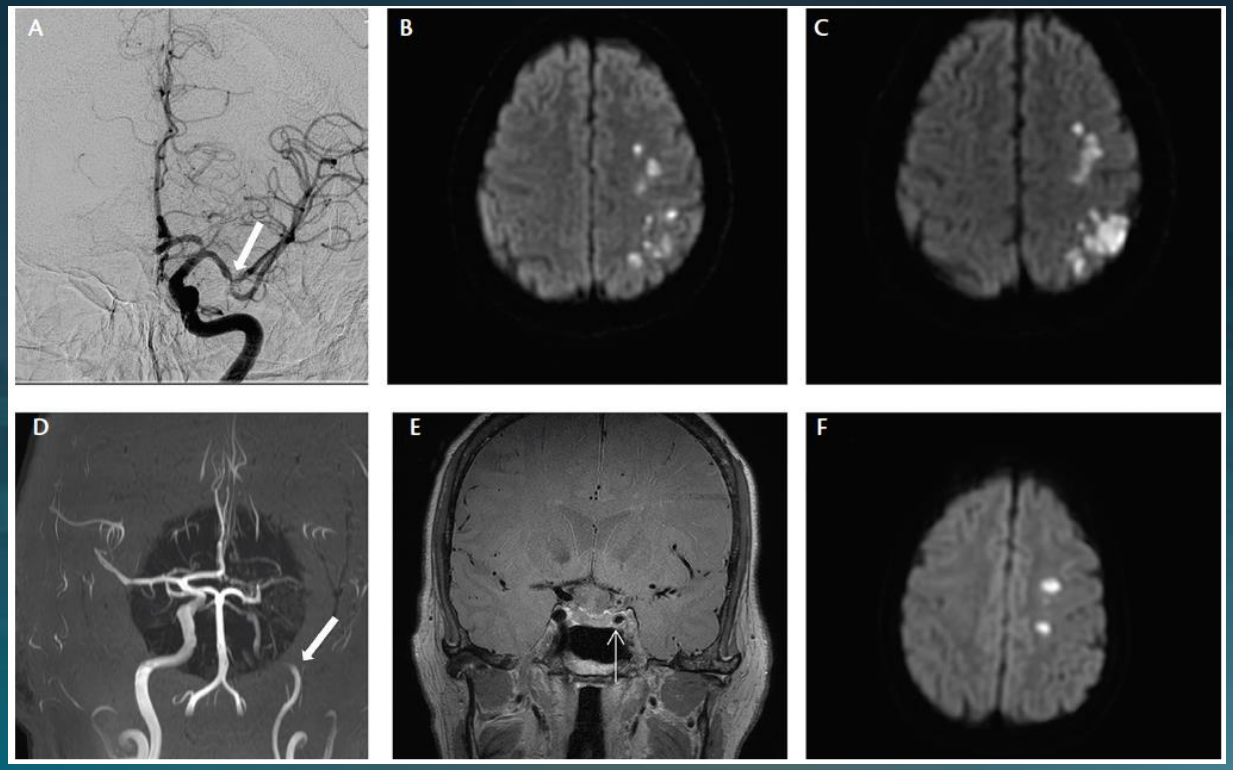
J Neurol Neurosurg Psychiatry 2007;**78**:1320–1324. doi: 10.1136/jnnp.2007.116103

See end of article for authors' affiliations

Objective: To report the nature of stroke in patients infected with human immunodeficiency virus (HIV) in a region with high HIV seroprevalence and describe HIV associated vasculopathy.
Methods: Patients with first ever stroke, infected with HIV and prospectively included in the stroke register of the Groote Schuur Hospital/University of Cape Town stroke unit were identified and reviewed.
Results: Between 2000 and 2006, 67 of the 1087 (6.1%) stroke patients were HIV infected. Of these, 91%

J Neurol Neurosurg Psychiatry 2007;78:1291.
doi: 10.1136/jnnp.2007.122416

VZV vasculopathy



Stroke and TB

Table 2. Summary Estimates of Stroke in Tuberculous Meningitis per Country

Region/country	No. of studies	Patients with stroke, No.	Total patients, No.	Point estimate (95% CI)
Middle East and North Africa				
Saudi Arabia	1	6	80	0.08
North America				
US	1	95	806	0.12
Latin America and the Caribbean				
Argentina	1	25	65	0.38
Ecuador	1	72	310	0.23
Mexico	1	11	24	0.46
East Asia and Pacific				
China	5	154	568	0.27 (0.22-0.31)
Hong Kong	2	29	104	0.28 (0.19-0.36)
Korea	1	8	38	0.21
Malaysia	2	46	93	0.46 (0.10-0.85)
New Zealand	1	34	104	0.33
Vietnam	2	51	147	0.35 (0.27-0.42)
Europe and Central Asia				
France	1	50	90	0.56
Italy	2	17	99	0.17 (0.10-0.24)
Netherlands	1	164	554	0.30
Spain	1	4	29	0.14
Turkey	6	91	1046	0.09 (0.05-0.15)
United Kingdom	1	12	25	0.48
South Asia				
India	20	567	1904	0.32 (0.25-0.38)
Pakistan	3	351	1228	0.29 (0.25-0.33)
Taiwan	7	117	392	0.31 (0.21-0.42)
Thailand	1	16	43	0.37
Sub-Saharan Africa				
South Africa	10	274	711	0.40 (0.25-0.55)
Total	71	2194	8460	0.30 (0.26-0.33)






Sy MCC, Espiritu AI, Pascual JLR. Global Frequency and Clinical Features of Stroke in Patients With Tuberculous Meningitis: A Systematic Review. *JAMA Netw Open.* 2022;5(9):e2229282. doi:10.1001/jamanetworkopen.2022.29282

Stroke with TBM

Zhang *et al. BMC Infectious Diseases* (2019) 19:362
https://doi.org/10.1186/s12879-019-4004-5

BMC Infectious Diseases

RESEARCH ARTICLE Open Access

Acute ischemic stroke in young adults with tuberculous meningitis

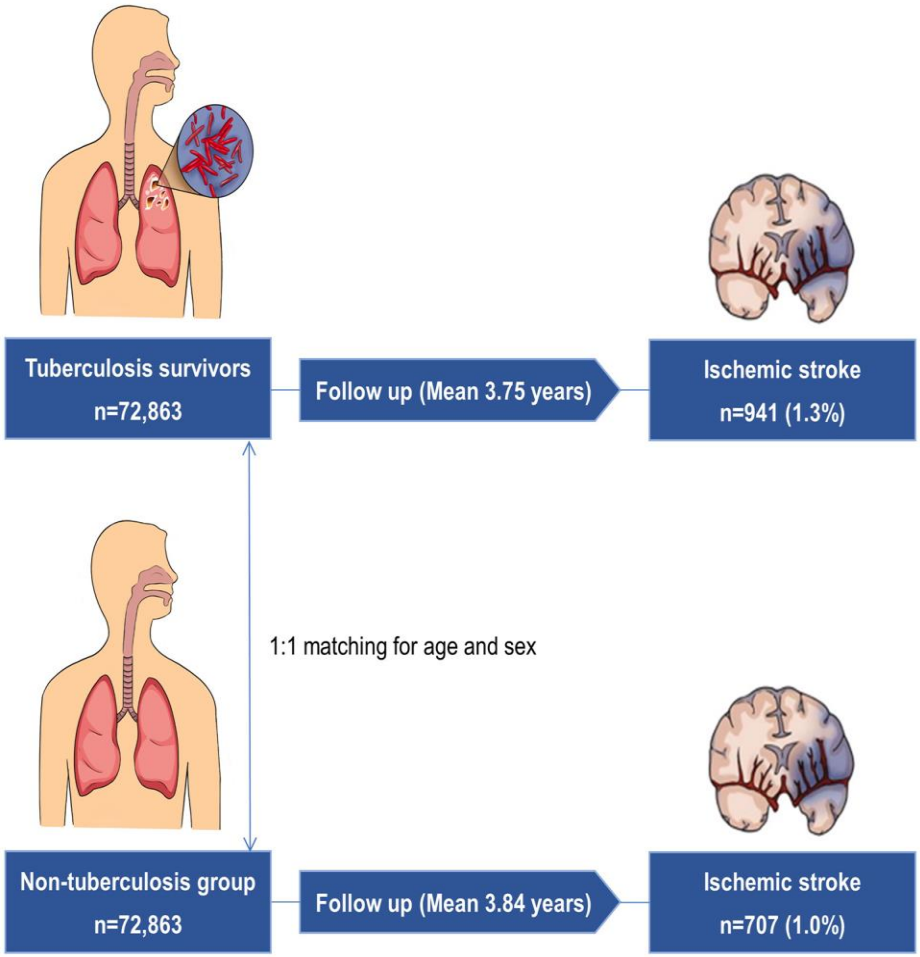
Liming Zhang[1], Xiaoyu Zhang[2], Huaqiang Li[1,3], Gang Chen[1*] and Meijia Zhu[2*]

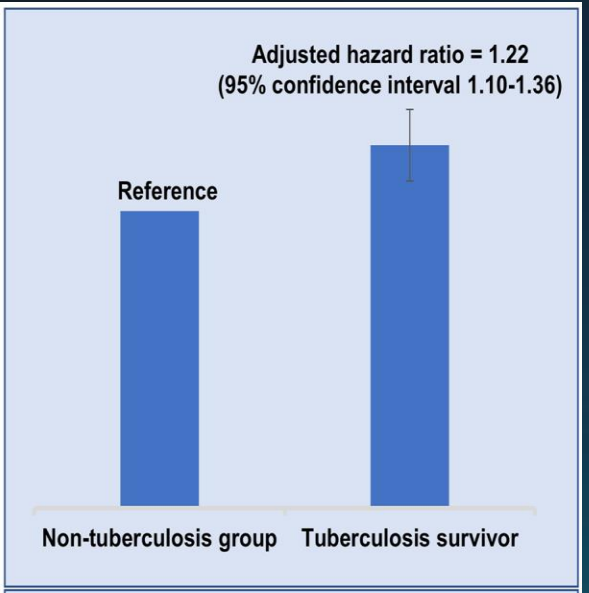
Abstract

Background: Ischemic stroke is a common complication in patients with tuberculous meningitis (TBM), which is associated with poor clinical outcome. However, risk factors of stroke in TBM patients were not fully understood, especially in those young adults. Therefore, the aim of our study was to identify risk factors for acute ischemic stroke in young adults with TBM.

Methods: TBM patients (18 to 50 years) without cerebral vascular risk factors were prospective recruited between Feb 2014 and Dec 2017. Patients were defined as stroke group and non-stroke group by brain magnetic resonance imaging (MRI). Demographic characteristics, clinical presentations, cerebrospinal fluid (CSF) examination, basal meningeal enhancement, hydrocephalus, tuberculoma and clinical outcome were compared between two groups. Binary logistic regression was performed to determine risk factors for acute ischemic stroke in young TBM patients.

Results: Fifty-two patients with TBM were included and 12 (23.1%) patients were in stroke group. Patients in stroke group were older. Clinical presentations were comparable between two groups except headache was more





Summary

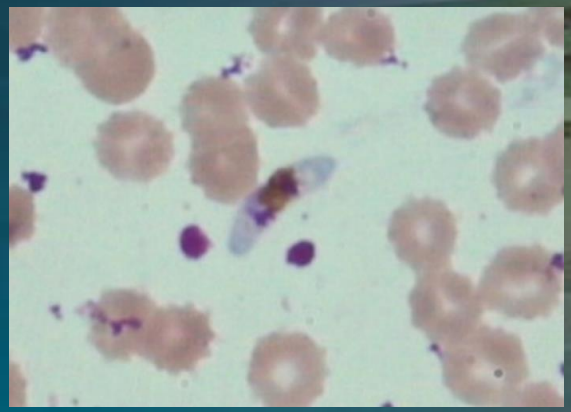
- We used Korean National Health Insurance data which covers almost entire Korean population (~50 million).
- A total of 72,863 tuberculosis survivors diagnosed between 2010-2017 and finished treatment were enrolled and 1:1 matched with non-tuberculosis comparison group by age and sex.
- During a median F/U of 3.8 years, 1.3% of tuberculosis survivors (941/72,863) and 1.0% of matched non-tuberculosis cases (707/72,863) developed ischemic stroke.
- The overall risk of ischemic stroke was higher in tuberculosis survivors (adjusted hazard ratio: 1.22, 95% confidence interval: 1.10–1.36) compared to the matched non-tuberculosis group.

Han Rim Lee. Stroke. Tuberculosis and Risk of Ischemic Stroke: A Nationwide Cohort Study, Volume: 53, Issue: 11, Pages: 3401-3409, DOI: (10.1161/STROKEAHA.122.039484)

Stroke in Infections

Protozoal:

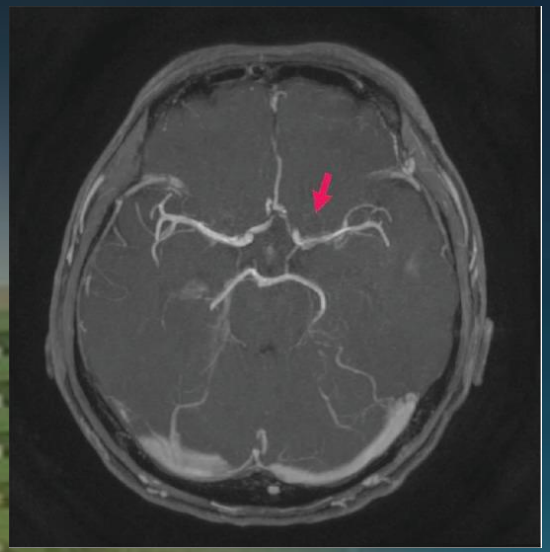
Malaria: very rare cause of stroke

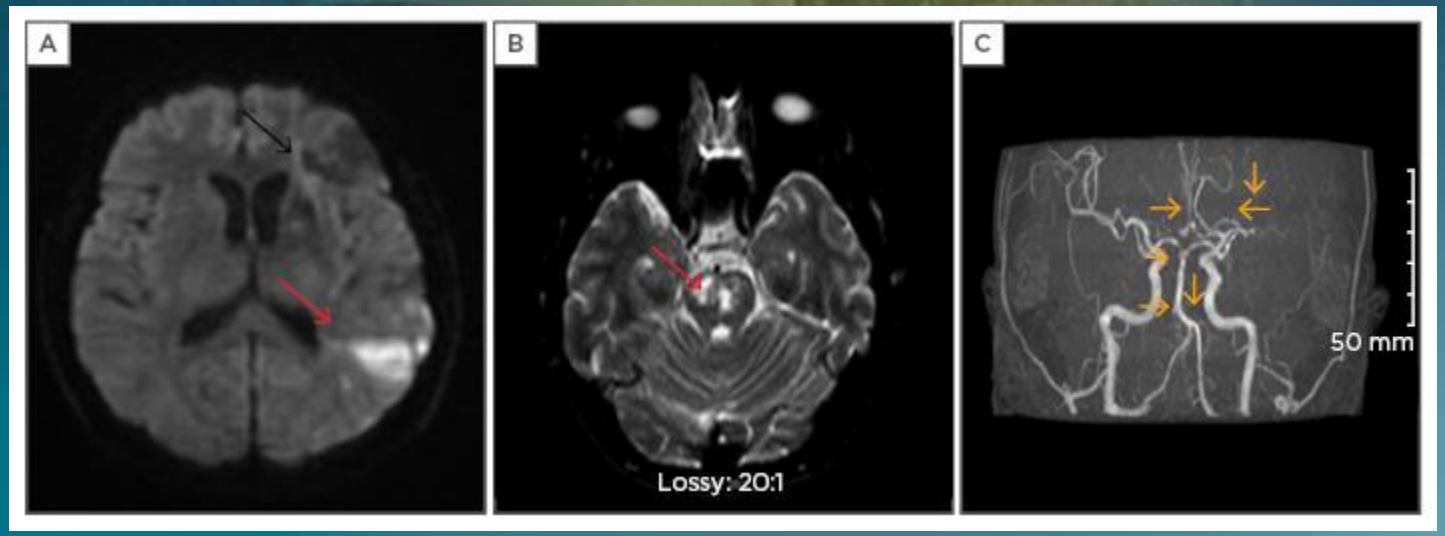


Stroke in Infections

3- Spirochaetal
Syphilis
HIV and Syphilis

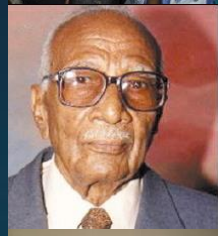
(The great Imitator)





Meningovascular syphilis

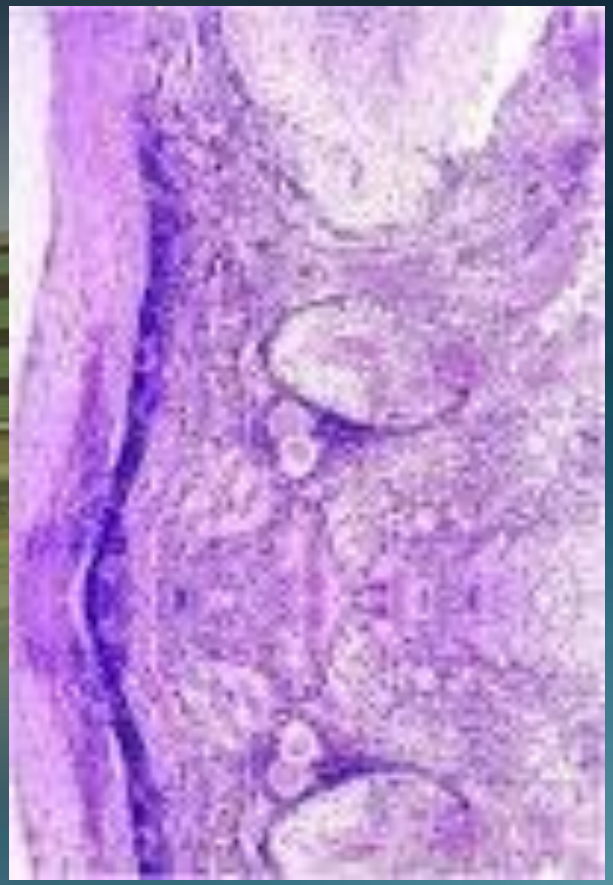
مستشفى سوبا الجامعي
Soba University Hospital



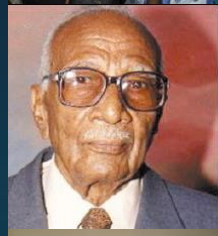
قسم علوم المخ والأعصاب
Department of Neurosciences

كلية الطب
FACULTY OF MEDICINE

Late Syphilis



Argyll Robertson pupils



COVID-19 and Stroke

During the follow-up period, recovered COVID-19 patients showed an increased risk of ischemic stroke (HR: 2.06, 95% CI: 1.75–2.41, p < 0.0001, I^2 = 63.7%) compared to subjects who did not experience COVID-19 infection but developed ischemic stroke over the same period

ARTICLES
https://doi.org/10.1038/s41591-022-02001-z
nature medicine

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OPEN

Long-term neurologic outcomes of COVID-19

Evan Xu[1], Yan Xie[1,2,3] and Ziyad Al-Aly[1,2,4,5,6] ✉

The neurologic manifestations of acute COVID-19 are well characterized, but a comprehensive evaluation of postacute neurologic sequelae at 1 year has not been undertaken. Here we use the national healthcare databases of the US Department of Veterans Affairs to build a cohort of 154,068 individuals with COVID-19, 5,638,795 contemporary controls and 5,859,621 historical controls; we use inverse probability weighting to balance the cohorts, and estimate risks and burdens of incident neurologic disorders at 12 months following acute SARS-CoV-2 infection. Our results show that in the postacute phase of COVID-19, there was increased risk of an array of incident neurologic sequelae including ischemic and hemorrhagic stroke, cognition and memory disorders, peripheral nervous system disorders, episodic disorders (for example, migraine and seizures), extrapyramidal and movement disorders, mental health disorders, musculoskeletal disorders, sensory disorders, Guillain–Barré syndrome, and encephalitis or encephalopathy. We estimated that the hazard ratio of any neurologic sequela was 1.42 (95% confidence intervals 1.38, 1.47) and burden 70.69 (95% confidence intervals 63.54, 78.01) per 1,000 persons at 12 months. The risks and burdens were elevated even in people who did not require hospitalization during acute COVID-19. Limitations include a cohort comprising mostly White males. Taken together, our results provide evidence of increased risk of long-term neurologic disorders in people who had COVID-19.

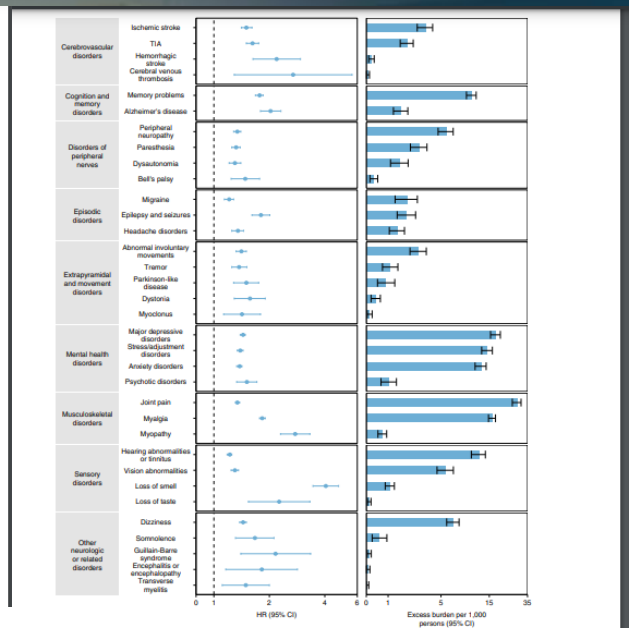


Fig. 2 | Risks and 12-month burdens of incident postacute COVID-19 neurologic outcomes compared with the contemporary control cohort. Outcomes were ascertained 30 days after the COVID-19-positive test until the end of follow up. COVID-19 cohort (n = 154,068) and contemporary control cohort (n = 5,638,795). Adjusted HRs (dots) and 95% (error bars) CIs are presented, as are estimated excess burdens (bars) and 95% CIs (error bars). Burdens are presented per 1,000 persons at 12 months of follow up. The dashed line marks a HR of 1.00; lower limits of 95% CIs with values greater than 1.00 indicate significantly increased risk.

Parkinson-like disease (HR 1.50 (1.28, 1.75); burden 0.89 (0.50, 1.34)), dystonia (HR 1.57 (1.29, 1.90); burden 0.40 (0.21, 0.63)) and myoclonus (HR 1.42 (1.13, 1.79); burden 0.14 (0.04, 0.26)). The respective risk and burden of a composite of these extrapyramidal and movement disorders were 1.42 (1.34, 1.50) and 3.98 (3.24, 4.77).

Mental health disorders. Mental health disorders included major depressive disorders (HR 1.44 (1.39, 1.48); burden 17.28 (15.43, 19.18)), stress and adjustment disorders (HR 1.39 (1.34, 1.44); burden 14.34 (12.66, 16.07)), anxiety disorders (HR 1.38 (1.33, 1.42); burden 12.44 (10.93, 13.99)) and psychotic disorders (HR 1.51

COVID-19 and Stroke

Marco Zuin[1,2], Maria Mazzitelli[3], Gianluca Rigatelli[4], Claudio Bilato[2] and Anna Maria Cattelan[3]

Abstract

Background: Data regarding the risk of ischemic stroke within 1 year after the post-acute phase of COVID-19 remain scant. We assess the risk of ischemic stroke in COVID-19 survivors after SARS-CoV-2 infection by performing a systematic review and meta-analysis of the available data.

Methods: Following the PRISMA guidelines, we searched Medline and Scopus to locate all articles published up to February 11, 2023, reporting the risk of incident ischemic stroke in adult patients recovered from COVID-19 infection compared to non-infected patients (controls) defined as those who did not experience the infection over the same follow-up period. Ischemic stroke risk was evaluated using the Mantel–Haenszel random effects models with adjusted Hazard ratio (HR) as the effect measure with 95% confidence interval (CI) while heterogeneity was assessed using Higgins I^2 statistic.

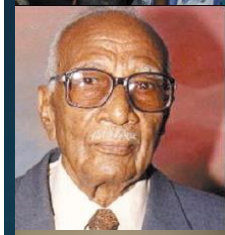
Results: Overall, 23,559,428 patients (mean age 56, 1 year, 54.3% males), of whom 1,595,984 had COVID-19, were included. Over a mean follow-up of 9.2 months, ischemic stroke occurred in 4.40 [95% CI: 4.36–4.43] out of 1000 patients survived to COVID-19 compared to 3.25 [95% CI:3.21–3.29] out of 1000 controls. Recovered COVID-19 patients presented a higher risk of ischemic stroke ((HR: 2.06, 95% CI: 1.75–2.41, $p < 0.0001$, $I^2 = 63.7\%$) compared to people who did not have COVID-19. COVID-19 patients hospitalized at the time of the infection have a subsequent higher risk of stroke during the follow-up compared to those non-hospitalized.

Conclusions: Recovered COVID-19 patients have a higher risk of ischemic stroke compared to subjects from the general population within 9 months from the index infection.

Keywords

Stroke, COVID-19, long-COVID

Date received: 5 June 2023; accepted: 6 July 2023



Neuro-helminthiasis

Neurocysticercosis
Paragonimiasis

Paragonimiasis

Paragonimiasis is a parasitic infection with a flatworm which may enter the body through eating undercooked crab or crayfish.

It is rare in the United States, though several cases have been reported in the Midwest.

Most commonly it is found in East Asian countries.29

Centrse for Disease Control & Prevention. Parasites :

The parasite does not often affect the central nervous system but the parasite may reach the brain either through the bloodstream or through the foramina at the base of the skull.

The adult form of the parasite both releases inflammatory substances and tunnels through tissues, which can result in headaches, seizures, and **strokes.**

ELSEVIER

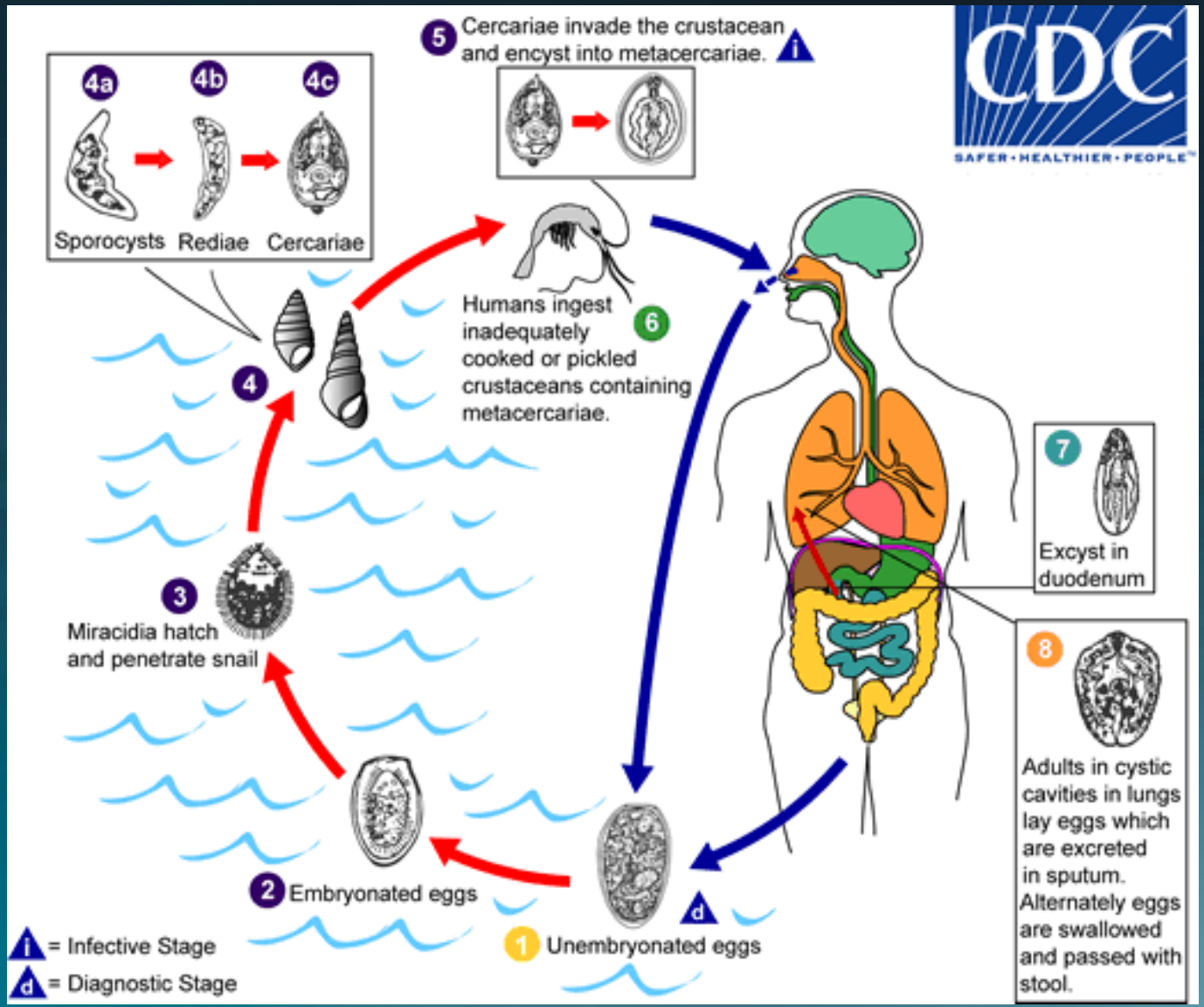
Transactions of the Royal Society of Tropical Medicine and Hygiene

Volume 86, Issue 4, July–August 1992, Page 417

Short report

Therapeutic effect of triclabendazole in patients with paragonimiasis in Cameroon: a pilot study

C. Ripert [1], B. Couprie [1], R. Moyou [2], F. Gaillard [1], M. Appriou [1], J. Tribouley-Duret [1]


5 Cercariae invade the crustacean and encyst into metacercariae.
i
4a
4b
4c
Sporocysts
Rediae
Cercariae
CDC
SAFER · HEALTHIER · PEOPLE™
Humans ingest inadequately cooked or pickled crustaceans containing metacercariae.
6
4
7
Excyst in duodenum
3
Miracidia hatch and penetrate snail
8
Adults in cystic cavities in lungs lay eggs which are excreted in sputum. Alternately eggs are swallowed and passed with stool.
2 Embryonated eggs
d
1 Unembryonated eggs
i = Infective Stage
d = Diagnostic Stage






Stroke in Infections

5- Post infectious Angiitis






UNUSUAL CAUSES OF STROKE

Infectious Causes of Stroke

Stroke is an often-devastating and not uncommon complication of many central nervous system infections.

By Jennifer E. Fugate, DO

Every year, an estimated 15 million people experience stroke worldwide. The impact and burden of strokes are substantial–one-third of these individuals (5 million) die from stroke

prodromal symptoms over weeks to months, including headache, malaise, and/or personality and behavioral changes. Neurosyphilis should be considered in any young adult with stroke who lacks traditional cerebrovascular risk factors, par

Neurocrit Care. 2010 Jun;12(3):369-74. doi: 10.1007/s12028-010-9335-4.

Infectious vasculopathy of intracranial large- and medium-sized vessels in neurological intensive care unit: a clinico-radiological study

J Katchanov [1], E Siebert, R Klingebiel, M Endres

Affiliations + expand

PMID: 20146025 DOI: 10.1007/s12028-010-9335-4

Abstract

Background: Infections are a well-known cause of cerebral vasculopathy and vasculitis. We aimed to analyze the frequency of intracranial vasculopathy attributable to infection, the spectrum of causative microorganisms, imaging, and cerebrospinal fluid (CSF) characteristics as well as clinical course and outcome.

Methods: We used our institution's medical record system to identify all patients diagnosed with nonatherosclerotic central nervous system vasculopathy from January 1, 1999 through February 28, 2009. We reviewed their clinical charts, imaging data, and results of CSF studies.

Cerebrovascular Diseases

Volume 26, Issue 5
November 2008

REVIEW ARTICLES | SEPTEMBER 23 2008

Cerebral Vasculitis and Stroke in Lyme Neuroborreliosis:

Two Case Reports and Review of Current Knowledge

Subject Area: Cardiovascular System , Neurology and Neuroscience

Raffi Topakian; Karl Stieglbauer; Karin Nussbaumer; Franz T. Aichner

Cerebrovasc Dis (2008) 26 (5): 455–461.
https://doi.org/10.1159/000155982 Article history

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جامعة أطور


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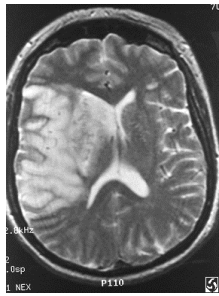
قسم علوم المخ والأعصاب
Internal carotid artery
Middle cerebral artery
Circle of Willis
Basilar artery
Bottom view of brain



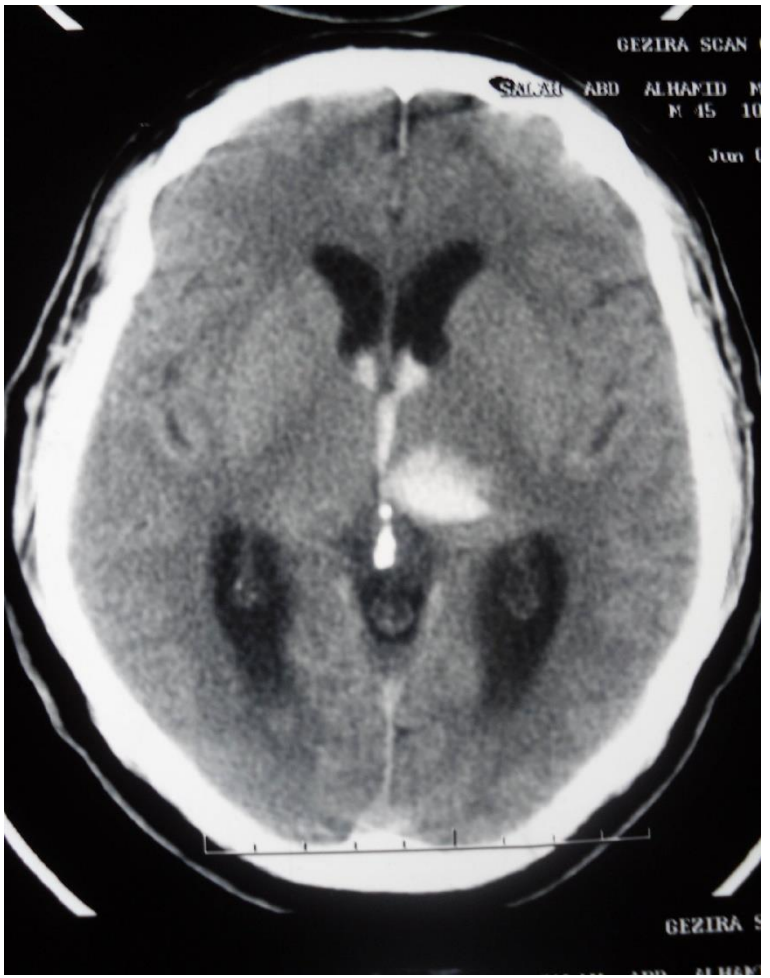
Stroke Mimics

Other Conditions presenting as Stroke

- Toxic/metabolic disturbance such as:
  - Hypoglycaemia
  - Drug and alcohol toxicity.
- Conditions which can cause dizziness or disturbed balance such as:
  - Syncope
  - Labyrinthine disorders — vertigo, Meniere's disease,
- Neurological conditions such as:
  - Seizure
  - Migraine with aura
  - Demyelination — multiple sclerosis
  - Peripheral neuropathies such as Bell's palsy
  - Spinal epidural haematoma
- Trauma
- Systemic or local infection including:
  - Central nervous system abscess
  - Encephalitis
  - Sepsis
- Encephalopathies such as:
  - Hypertensive encephalopathy
  - Wernicke's encephalopathy
- Space occupying lesions including:
  - Tumour
  - Subdural haematoma
- Other conditions such as:
  - Acute confusional state
  - Dementia
  - Vasculitis
  - Functional

Stroke Mimics

Haemorrhagic Met. From Thyroid Ca

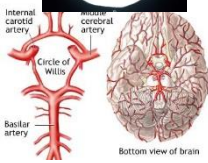


28 yr M brought from Prison. Multiple head Injuries

Stroke Chameleon

Stroke that presents like other conditions

- Seizures:
  - e.g. Temporal lobe infarct presenting as Non convulsive SE
- Transient Amnesia (TGA)
- Dysphasia/ aphasia (MCA)
- Acute Delirium
- Acute paraparesis/ Monoparesis
  - Ant Spinal Artery Syndrome – Myelopathy in Spinal Dural fistula
- Vertigo / Loss of Balance (POCS) – BPPV
- Severe migraine / Thunderclap Headache
- Visual loss-
  - Unilateral (Amaurosis fugax)
  - Bilateral(cortical blindness)
- Abnormal Movements:
  - Hemiballismus/ Rubral tremor/ ataxic tremor



EMJ Online First, published on July 20, 2010 as 10.1136/emj.2009.075788

Emergency casebook

Acute mutism: a useful lesson

Melissa Maguire,[1] Osheik Seidi,[1] Mark Baker,[1] Arun Gupta,[2] Cyrus Muwanga[3]

a regional infarct in the supply area of the left middle cerebral artery, with a trace of haemorrhagic component (figure 1B). MR angiograms of both the extracranial and intracranial major vessels did not reveal any abnormalities. An autoimmune screen

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EMJ Online First, published on July 20, 2010 as 10.1136/emj.2009.075788
Emergency casebook

Acute mutism: a useful lesson

Melissa Maguire,[1] Osheik Seidi,[1] Mark Baker,[1] Arun Gupta,[2] Cyrus Muwanga[3]

Broca's aphasia may manifest as mutism in some patients. Mutism may be misinterpreted as part of a depressive illness in patients with a psychiatric history. We report on a patient presenting with acute mutism who had a history of amphetamine and cannabis abuse and was later found to have sustained an infarct of the dorsolateral frontal cortex. Recognition of this mode of presentation will aid early diagnosis and treatment.

A 40-year-old right-handed man presented to casualty with a 3-day history of mutism. He had had an altercation with his partner and had left the house to stay with a friend. Later that evening he was found mute, sitting on the sofa having been incontinent of urine. During the next two days he was cared for by his partner but remained mute, with minimal interactions with others.

He had a history of depression since April 2007, following the death of his mother. He had been low in mood and was consuming large quantities of amphetamine (1 ounce per week) and diazepam (100 'street' tablets per week), smoking cannabis and drinking eight cans of lager a day. A few weeks before admission he had tried to overdose with diazepam. He had a history of self-harm and drug overdoses. He had been diagnosed with anti-social personality disorder and had a forensic history of domestic violence. There was no relevant past medical history. There was a family history of depression. He smoked 20 cigarettes per day.

On admission to casualty he could not communicate verbally but could respond to questioning through gesture. His temperature was 37.2°C, blood pressure 128/78 mm Hg and pulse 78 beats per minute and regular. His score on the Glasgow Coma Scale was 15/15, and on initial assessment no evidence of head injury or abnormal neurological findings had been reported. Toxicology screening of urine was positive for cannabis and benzodiazepines. Blood investigations revealed neutrophilia (11.5×10^9/l) and leucocytosis (14×10^9/l) with normal inflammatory markers and no other abnormalities.

He was referred to psychiatric services with mutism thought to be secondary to a depressive illness. He was communicating with the staff through gestures and writing. Objectively his mood was low but reactive. Thoughts, perception, cognition, judgement and insight were difficult to assess due to his mutism. Formal neuropsychological assessment was not permitted, because of difficulties in communication. During his admission he started to produce a few words with perseverance of speech. This prompted an urgent CT brain scan and referral to neurology services.

Further neurological examination revealed normal phonation, partial receptive dysphasia and complete expressive dysphasia. He had right upper motor neuron facial weakness, right upper limb dyspraxia, right hemi-neglect and right-sided pathologically brisk reflexes. Both plantar responses were flexor. Sensory examination was not possible and no cerebellar or extrapyramidal signs were detected. The general examination was normal, with no carotid bruits observed.

The CT brain scan showed a 3.7 cm×3.9 cm low attenuation area in the left dorsolateral frontal cortex (figure 1A). Subsequent MR imaging confirmed a regional infarct in the supply area of the left middle cerebral artery, with a trace of haemorrhagic component (figure 1B). MR angiograms of both the extracranial and intracranial major vessels did not reveal any abnormalities. An autoimmune screen was negative.

This patient presented with acute mutism secondary to a dorsolateral frontal cortex infarct, likely related to cerebral vasculopathy as a result of chronic amphetamine and cannabis abuse. The patient was given aspirin and simvastatin and was advised to stop smoking and using illicit drugs. He was referred to speech and language services and has since made a good recovery of his speech. He is continuing psychotherapy and support from the substance abuse services.

Acute mutism may occur in both organic and non-organic disease and may cause diagnostic difficulties. Important organic causes include head injury, encephalitis and lesions affecting the dorsolateral frontal cortex, causing Broca's aphasia. In this case, Broca's aphasia was caused by infarct attributable to misuse of amphetamine and cannabis, both of which have been implicated in stroke independently,[1 2] causing severe hypertension and vasospasm.[2] Arterial dissection can occur in amphetamine abuse.[3] Expression, naming and production of spontaneous speech are affected, resulting in non-fluent aphasia, with telegraphic speech and agrammatism. Comprehension appears relatively spared. Hypophonia, limb apraxia and hemiparesis may be present.

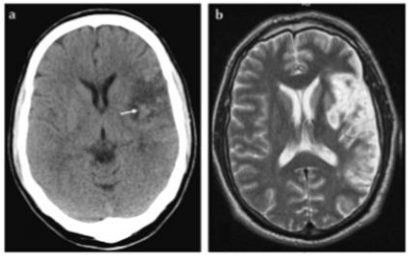

Figure 1 CT brain scan image (A) showing a large hypodense area in the left dorsolateral frontal (Broca's) area due to cerebral infarction, with hyperdensities representing haemorrhagic changes in the centre (white arrow). The same is confirmed on a T2 axial MRI (B), which revealed slight posterior extension of the infarct.

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Emergency casebook

Migraine Stroke

JOAMS (2012) Vol. 02, No.01 *Misbah & Seidi, Migraine Stroke, 2012*

Migraine Stroke in a young man from Sudan: a Case Report

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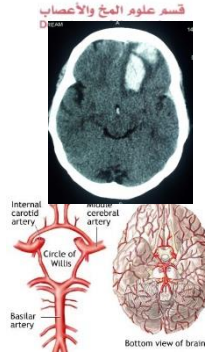
Case Report:

A 43 years old male Sudanese gentleman was known to suffer from migraine without aura from his teenage. He usually gets pain on both sides of his head that had a throbbing nature. It was intense at times interfering with his work and social life.

It was associated with intense photo- and phonophobia. other neurological or general signs of note. This defect

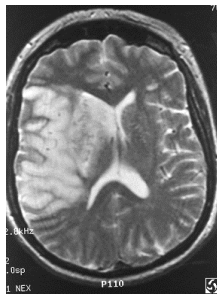


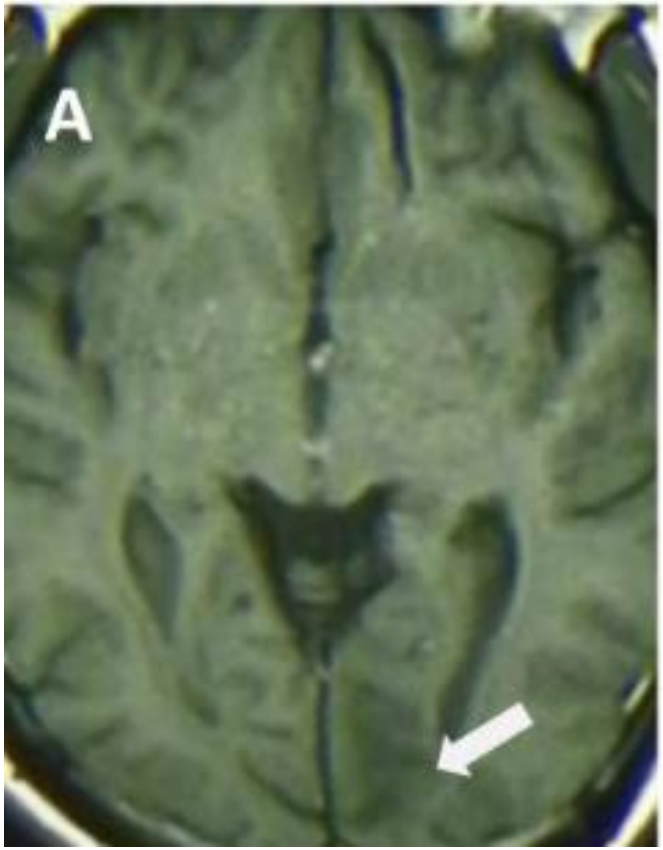
No established Guideline

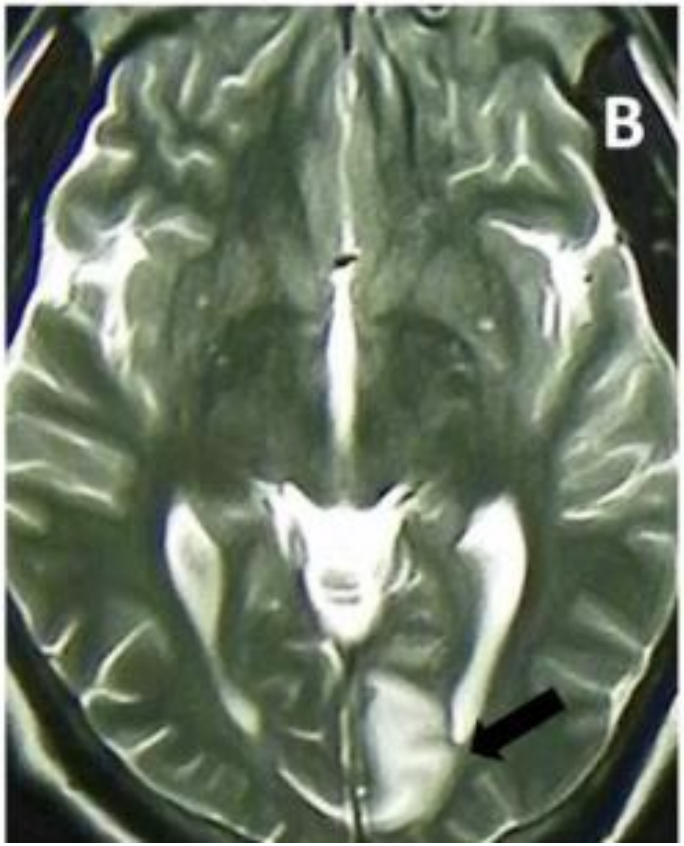
My recommendations:

- Closely monitor BP – avoid lowering unless > 200/110
- Manage pain

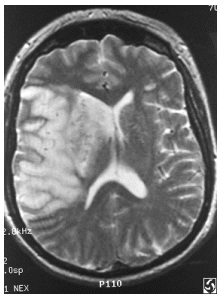
TRIPTAN are contraindicated



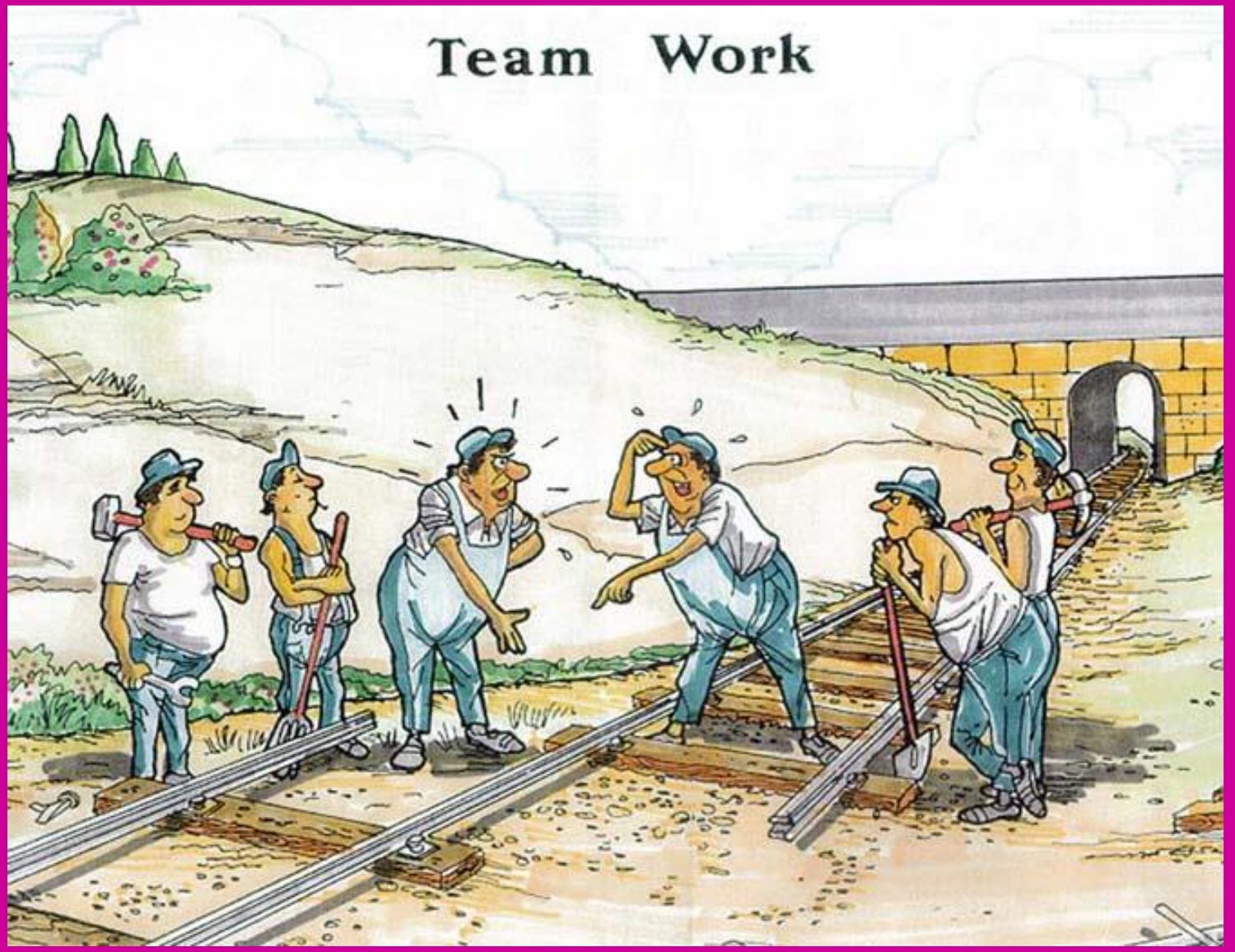

A


B


قسم علوم المخ والأعصاب
Internal carotid artery
Middle cerebral artery
Circle of Willis
Basilar artery
Bottom view of brain



OUTREACH TRIPS




Dementia


NO LA MAFUNZO YA
BONGO KUSINI MWA
SAHARA
-SAHARAN AFRICA
IFE SPAN
28 - 31 OKTOBA
2023

قسم علوم المخ والأعصاب
Department of Neuroscinces

Our Students & Youth = Our Future

مستشفى سوبا الجامعى
Soba University Hospital

Thank You

كلية الطب
FACULTY OF MEDICINE

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14th RTC -EAN
Dar es Salam, Tanzania
30th October 2023