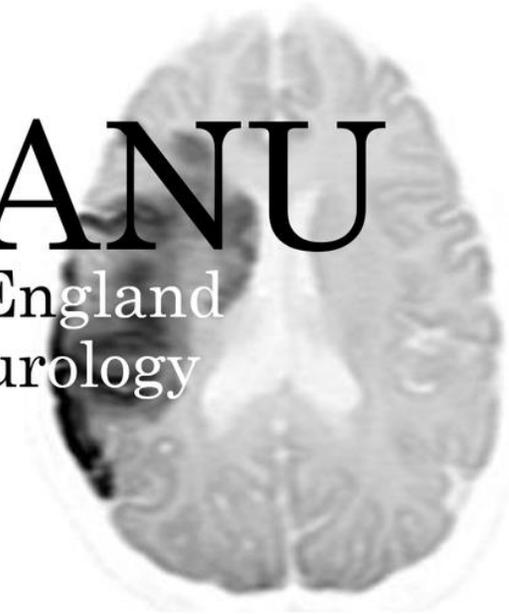


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Stroke – Masterclass in Presentation & Management

Dr Jane Molloy

Dr Tim Lavin



Overview

Overarching – Simple things done well

- Stroke classification / syndromes
- Illustrative cases / MCQs
- Questions

Stroke Types



- **Ischaemic 87%**
 - **Embolic, thrombotic**, hypoperfusion (think 'type 2'), **CVST**
 - We will cover those in **bold** only
 - (VITT – see current BSH guidance –
 - <https://b-s-h.org.uk/media/19590/guidance-version-17-on-mngmt-of-vitt-20210420.pdf>
- Haemorrhagic 13%

Stroke Classification



- Bamford (Oxford) classification
 - Based on clinical findings only
- Others have followed – eg TOAST
- Some limitations – but basics – can supplement with NIHSS and (MR) imaging (especially LACS, small PACS)

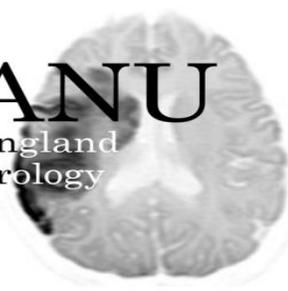
Bamford et al, JNNP 1988

Bamford Classification



- TACS – ALL 3 of:
 - Unilateral weakness (and/or sensory deficit) of the face, arm and leg
 - Homonymous hemianopia
 - Higher cerebral dysfunction (dysphasia, visuospatial disorder)
- PACS
 - 2 of the above 3

Bamford Classification



- POCS – any one of:
 - Cranial nerve palsy and a contralateral motor/sensory deficit
 - Bilateral motor/sensory deficit
 - Conjugate eye movement disorder (e.g. horizontal gaze palsy)
 - Cerebellar dysfunction (e.g. vertigo, nystagmus, ataxia)
 - Isolated homonymous hemianopia

Bamford Classification



- LACS – subcortical stroke (no ‘higher’ deficit) – 5 types:
 - Pure sensory stroke
 - Pure motor stroke
 - Sensori-motor stroke
 - Ataxic hemiparesis
 - Clumsy hand/dysarthria syndrome



Patient 1



Background

84yr old

R handed

PMHx of AF, CCF, IHD

mRS 1

Warfarin, Bisoprolol, Frusemide



Presentation

Low impact RTA

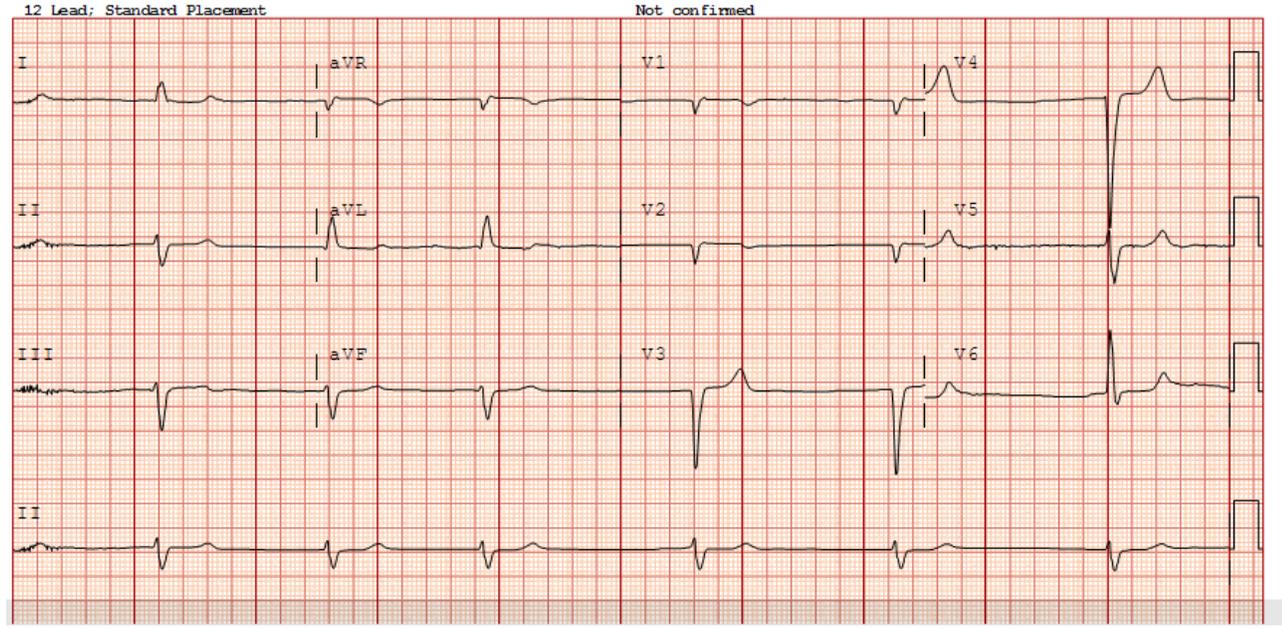
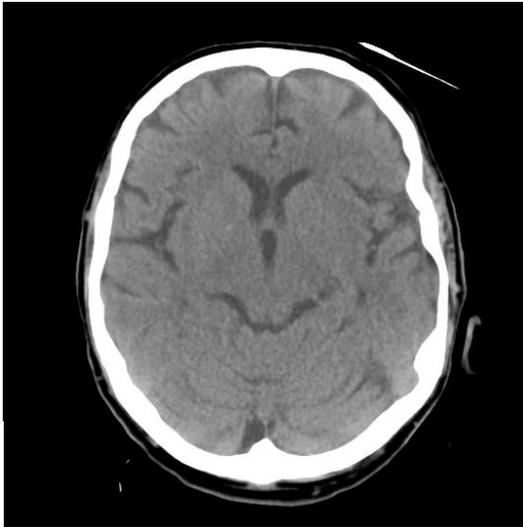
**Found with dense R sided weakness in
car**

Patient 1



- NIHSS 19
- Right Homonymous hemianopia
- Right dense hemiparesis
- Aphasic
- Eye deviation to left

Plain CT imaging and ECG



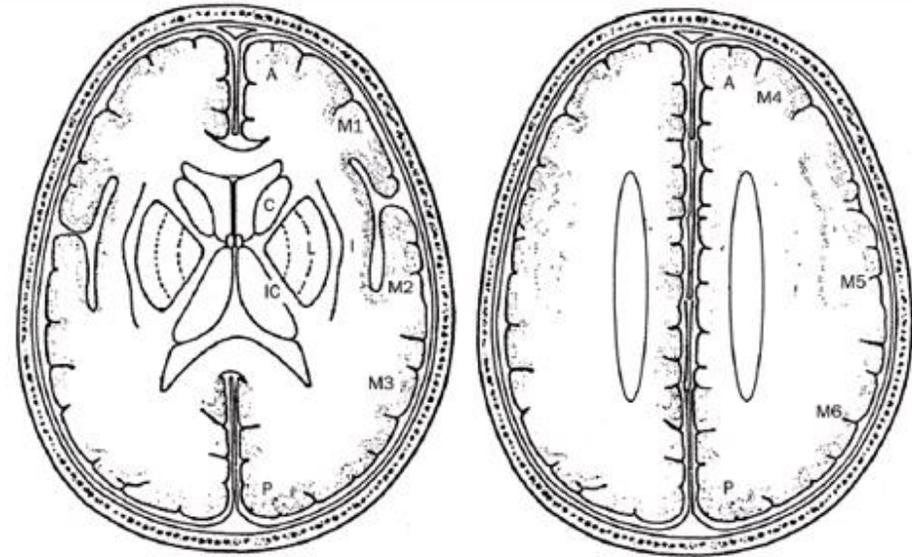
ECG – Slow AF

CT Imaging – hyperdense MCA and subtle asymmetry in the grey-white matter differentiation of the left corpus striatum, lentiform nucleus and internal capsule

ASPECTS (Alberta stroke program early CT score)

- Method of communicating early ischaemic change on CT
- Evaluated from 2 standard images
 - 3 subcortical regions
 - 7 cortical regions
- Limited to MCA territory
- In initial ASPECTS study -High score predicted good functional outcome following reperfusion

ASPECTS study form



The ASPECTS value is calculated from two standard axial CT cuts: one at the level of the thalamus and basal ganglia (left), and one just rostral to the basal ganglia (right). A: anterior circulation; P: posterior circulation; C: caudate; L: lentiform; IC: internal capsule; I: insular ribbon; MCA: middle cerebral artery; M1: anterior MCA cortex; M2: MCA cortex lateral to insular ribbon; M3: posterior MCA cortex; M4, M5, and M6 are anterior, lateral, and posterior MCA territories immediately superior to M1, M2, and M3, rostral to basal ganglia

Reproduced with permission from: Barber, PA, Demchuk, AM, Zhang, J, Buchan, AM. Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before thrombolytic therapy. ASPECTS Study Group. Alberta Stroke Programme Early CT Score. *Lancet* 2000; 355:1670. Copyright © 2000 The Lancet.

LEFT HEMISPHERIC TACS

Atrial Fibrillation

Early Imaging

Anticoagulation

Within treatment window for reperfusion

- Within 4.5 hrs of symptom onset, 18 years of age or older
- NIHSS => 6
- Pre-stroke mRS =< 2
- (ASPECTS 8)
- **Patient considered for tPA: no; Because: On warfarin**
- Potential candidate for IA procedure

CT Angiogram Arch to Circle of Willis

There is occlusion of the proximal M1 segment, measuring approximately 18mm. Some filling of the distal M2 branches suggesting moderate collateralisation.

Intraarterial Thrombectomy within 6hr

- IV alteplase causes effective recanalisation in 50% compared to 79% in IAT
- Eligibility
 - NIHSS greater than 5, older than 18, Pre-stroke mRS 0-2
 - CAROTID T, A1, M1 or a single M2 occlusion and ASPECTS > 7
 - Basilar occlusion

Intraarterial Thrombectomy 6-24hr

- Basilar or Vertebral Artery Occlusion
 - Trials did not include POCS- Larger trials are needed
 - Better collateral circulation
- MCA/ACA (DAWN and DEFUSE 3)
 - Require imaging based criteria - CT perfusion or MRI
 - Target mismatch between the ischaemic core and penumbra
 - Practically difficult

Benefits of IAT

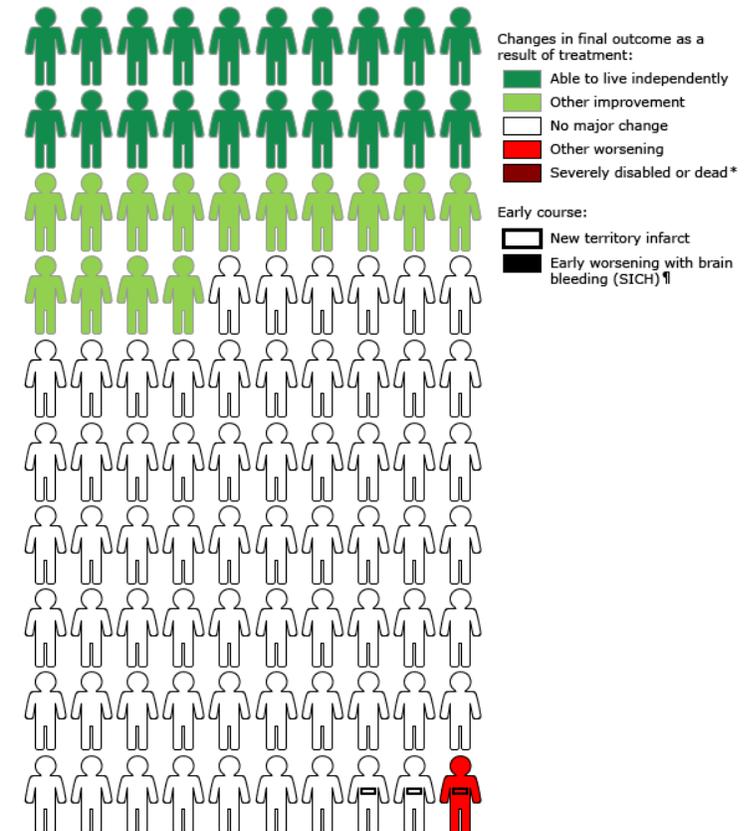
Early

- NNT to achieve functional independence- 3 to 7.5
- Meta-analysis > 1000 patients
 - 90-day mRS score of 0 to 2 - 46 vs 27%, interventional to control

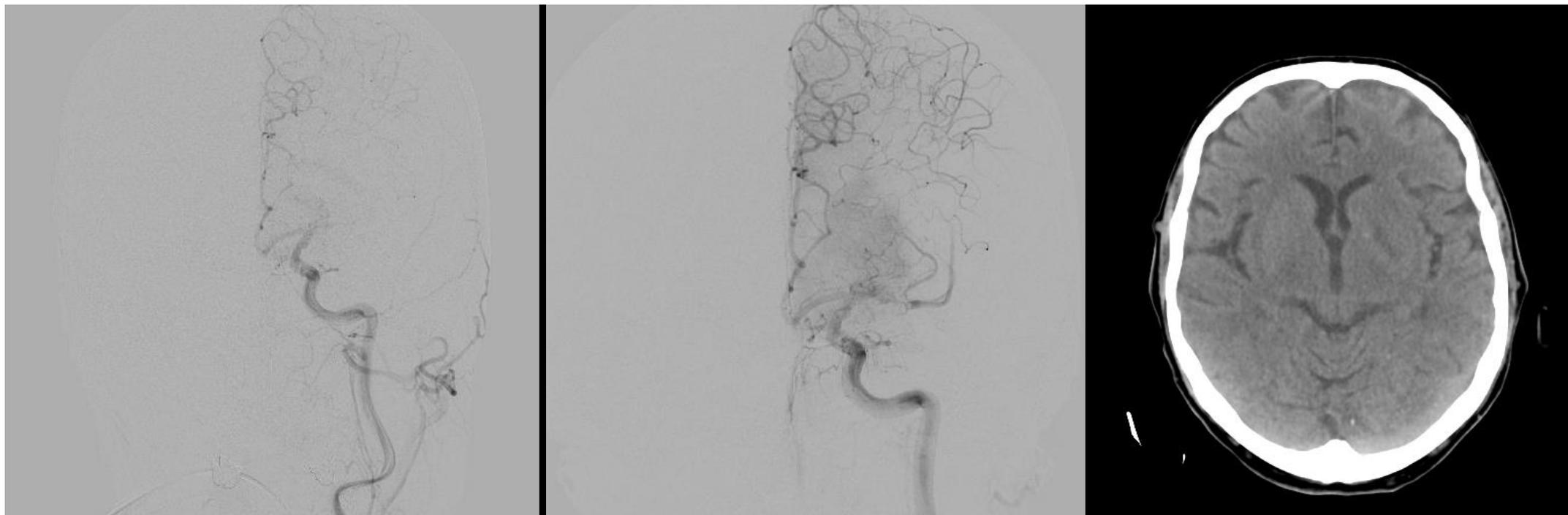
Late

- DAWN trial
 - 206 patients
 - Functional independence, mRS 0-2 49% vs 13%

Visual decision aid depicting the benefits and risks of endovascular thrombectomy added to IV tPA versus IV tPA alone



Progress





What about the AF?



AF and Ischaemic Stroke

- Cause of 20% ischaemic Strokes
- **Detection of AF following Stroke - 20%**
 - **routine ECG on admission**
 - **inpatient telemetry lasting 12–72 hours**
 - **further outpatient monitoring, usually for at least 1–7 days.**
- Neurogenic AF - lower risk

Indications for vitamin K antagonists over direct oral anticoagulants

Absolute

- Prosthetic (mechanical) heart valve
- Valvular atrial fibrillation (due to moderate or severe mitral stenosis, usually of rheumatic origin)
- Severe renal impairment

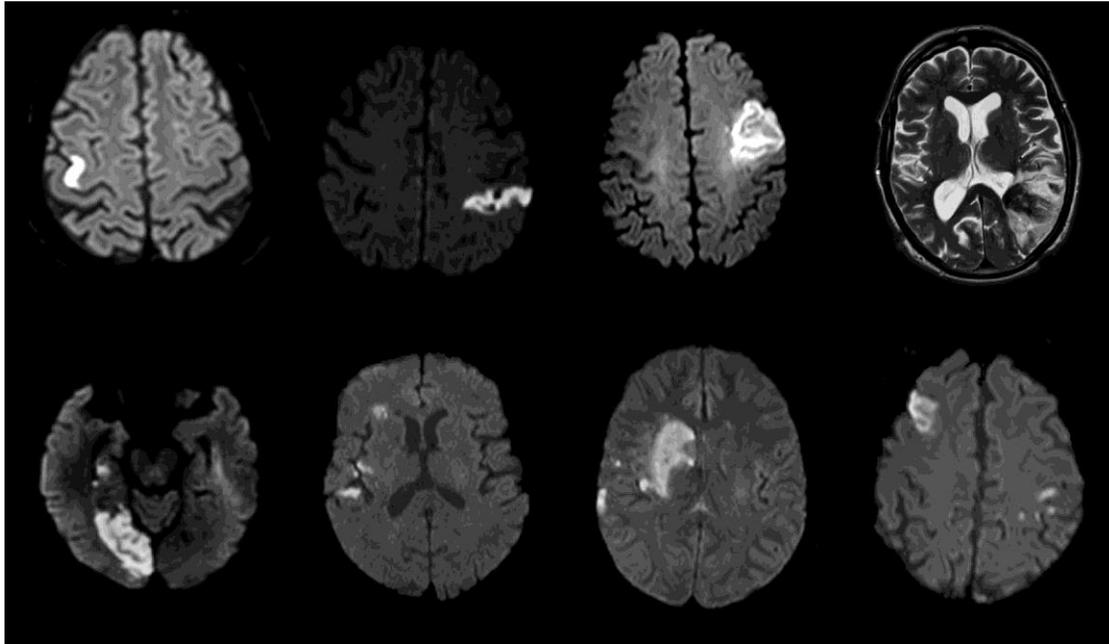
Relative

- Comorbid malignancy
- Patient choice (eg, if long established on warfarin)
- Extremes of body weight (pharmacokinetics/dosing of direct oral anticoagulants unclear)
- Likelihood of poor compliance without monitoring blood tests

Best JG, et al. Pract Neurol 2019;19:208–224. doi:10.1136/practneurol-2018-002089

Prolonged Cardiac Monitoring after Stroke?

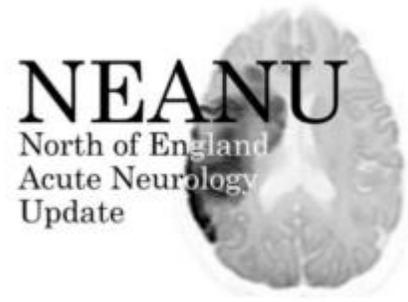
Imaging cortical, haemorrhagic transformation



Cryptogenic Stroke, Negative Vascular Imaging

	Crystal AF	EMBRACE	FIND RANDOMISED
Population	Stroke within 90d, negative vascular imaging, TOE and 24 ECG monitoring	Stroke within 6months, negative vascular imaging, echo, 24hr ECG monitoring	Stroke, negative vascular imaging and admission ECG
Intervention	Implanted cardiac monitor	Non invasive 30d loop recorder	10d HOLTER enrolment, 3 months and 6 months
Control	Routine Care	Add 24hr HOLTER	Add 24hr HOLTER
Results (AF pick up)	12m 30% vs 2% 36m 30% vs 3%	1m 16% vs 3.2%	6m 13.5% vs 4.5%

Warfarin/Anticoagulation following Ischaemic Stroke



- Studies based on the use of Heparin given early (<48hr) increased risk of ICH without reducing risk of stroke recurrence, therefore current practice 14d.
- However risk of stroke recurrence is 5%, therefore many anticoagulated before 14 days
- 1-3-6-12 “rule” for TIA, minor, moderate and large infarct
- Lower ICH risk for DOAC
- OPTIMAS trial- early anticoagulation within 4 days (UK over 3000 patients)

Case conclusion



- Time is Brain
- Early Identification of suitable candidates for iv Alteplase and IAT is vital
- Identification and treatment of AF post Stroke

Patient 2



Background

71yr old

R handed

PMHx psoriasis

mRS 1



Presentation

Noted left arm felt heavy while driving

Left leg gave way leaving car

Falls at home – lives alone

Patient 2



Day 1 –
noon – left
arm weak

Day 3 –
0330 2nd
fall - 999

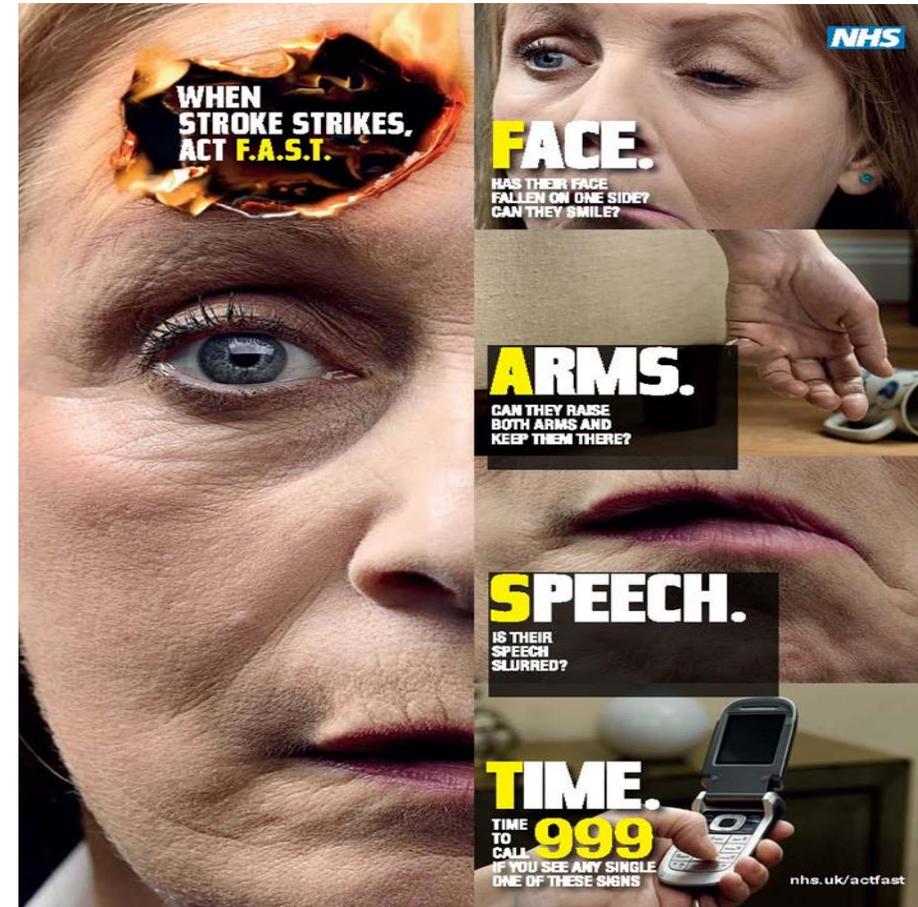
Assessment
in A+E 0637

- NIHSS 5
- Left facial droop - 1
- Left hemiparesis – arm 2, leg 1
- Ataxia left leg – 1
- BP 220/112 (180/110)

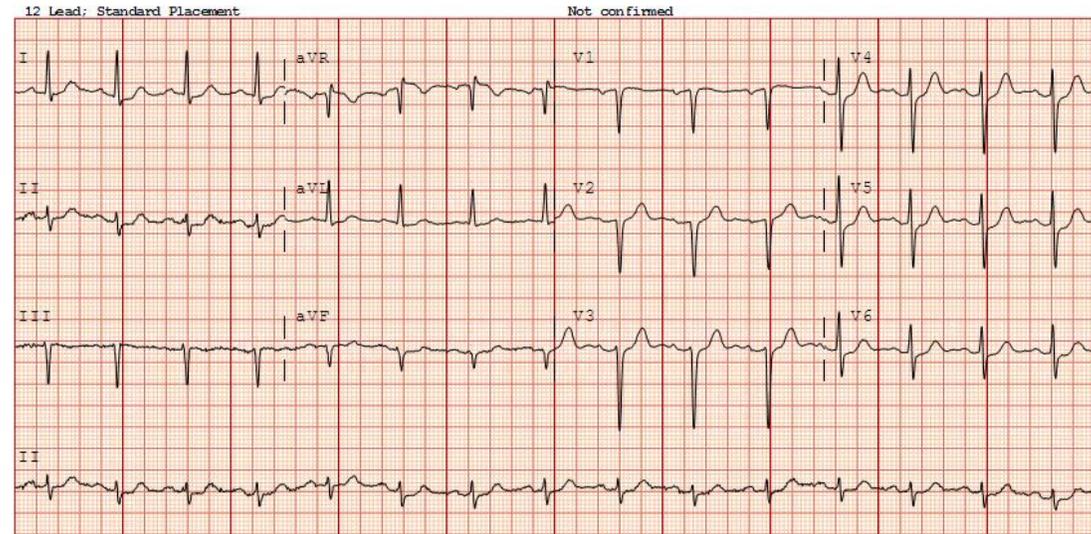
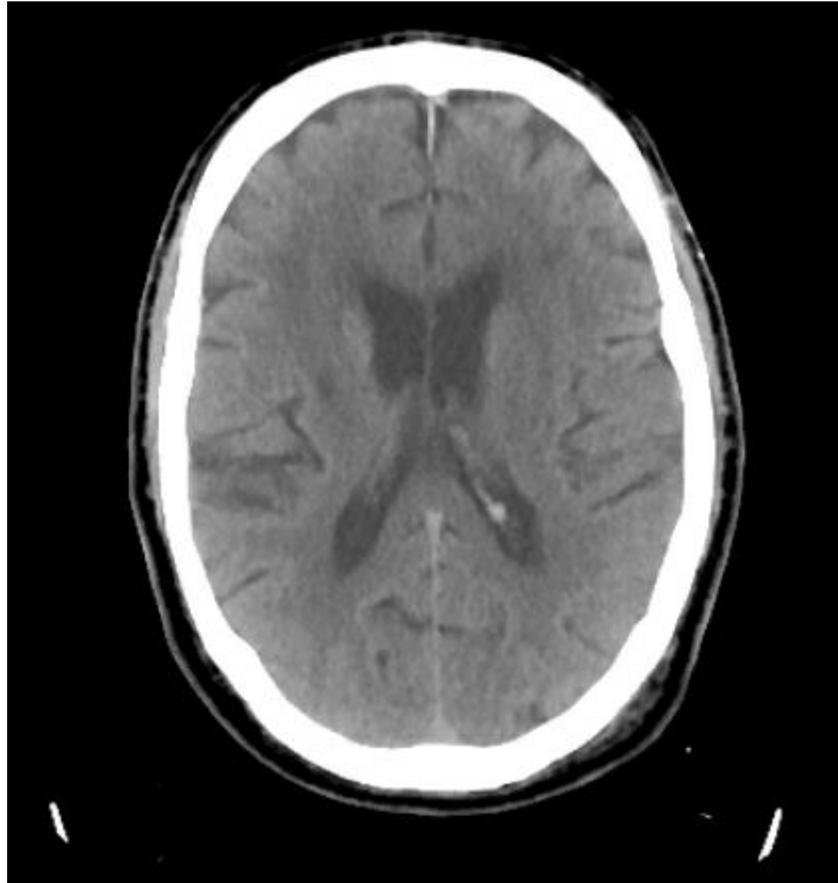
FAST test



- Accepted Pre-hospital Stroke recognition instrument
- High reliability – will identify >85% of stroke presentations *Harbison et al 2003*
- Limitations – false positives / false negatives / does not specify ‘sudden onset’
- Other Scales higher sensitivity but lose on specificity *Zhelev et al Cochrane Library 2019*
- Accepted need for better identification of LVO to ‘triage’ patients to ED for IVT / IAT



Patient 2 - Plain CT imaging and ECG



ECG –SR; inferior & anterior Q waves

CT Imaging – old lacunar infarcts right basal ganglia and left frontal periventricular white matter. Periventricular microvascular ischaemic change

What is the best treatment?

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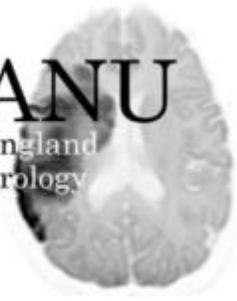
MCQ

Stroke 'Treatments'



Treatment	Eligible (%)	NNT (good outcome)	No. patients benefit / 100 patients
Alteplase (IVT)	15	7	2
Thrombectomy (IAT)	10	2-7	2
Aspirin	85	89	1
SU admission	100	30	30

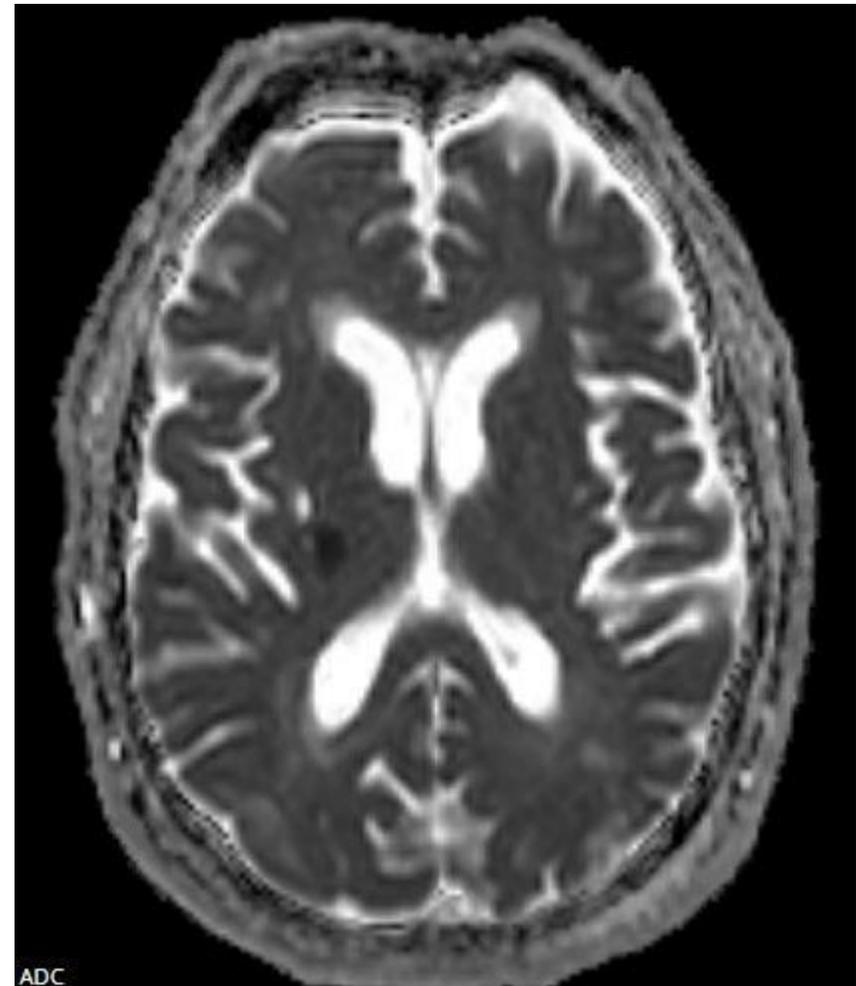
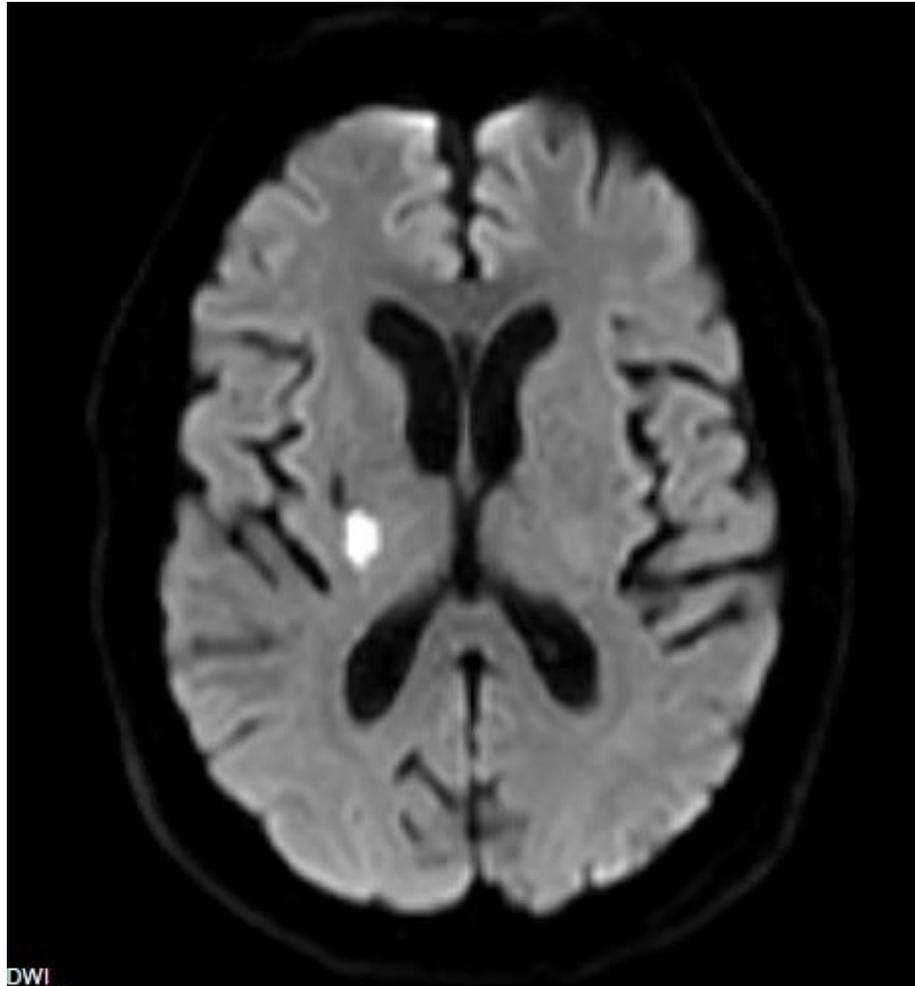
Hypertension management in AIS



- Avoid lowering in first 7 days unless SBP >220 if large artery disease (hypoperfusion)
- Treat to >180/110 if otherwise a candidate for iv thrombolysis
- Post-tPA / recanalization target BP 140-150 (if no CAS)
- Separate guidance in ICH



Right hemisphere LACS



Patient 2



LACI Subtype	Stroke Location (<15mm max diameter, MR)
Pure motor stroke (PMS) (33-50%)	PLIC / basis pontis
Ataxic hemiparesis	PLIC / basis pontis / corona radiata
Clumsy hand – dysarthria syndrome	Basis pontis / genu of IC
Pure sensory stroke (PSS)*	Ventral postero-lateral nucleus of thalamus
Mixed sensori-motor	Thalamus / PLIC

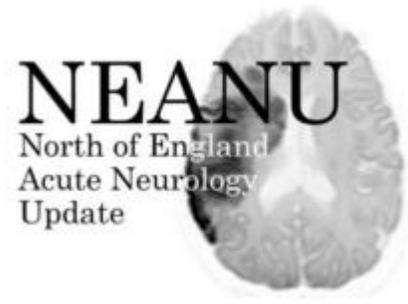
*cf Déjerine-Roussy syndrome – severe contralateral dysaesthesia weeks – months from thalamic stroke



LACS – small vessel disease (svd)

- 25% all stroke presentations
- Primary pathology underlying vascular cognitive impairment
- Older (>70) hypertension, DM implicated
- Commoner in non-Caucasian population

LACS



- Genetic factors important – younger age group
 - rare monogenic variants account for about 1.5% - DNA-LAC1 (<70 years)
 - most are Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) variants
 - second commonest affected gene is HTRA1 *Tan et al, Neurology 2019*
- Further research studies recruiting – DNA-LAC 2, Cambridge 7T study; LACI-2 Trial (ISMN XL, cilastazol)

Case conclusion



- Recognition is key
- Outside of hyperacute treatments there are still benefits from SU care – ALL are eligible
- Stroke as primary presentation of risk factors – 70% could be prevented

Patient 3



Background

63yr old

HTN, Hypercholesterolaemia

IHD with CABG 2018

L THR



**Presented initially 4 weeks earlier at
different hospital**

Headache

Nausea

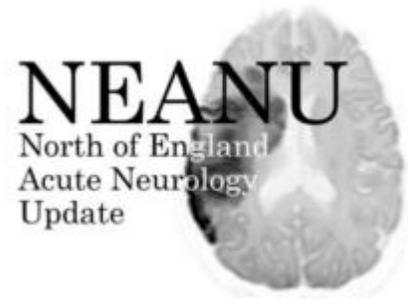
Non specific dizziness

**Double Vision - horizontal separation of
images**

- CT Venogram was performed
 - Chronic dural sinus stenosis/thrombosis in right and left transverse sinuses
- Intracranial CTA was performed, poor quality but patent intracranial vessels.
- MRI Brain did show some abnormal brain lesions? Venous infarcts?

Working Diagnosis was CVST

Current Admission



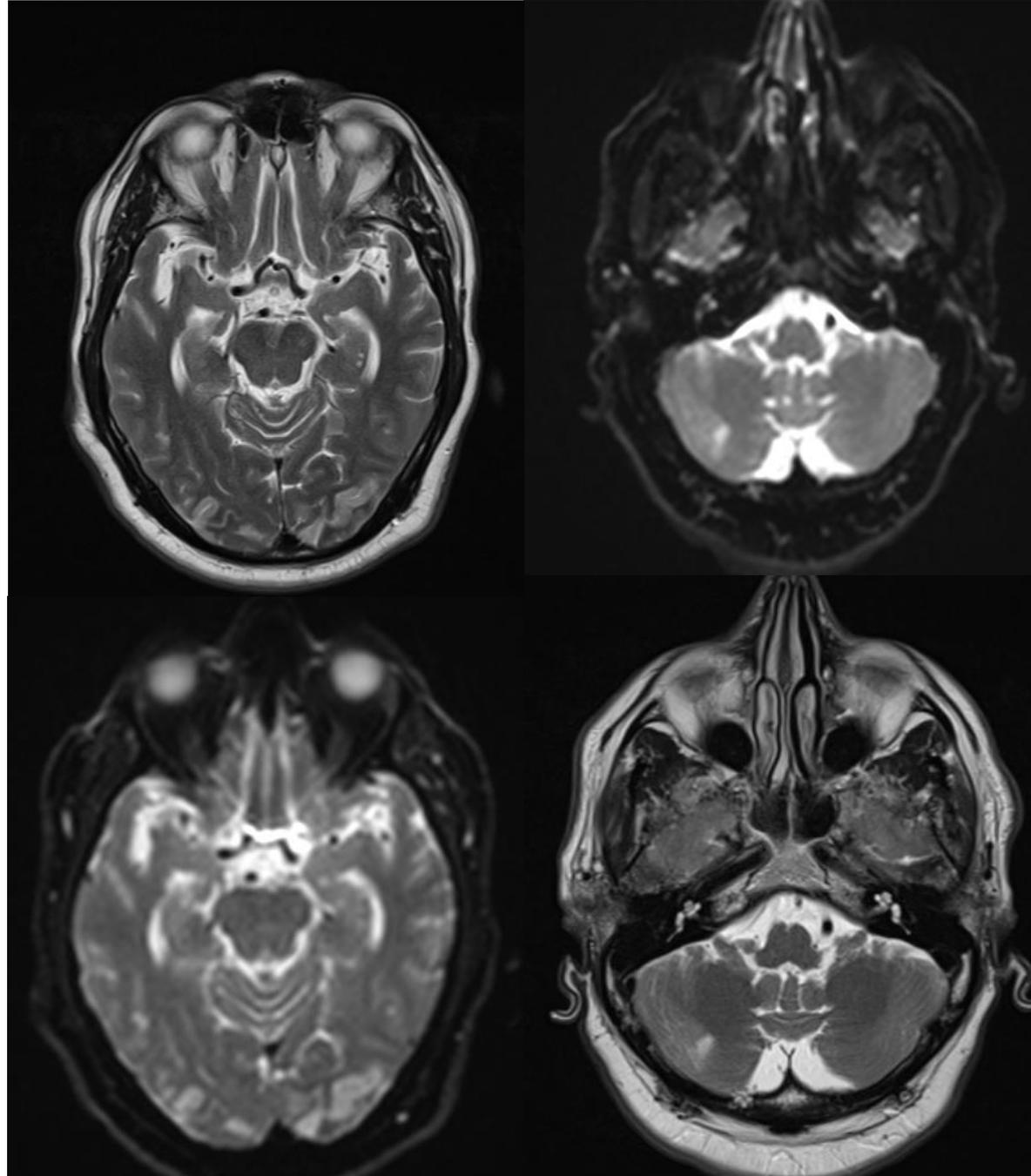
- Worsening symptoms of
 - diplopia
 - **Come and goes- sometimes multiple images**
 - **Not in any direction of gaze, not improved in any head posture**
 - Vertigo - persistent
 - Headache - non specific
 - Intermittent Vomiting
- Clinical Examination:
 - Monocular and Binocular Diplopia- variable in all directions
 - No objective ophthalmoplegia
 - No UMN signs
 - No cerebellar signs

What is the cause of the diplopia?

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MCQ

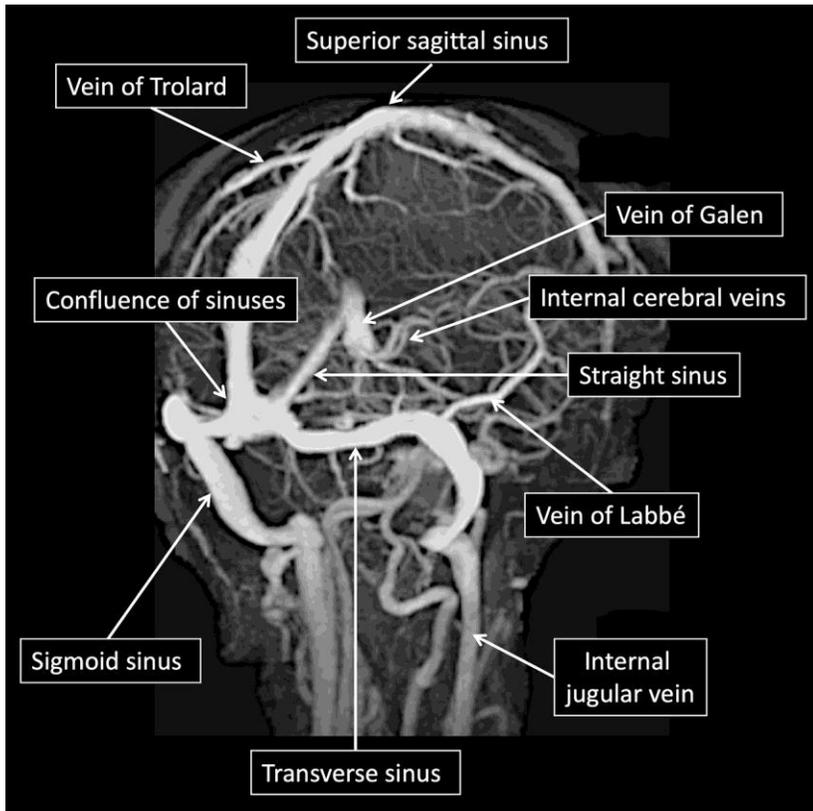


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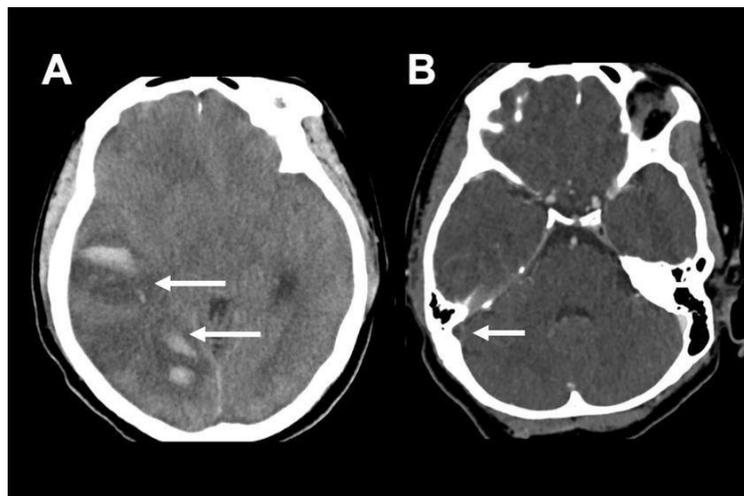
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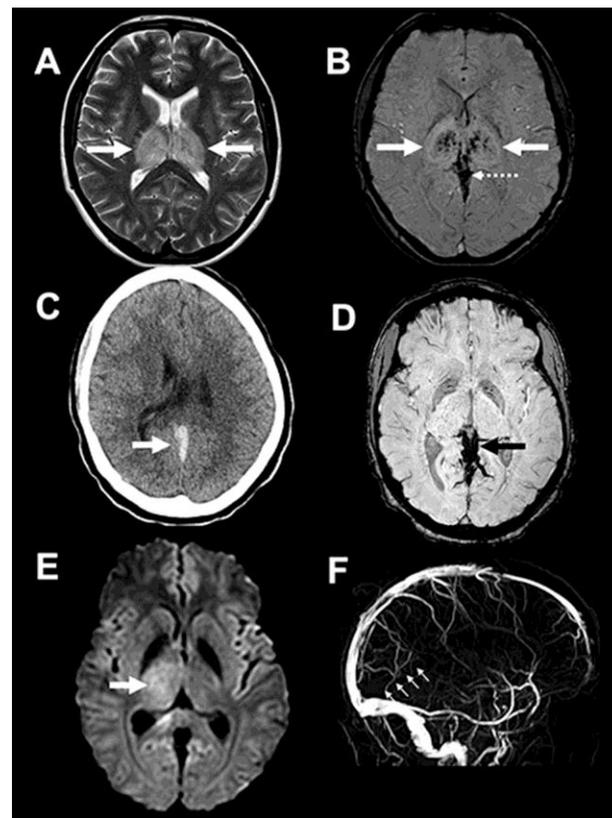
A Worsening CVST? Does the clinical picture correlate?



Occluded Sinus/Vein	Clinical Presentation
Transverse Sinus (44-73%)	Seizures Pyramidal Signs Aphasia If cerebellar veins- ataxia
Superior Sagittal (39-62%)	Aphasia, Hemianopia, Hemisensory loss, hemiparesis, seizures
Sigmoid Sinus (40%-47%)	Mastoid Pain VI- VIII CN Palsy
Deep venous system	Coma Fluctuating Alternating hemiparesis
Cortical Veins	Focal dependent on site
Cavernous Sinus	Ocular pain III, IV, VI and V1



Transverse Sinus



Straight Sinus affecting deep venous drainage.

What is your next investigation?

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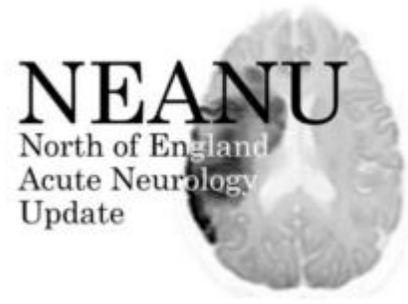


MCQ



Diagnosis: POCS

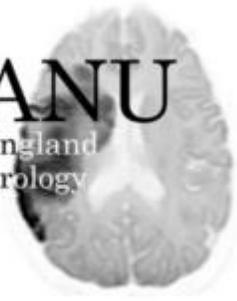
Re-assessment



- Rpt CT Venogram was normal
- CT Angiogram showed a focal proximal severe basilar artery stenosis

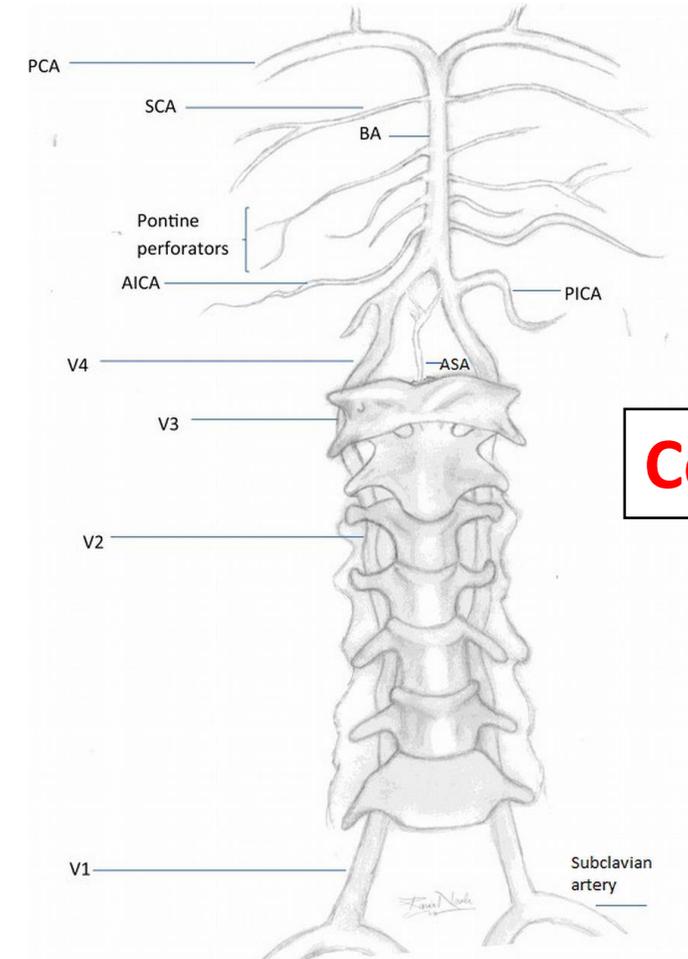
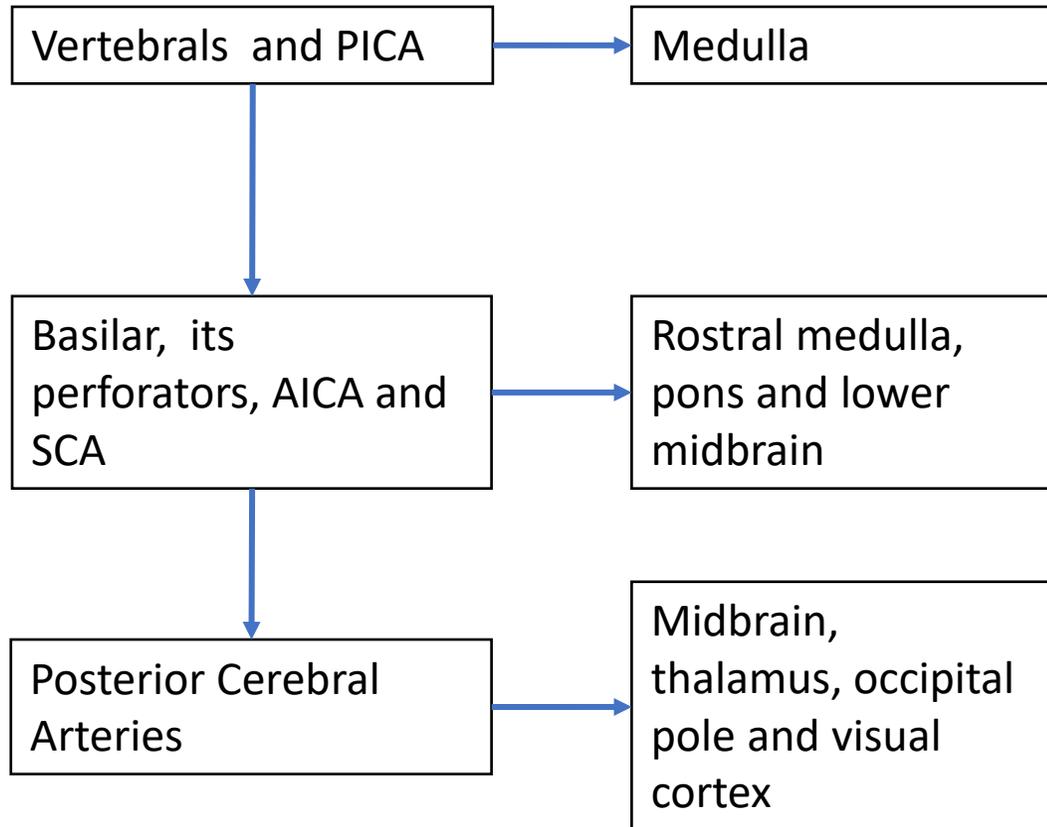
Recurrent POCS due to Basilar Artery Stenosis

Progress



- Treated with dual antiplatelet therapy
- Assessed for basilar stenting by neurovascular MDT, deemed not for endovascular treatment

Posterior Circulation Anatomy



Collaterals++++

Fig 1 Posterior circulation vessels.

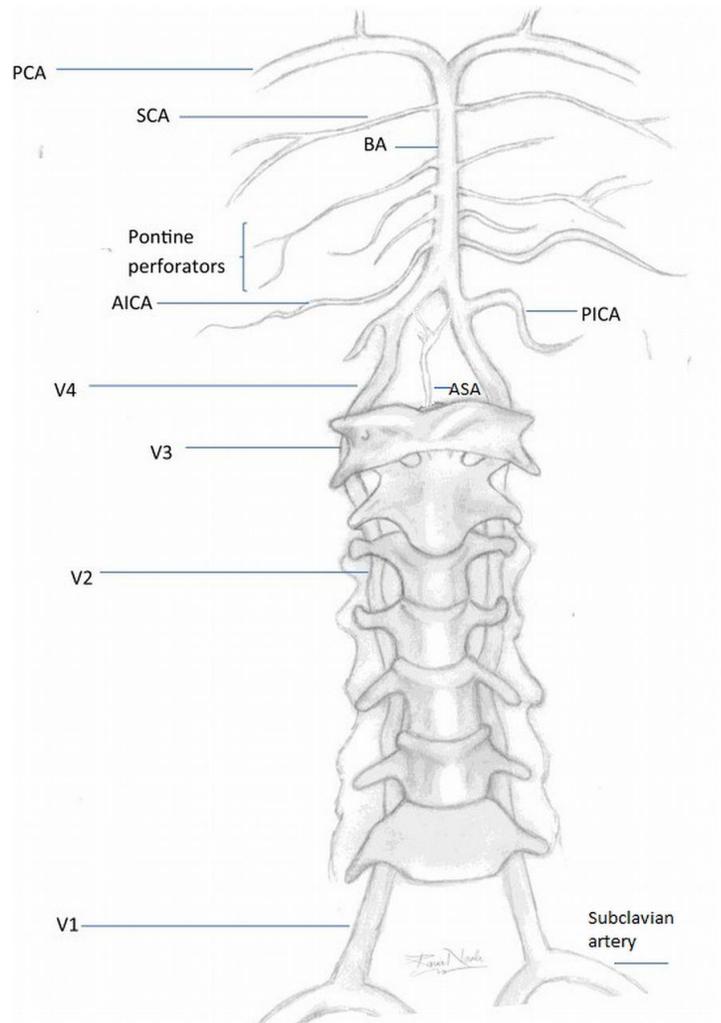


Fig2 PICA

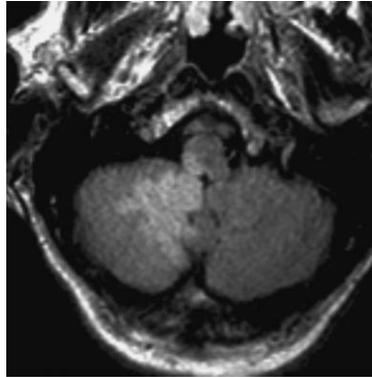


Fig 3 Basilar

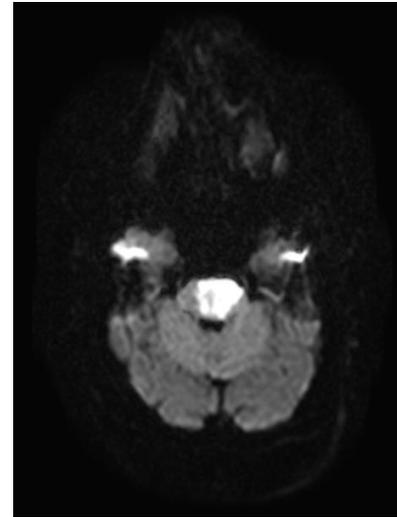


Fig 4 Pontine perforator

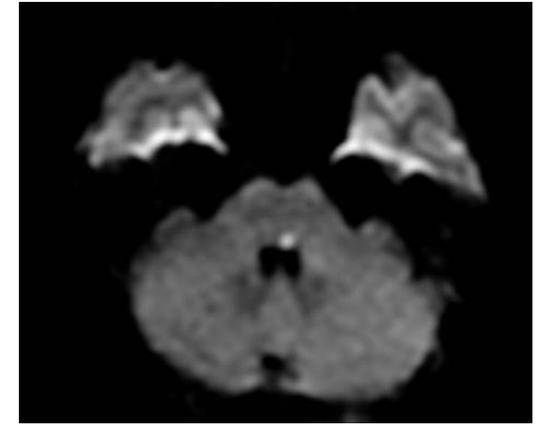


Fig 5 AICA

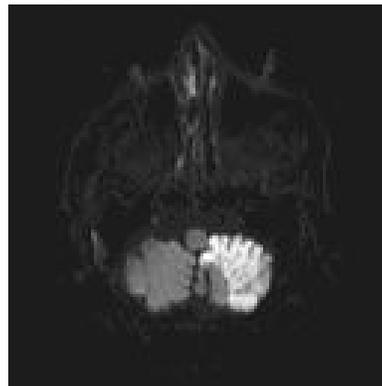


Fig 6 SCA

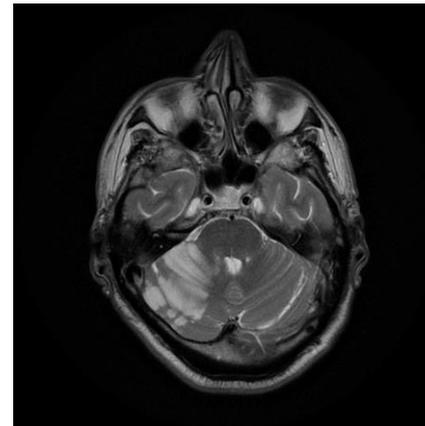


Fig 7 PCA

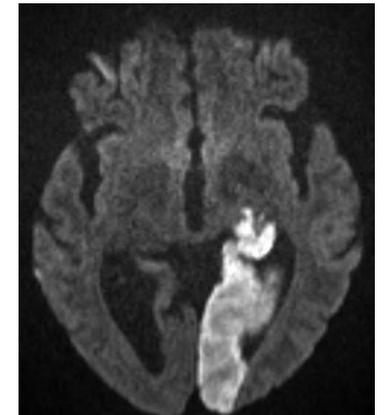
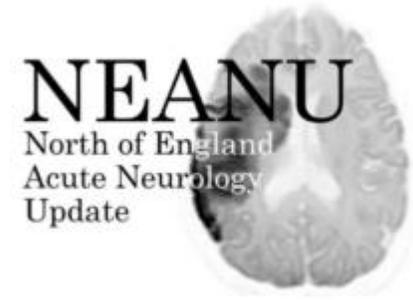


Figure 1. Ursula G Schulz, and Urs Fischer J Neurol Neurosurg Psychiatry 2017;88:45-53. Figure 2 Case courtesy of Dr Mauricio Macagnan, [Radiopaedia.org](https://radiopaedia.org/). From the case [rID: 45051](https://radiopaedia.org/cases/45051). Figure 3>Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 896. Figure4 Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 4187. Case courtesy of Dr Ammar Haouimi, Radiopaedia.org, rID: 74565. Case courtesy of Dr Sandeep Bhuta, Radiopaedia.org, rID: 15054 Fig 7: [Radiology Quiz 84104 | Radiopaedia.org](https://radiopaedia.org/)

Presentation of Posterior Circulation Strokes



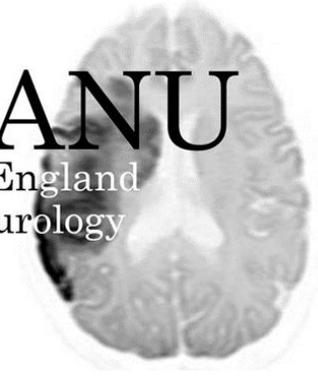
From 407 patients from the NEMC-PCR, the most **frequent presenting symptoms** were:

- dizziness (47%)
- unilateral limb weakness (41%)
- dysarthria (31%)
- headache (28%)
- nausea or vomiting (27%).

From 407 patients in NEMC-PCR, the most **frequent clinical signs** were:

- unilateral limb weakness (38%)
- gait ataxia (31%)
- unilateral limb ataxia (30%)
- dysarthria (28%)
- nystagmus (24%)

2/3 of basilar artery stenosis will have Transient Neurological Attacks prior to Stroke



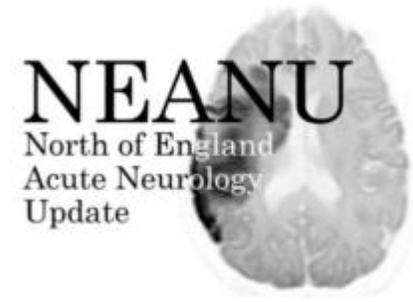
HINTS in differentiating POCS from peripheral vestibulopathy

- Acute Vestibular Syndrome (AVS)- 5% POCS
- Unidirectional nystagmus following Alexanders Law not specific ENOUGH to exclude central lesion
- HINTS - is 100% sensitive and 96% specific in the initial study

Exam	Peripheral	Central
Head Impulse Test HI	Loss of eye fixation with head impulse "positive"	Intact VOR "negative"
Nystagmus N	None or Horizontal Unidirectional	Vertical Rotatory or horizontal bidirectional
Test of Skew TS	No skew	Skew positive

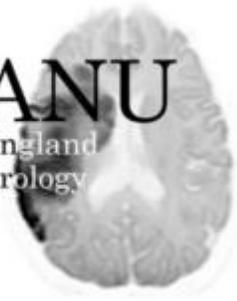
A "central" finding = MRI imaging.

Basilar Artery Occlusion- Long Term Management



- Prodromal Minor Stroke is associated with a poorer long term outcome in POCS (unlike in anterior)
- Stenting in SAMMPRIS trial was associated with worse outcome
- WEAVE study suggested possible role for stenting, but not statistically robust
- Can be considered in some cases, but overall management is medical.

Key Points



- POCS has a variable presentation, but some more frequent features
- IAT is an option potentially up to 24hr for severe cases due to robust collaterals
- CVST is over diagnosed radiologically- ask if the clinical syndrome fits

Patient 4



Background

32yr old

R handed

PMHx SAH 2016 (clipped)

LICA occluded, R dysplastic

mRS 1

Atorvastatin 10mg



Presentation

Numb L face and arm

3 attacks in 48 hours

30 minutes each

Well between attacks and at time of
assessment

Patient 4

O/E (NIHSS) 0

ABCD satisfactory, BP 124/80

ECG

NSR

CT

excludes haemorrhage

CTA

abnormal right M2 ?dysplastic

?clot

Clinical diagnosis?

Progress

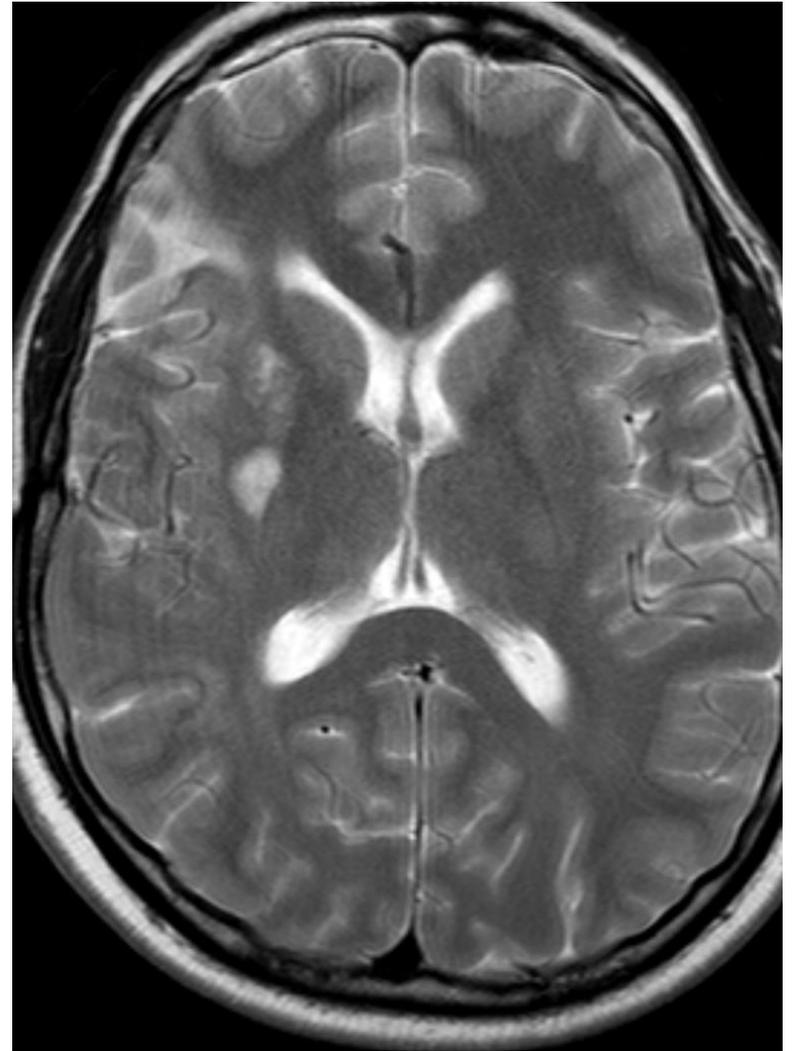
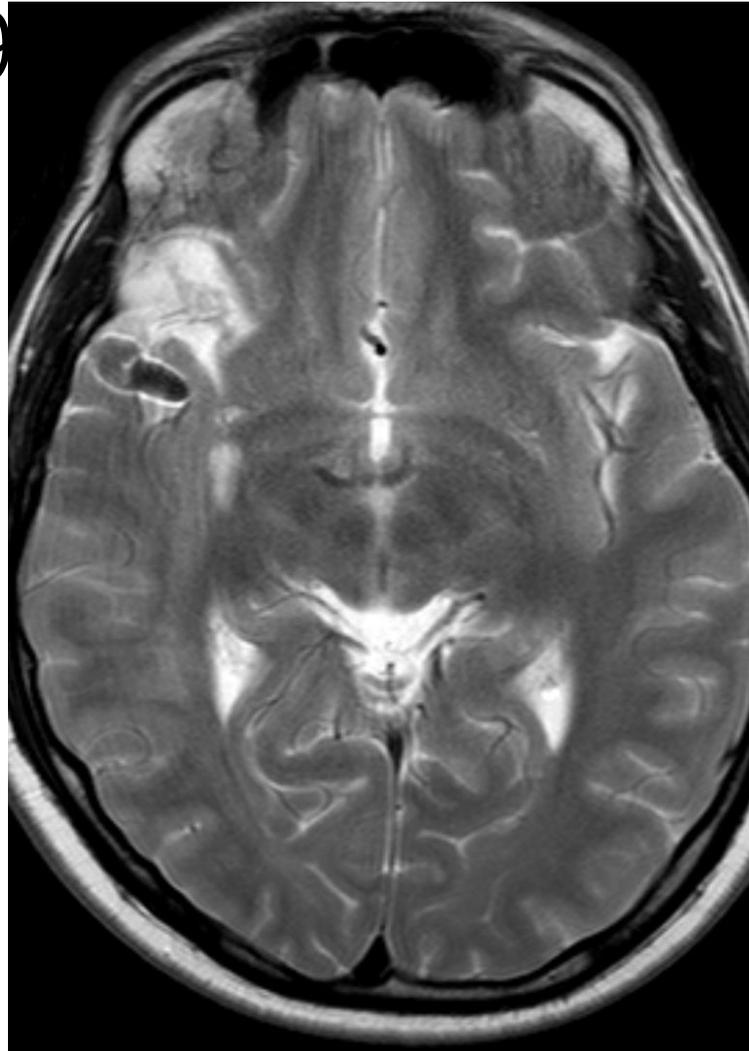
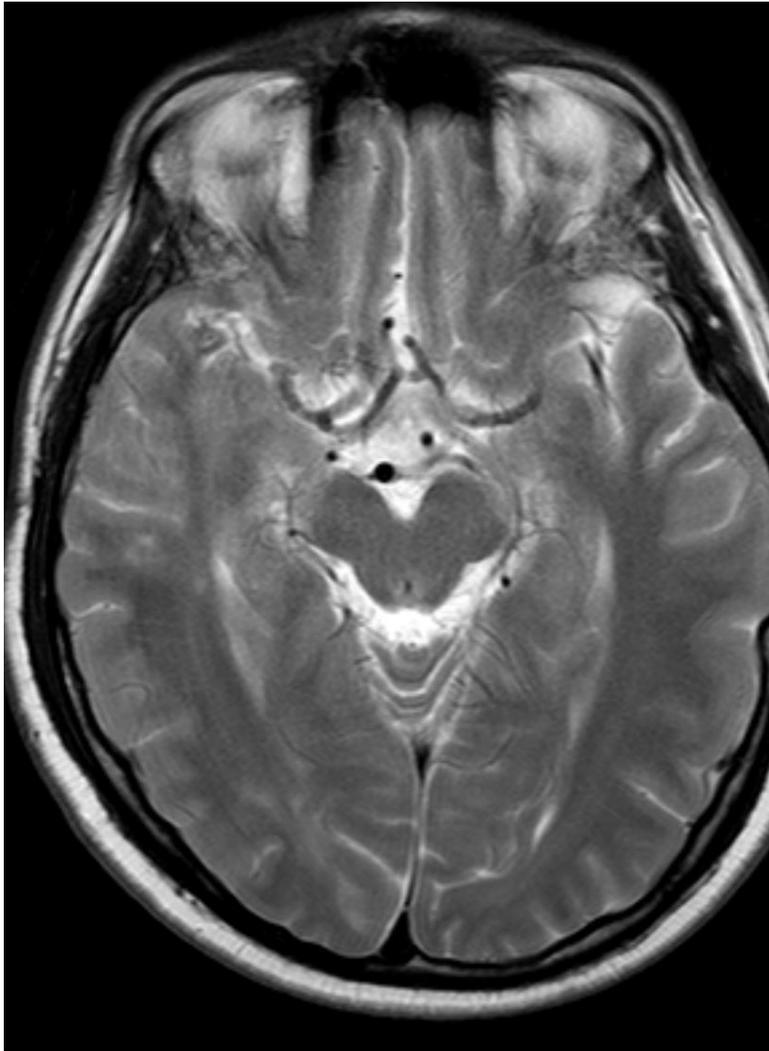


Crescendo TIA

Home with aspirin 75mg

Reattended 27.06.19

Sudden onset L sided weakness / numbness



69

Progress



Referral to FMD service

dysplastic vessels (no stenosis / beading noted)

previous aneurysm

home on high dose aspirin / clopidogrel at 2 weeks

Progress



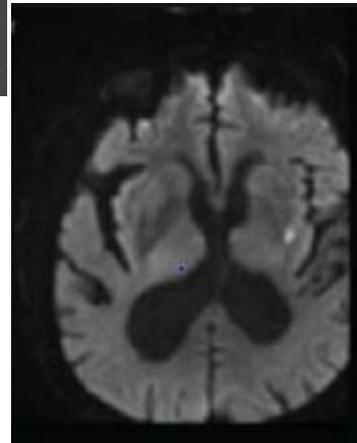
09.10.20 woke with left sided facial numbness / tingling
 vertigo / vomiting
 double vision
 no lateralising ataxia
 no crossed sensory change

Clinically - POCS

MR



- Left lateral medulla infarct
 - Incomplete wrt syndrome
- Unexpected left lentiform nucleus infarct



What next?

What next?

Young age

No new vessel changes

No inflammatory marker increase

Multiple territory events now despite antiplatelets

Prolonged ECG monitoring SR throughout (5+7 days)

Previous bubble echo (TTE) normal

Progress



Bubble echo with femoral injection

Confirms large PFO with significant right to left shunt

TP screen (and vasculitis screen) previously normal

Repeat – normal; LP negative OCB (but 6wbc...)

PFO closure?

Clinically commissioned from July 2019

Criteria to be met:

Stroke / TIA with confirmatory imaging

PFO with clinically significant shunt / atrio-septal aneurysm

Absence of 'clinically significant' AF (would AC)

Full investigation of stroke risks

MDT agree most likely stroke mechanism paradoxical embolism

<60 years old

Data recorded to UK Central Cardiac Audit database

Latest

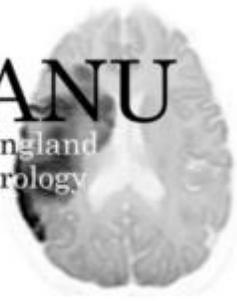
What next?

Referred for PFO closure (concern over anticoagulation / DAP with previous SAH) – completed March 2021

Further vessel imaging; genetics screening (Ehlers Danlos, Loeys-Dietz)

International discussion

Key Points



- Recurrent stroke despite treatment – possible but review thinking
- With multi-territory infarcts look hard at the heart (esp young age)
- Share uncertainty

Thank you