When, why and how to treat carotid disease

Timing to intervene in symptomatic carotid lesions is in my practice based on ?

Julian Scott , Stuart Currie

Leeds Teaching Hospital NHS Trust

Leeds

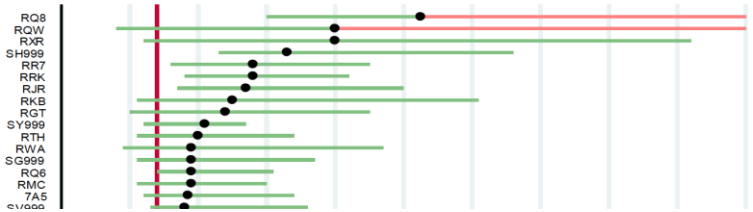
West Yorkshire

UK

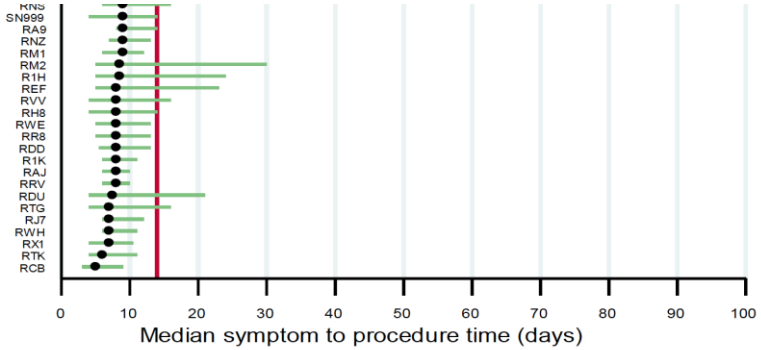
Financial Disclosures

- None

Figure 2.2: Median time (and interquartile range) from symptom to procedure by NHS trust for procedures done between January and December 2015



- There remain considerable variations between NHS vascular units with regard to the provision of carotid endarterectomy within 14 days of symptoms. NHS trusts should optimise referral pathways within their networks and implement improvements to drive down the waiting times
- All staff involved in organising and delivering care to patients who require carotid surgery need to examine their data and assess their performance against standards within NICE Guideline CG68

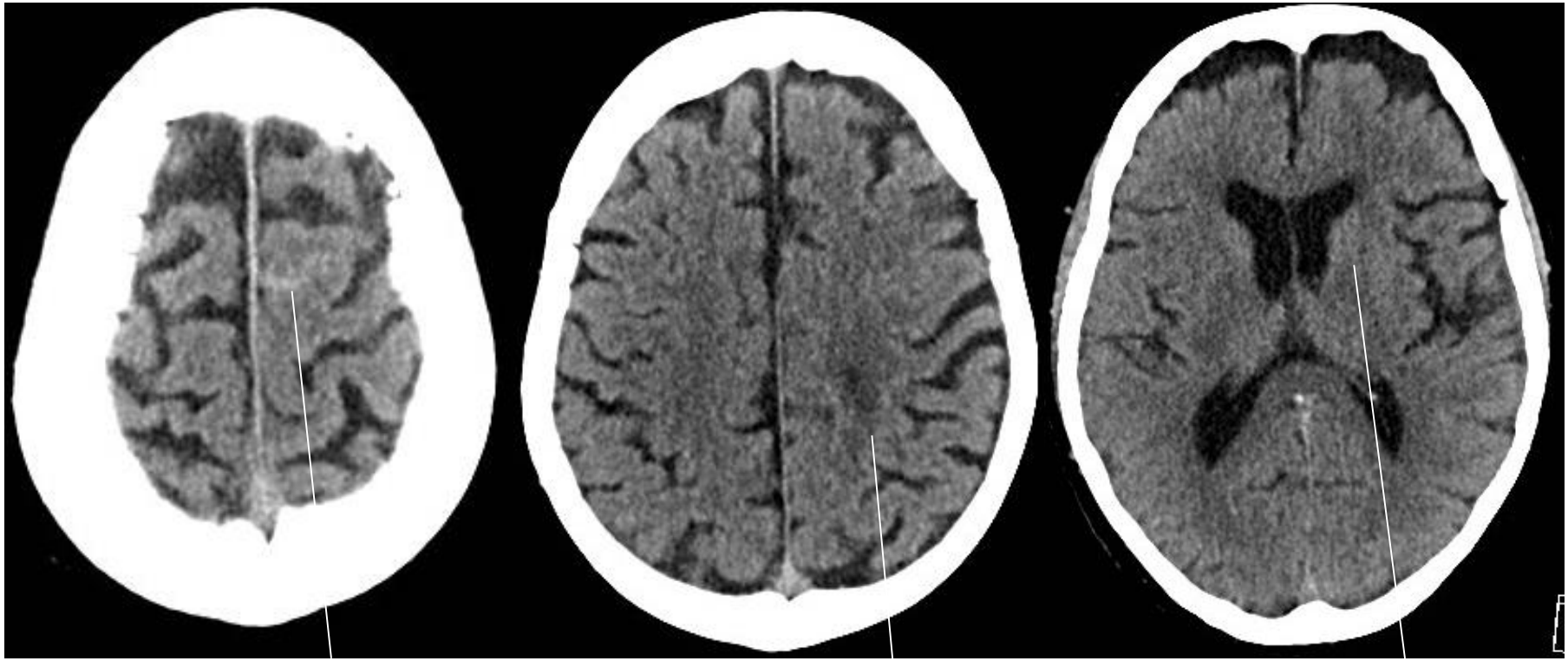


Case A

History

- 01/12/2017
- 87 year woman
- 6 days of intermittent weakness of right arm
- One episode of weakness of right leg
- Atrial fibrillation, Sick Sinus Syndrome, Pacemaker, Hypertension, Non Smoker,
- Medications; Apixaban, Atorvastatin, Bisoprolol, GTN, ARB (Losartan), Diuretic (Indapamide),
- O/E slight reduction in power of right arm
- Admitted under Stroke Physicians

Unenhanced CT
01/12/2017

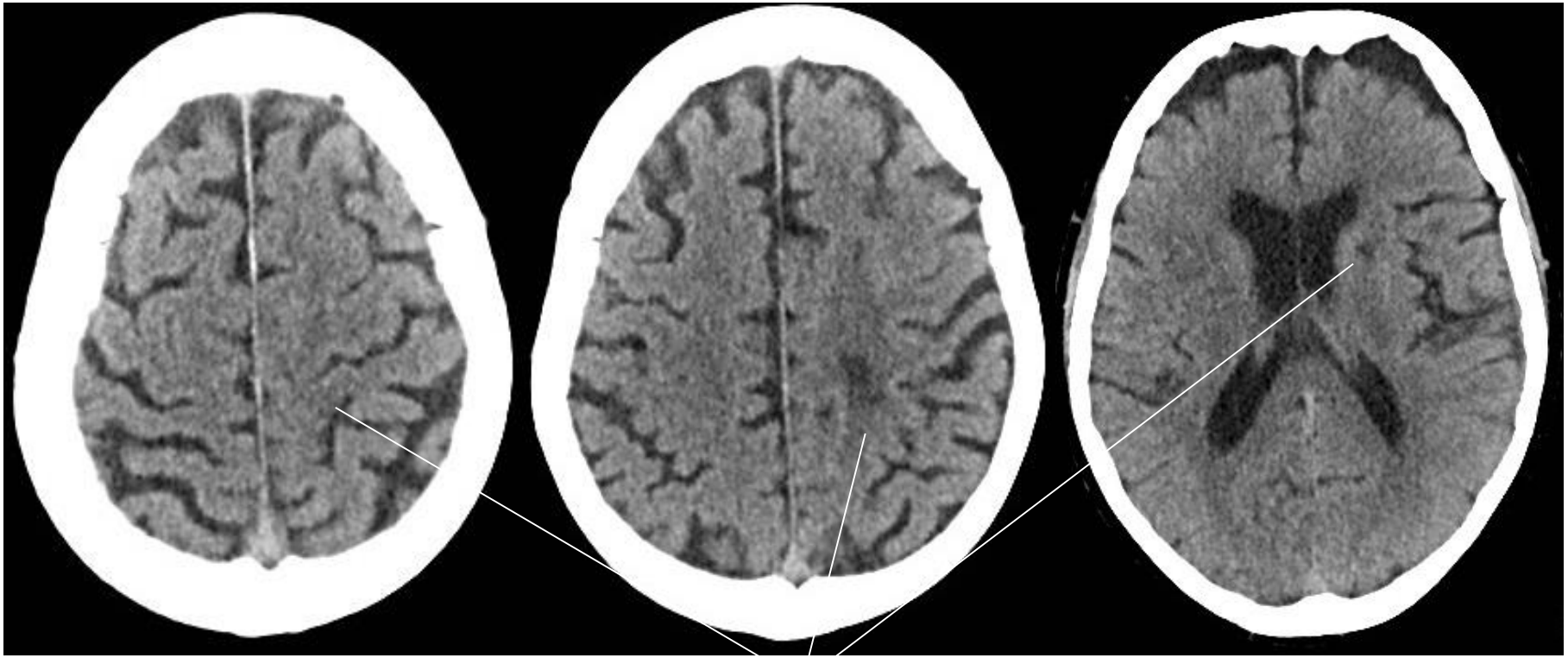


Acute subarachnoid haemorrhage in left paramedian frontal sulcus, associated with high grade ICA stenosis

Acute ischaemia anterior left parietal lobe

Normal left caudate nucleus

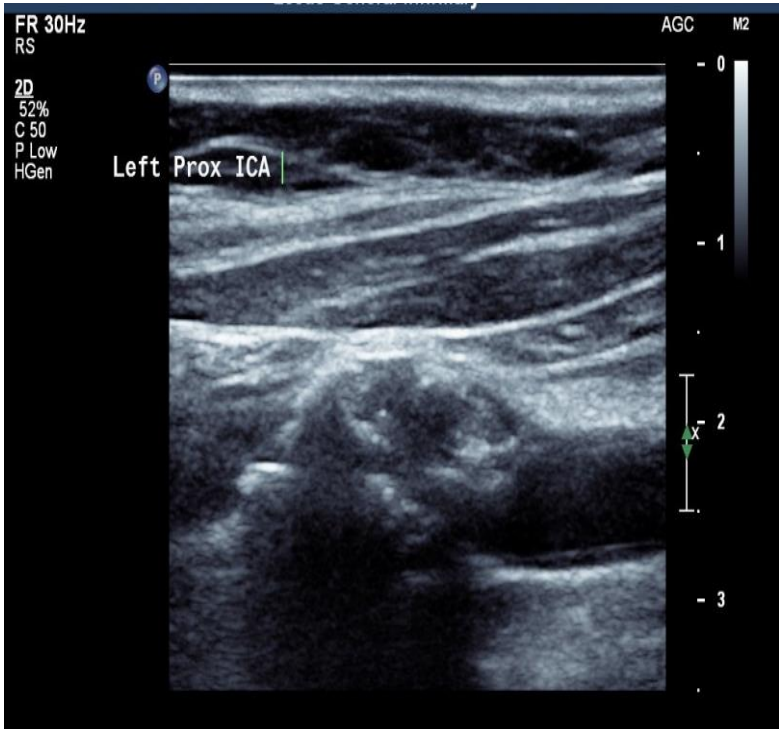
Unenhanced CT
07/12/2017



Progression of left parietal ischaemia and new left caudate nucleus ischaemia

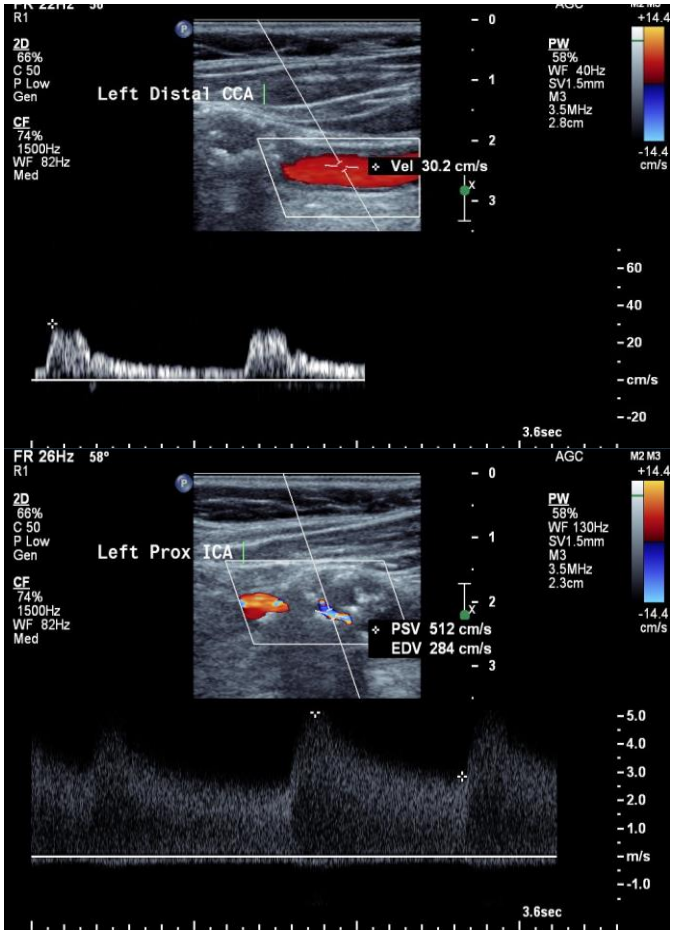
Carotid Duplex 08/12/2017

- Right Carotid:
  CCA PSV 57 cm/s
  ICA PSV 69 cm/s
  ICA EDV 25 cm/s
  ECA PSV 62 cm/s
  IC/CC ratio: 1.2:1
  % ICA stenosis: 0-49 %
  Vertebral direction: antegrade
  Plaque location: Bulb, Prox ICA
  Plaque classification: 5



Carotid Duplex 08/12/2017

- Left Carotid:
  CCA PSV 30 cm/s
  ICA PSV 512 cm/s
  ICA EDV 284 cm/s
  ECA PSV 98 cm/s
  IC/CC ratio: 17:1
  % ICA stenosis: 70-99 %
  Vertebral direction: antegrade
  Plaque location: Bulb, Prox ICA
  Plaque classification: 5



How do you manage ?

Eur J Vasc Endovasc Surg (2018) 55, 3–81

Editor's Choice — Management of Atherosclerotic Carotid and Vertebral Artery Disease: 2017 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)

A.R. Naylor [a], J.-B. Ricco [a], G.J. de Borst [a], S. Debus [a], J. de Haro [a], A. Halliday [a], G. Hamilton [a], J. Kakisis [a], S. Kakkos [a], S. Lepidi [a], H.S. Markus [a], D.J. McCabe [a], J. Roy [a], H. Sillesen [a], J.C. van den Berg [a], F. Vermassen [a], ESVS Guidelines Committee [b], P. Kolh, N. Chakfe, R.J. Hinchliffe, I. Koncar, J.S. Lindholt, M. Vega de Ceniga, F. Verzini, ESVS Guideline Reviewers [c], J. Archie, S. Bellmunt, A. Chaudhuri, M. Koelemay, A.-K. Lindahl, F. Padberg, M. Venermo

Keywords: Carotid, Vertebral, Stroke, Transient ischaemic attack, Endarterectomy, Stenting, Medical therapy, Screening, Dementia, Asymptomatic, Symptomatic, Thrombolysis, Imaging, Bypass, Surgical techniques, Complications, Patch infection, Restenosis

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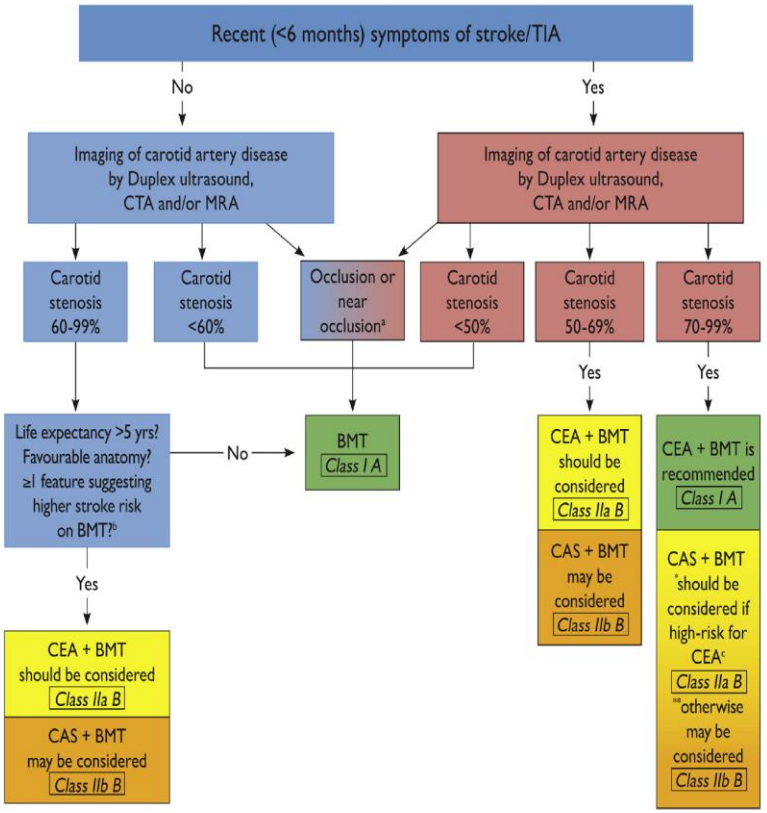
* Corresponding author. *E-mail address*: ross.naylor@uhl-tr.nhs.uk

http://dx.doi.org/10.1016/j.ejvs.2017.06.021



Question

- How would you manage the patient?

- Blink
- Malcolm Gladwell
- The Power of Thinking without thinking

Follow up

- Patient declined intervention

- Cerebral amyloid angiopathy
- Mimics TIA
- High risk of symptomatic intra cerebral haemorraghe

Int J Stroke. 2013 Feb;8(2):105-8

Best Medical Therapy (BMT)

- Compliance
- Statins (LDL)
- Antihypertensive medication (Reduce Systolic BP 10mmHg and Diastolic 5mmHg)
- Cognitive impairment
- Depression
- Inadequate follow up
- Side effects
- Lack of belief/insight
- Complexity

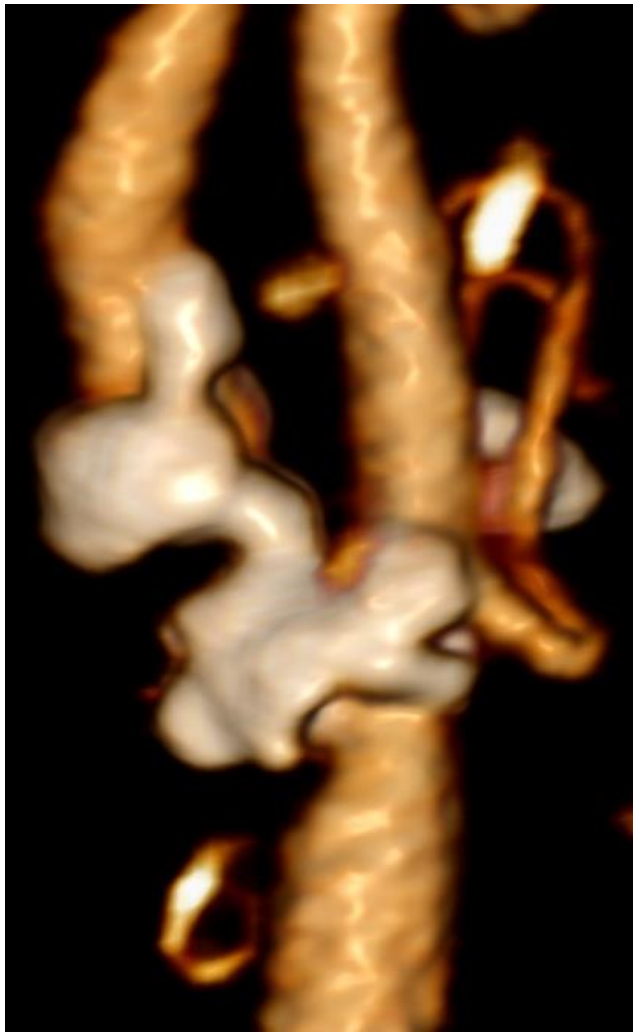
01/03/2018

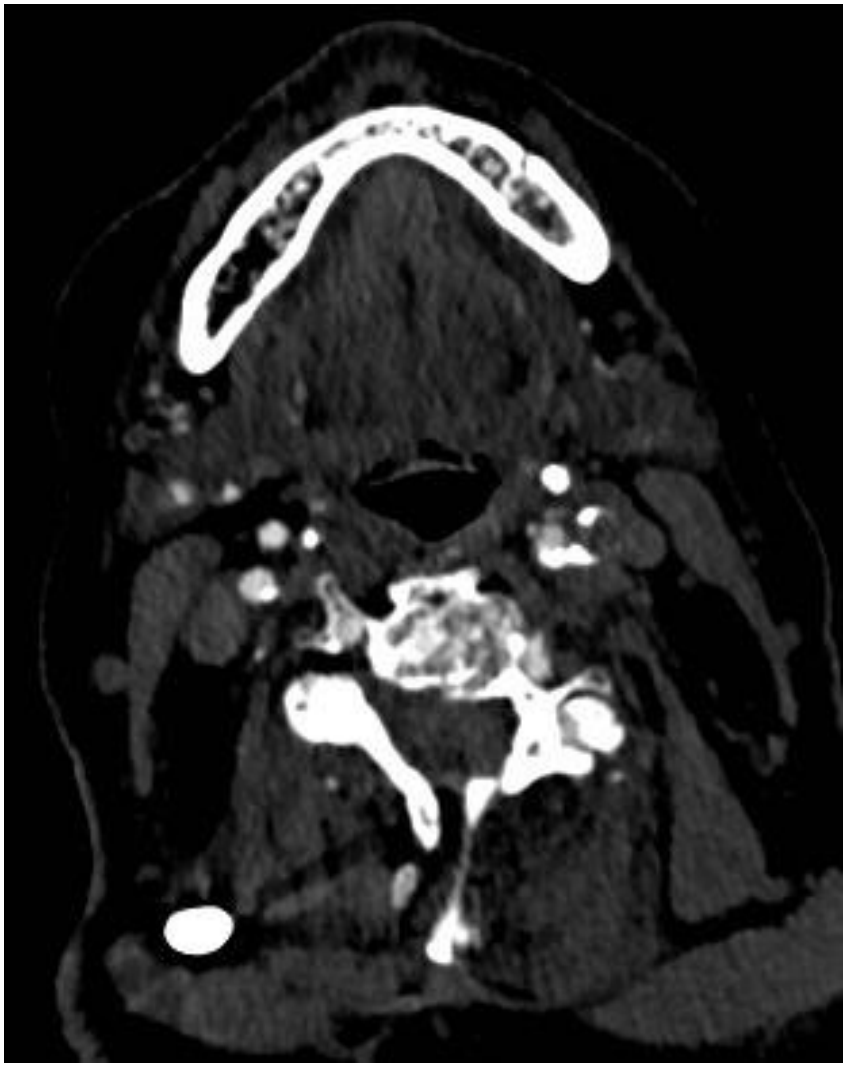
- Fell after getting up quickly and she couldn't get up; there were no abnormal movements and no loss of consciousness.
- A CT head scan was done in A&E to rule out any bleed. The episode was considered a pre-syncope.

5th March 2018

- A neighbour rang her, and noted she was trying to get the words out but couldn't say them, full recovery in less than one hour.
- A repeat CT head scan showed no bleed and no further changes compared to 1st March 2018
-

19th March 2018





How do you manage ?

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Editor's Choice — Management of Atherosclerotic Carotid and Vertebral Artery Disease: 2017 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)

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Keywords: Carotid, Vertebral, Stroke, Transient ischaemic attack, Endarterectomy, Stenting, Medical therapy, Screening, Dementia, Asymptomatic, Symptomatic, Thrombolysis, Imaging, Bypass, Surgical techniques, Complications, Patch infection, Restenosis

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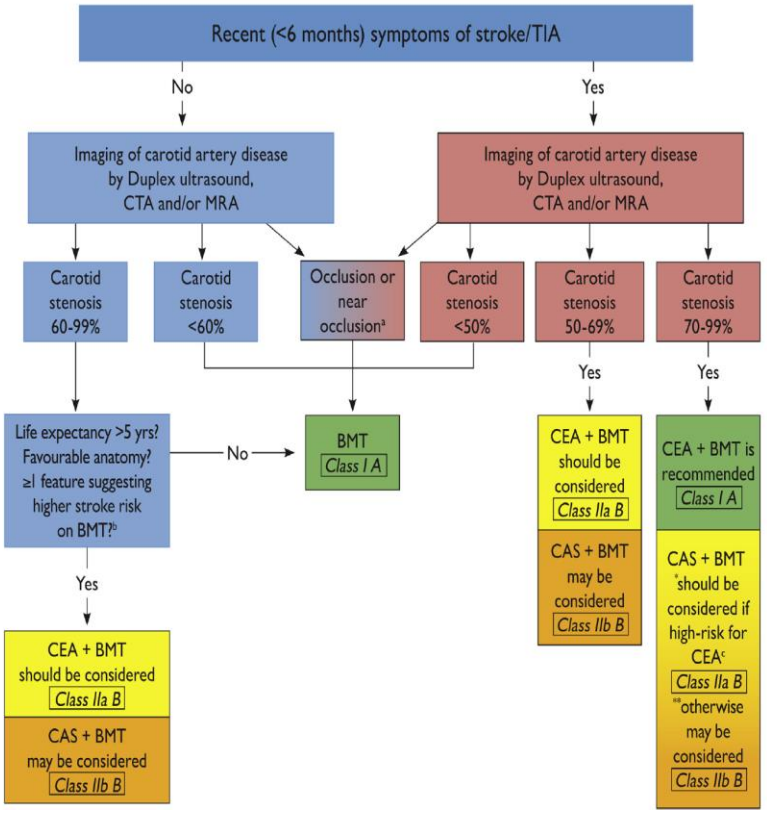


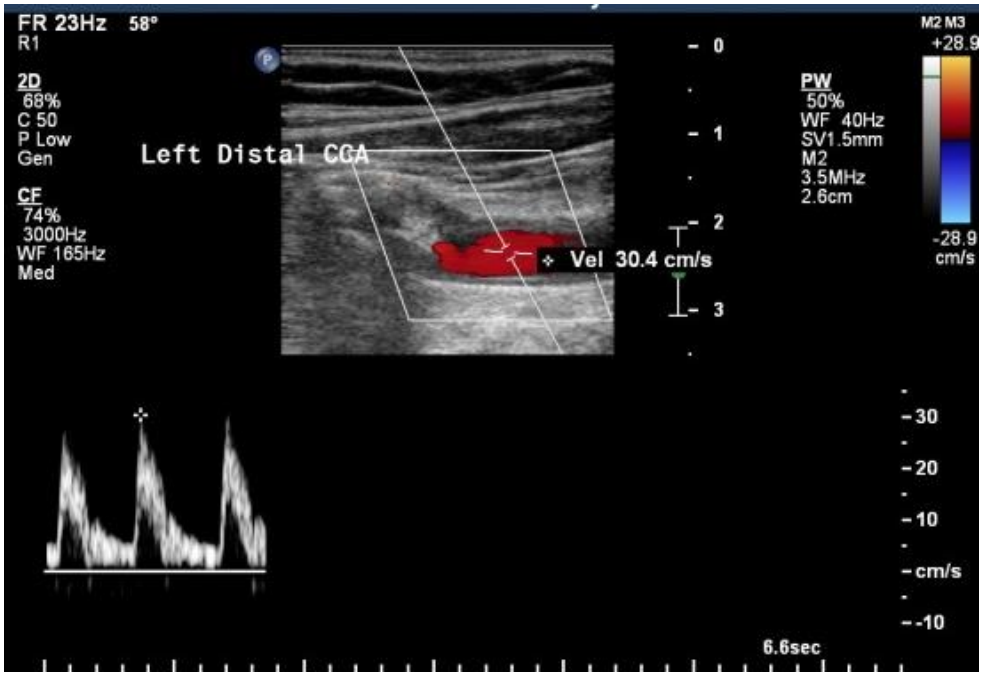
21/03/2018

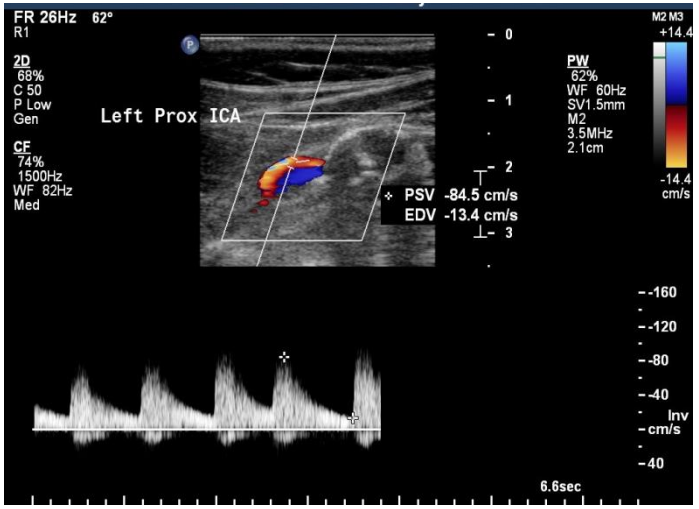
- Under the stroke physicians
- Apixaban Stopped for 48 hours
- Surgery 22/3/2018
- Clinical Decision Unit
- Sudden onset right sided weakness and aphasia while in CDU.
- What happens now?

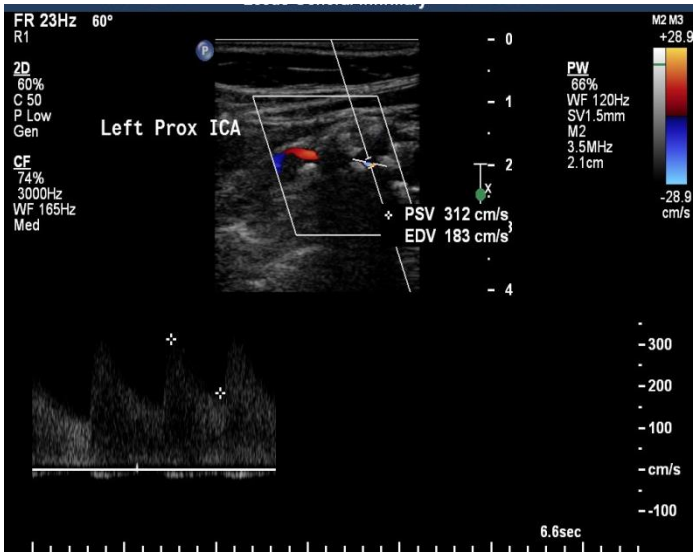
Follow up

- Seen by the Brain Attack Team (BAT)
- Decision made to thrombolysis.
- Progress: aphasia and weakness fully resolved post-thrombolysis.

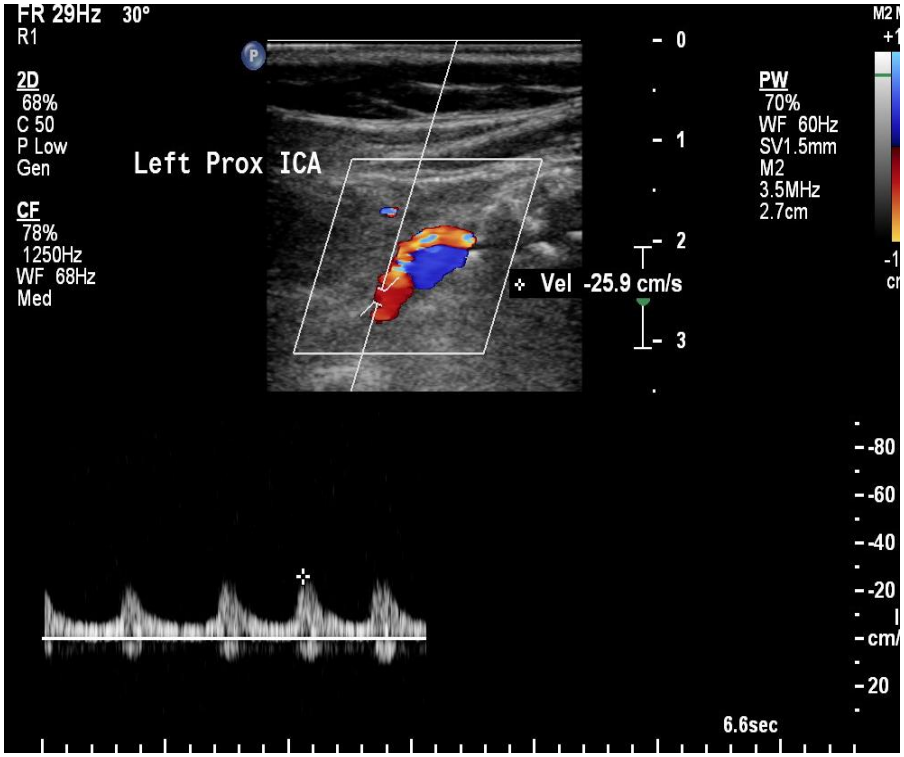
Duplex 22/03/2018

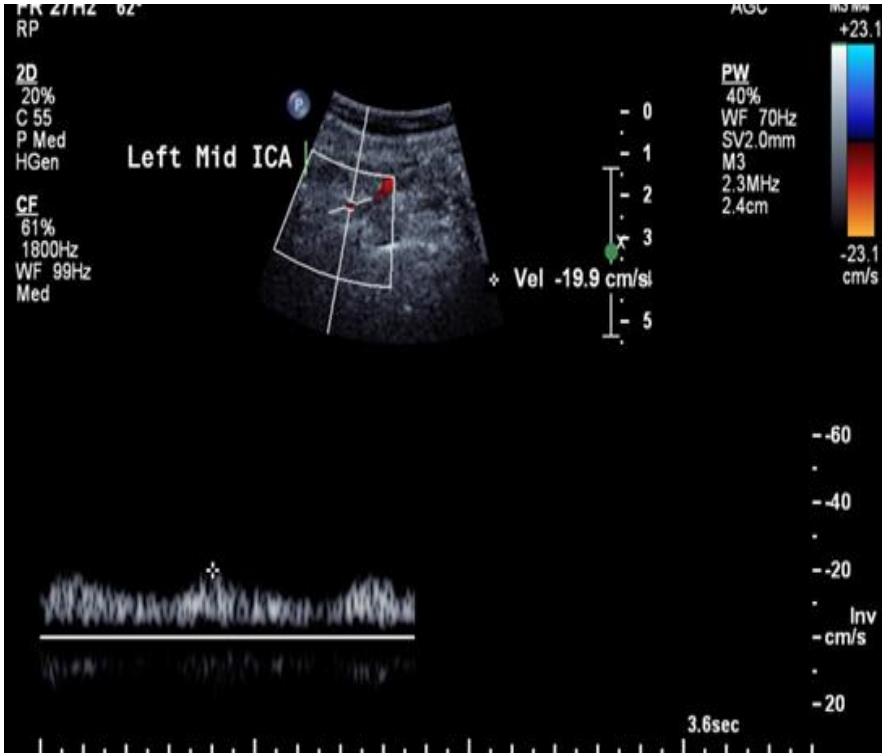






Duplex 22/03/2018
Post thrombolysis





Question

- How would you manage the patient

2.3.6. Timing of carotid interventions after intravenous thrombolysis. An important concern when performing CAS/CEA after intravenous thrombolysis (IVT) is an increased risk of ICH following reperfusion of ischaemic cerebral tissue. A

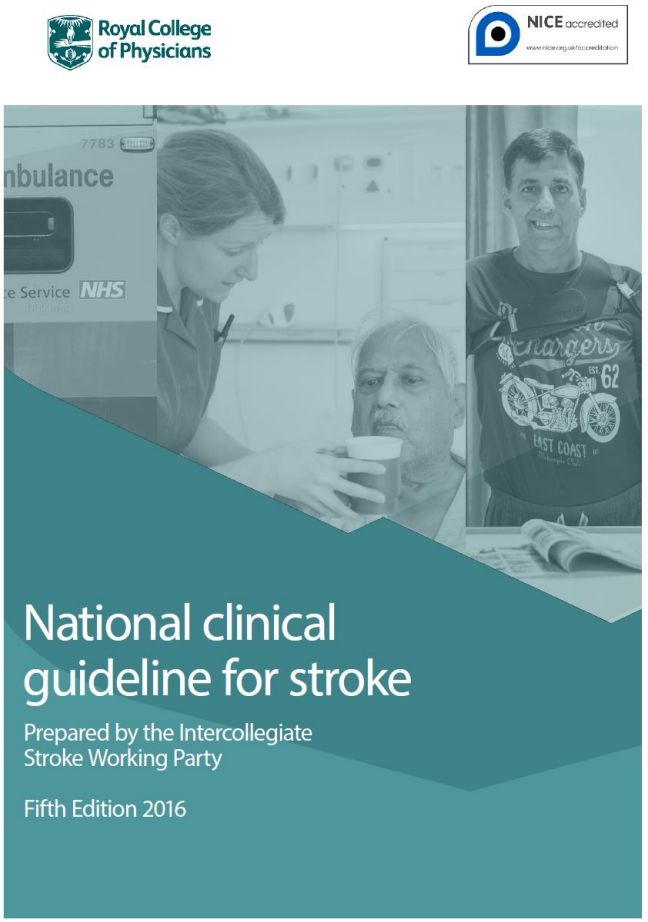
Recommendation 44	Class	Level	References
Early carotid endarterectomy (within 14 days) should be considered after intravenous thrombolysis in symptomatic patients if they make a rapid neurological recovery (Rankin 0—2), the area of infarction is less than one-third of the ipsilateral middle cerebral artery territory, a previously occluded middle cerebral artery mainstem has recanalised, there is a 50—99% carotid stenosis and no evidence of parenchymal haemorrhage or significant brain oedema	IIa	C	244—246
Recommendation 45			
It is recommended that intravenous heparin and antiplatelet therapy be withheld for 24 hours after completion of intravenous thrombolysis, but antiplatelet therapy should then be commenced before any carotid intervention is undertaken	I	C	247
Recommendation 46			
It is recommended that patients undergoing early carotid interventions after thrombolysis should have post-interventional hypertension actively treated to reduce the risks of parenchymal haemorrhage	I	C	244

Low procedural risk

- Rapid recovery after IVT (Rankin 0-2)
- Area of infarction< 1/3 MCA territory
- Recannalisation of the previously occluded MCA mainstem
- ICA stenosis 50-99%
- No evidence of parenchymal haemorrhage or significant brain oedema

Timing

- Unknown
- Antiplatelet and heparin should be stopped for 24 hrs after IVT
- CEA < 14 days 3.4% 30 day death and stroke versus < 7 days 5.1%
- Careful BP monitoring post op



https://www.strokeaudit.org/SupportFiles/Documents/Guidelines/2016-National-Clinical-Guideline-for-Stroke-5t-(1).aspx

Case C

History

- Patient attends his General Practitioner
- Last 6-8 months left leg gives way on walking and if there is nobody to support him he will fall. Investigated Neurosurgery/Neurology
- Last 2 months almost at the same time he describes left arm shaking which then drifts from left to right, this last approx 4 mins.

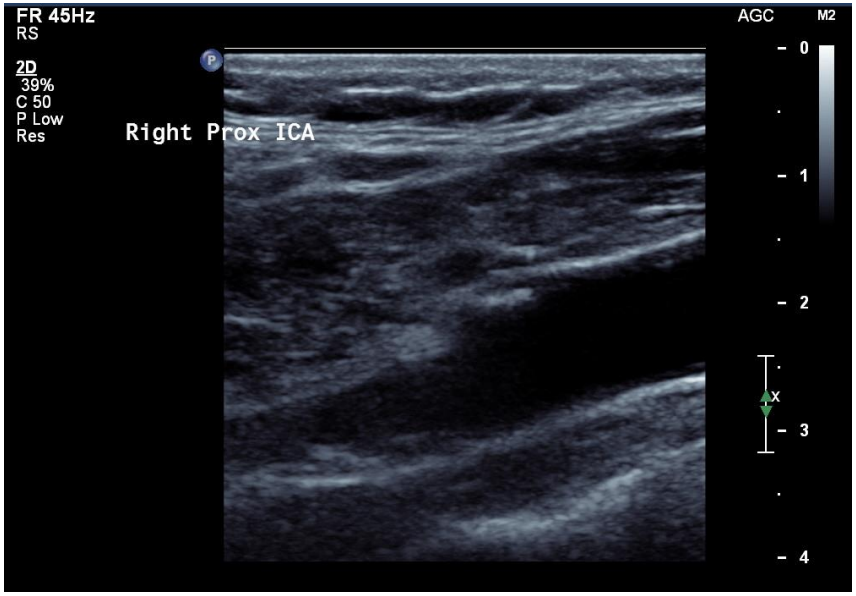
History

- Last 3-4 weeks Left leg, then Left arm symptoms followed by Right eye symptoms; Entire visual field blurred with associated diamond shaped flashing lights this last approx 30 minutes and then spontaneously recovers. There are no associated "shutter symptoms".
- In the last month there have been x 20 attacks affecting the left arm and leg and x 30 attacks affecting the right eye

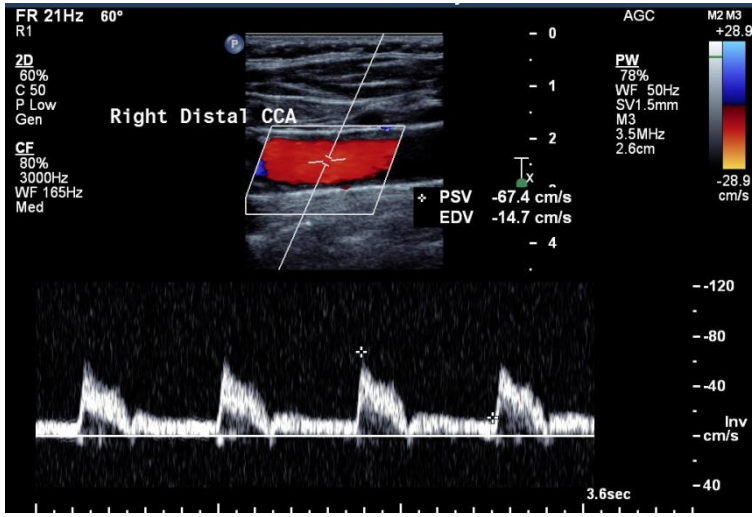
Past medical history

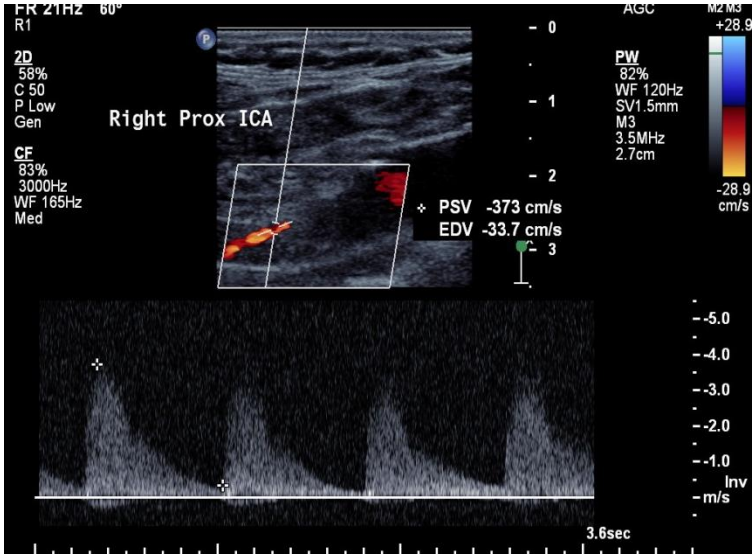
- Hypertension
- Type II DM
- Hyperlipidaemia
- Previous Left CEA
- Wife suggest could it be same as the left side
- Referred for outpatient ultrasound scan

Carotid Duplex scan



- Right Carotid:
  CCA PSV 67cm/s
  ICA PSV 373cm/s
  ICA EDV 34cm/s
  ECA PSV 194cm/s
  IC/CC ratio: 5.6:1
  % ICA stenosis: 70-99%
  Vertebral direction: Antegrade
  Plaque location: Bulb, Proximal ICA
  Plaque classification: T2
  Intimal thickness: 0.9mm


FR 21Hz 60°
R1
2D
60%
C 50
P Low
Gen
CF
80%
3000Hz
WF 165Hz
Med
Right Distal CCA
AGC
M2 M3
+28.9
PW
78%
WF 50Hz
SV1.5mm
M3
3.5MHz
2.6cm
-28.9
cm/s
PSV -67.4 cm/s
EDV -14.7 cm/s
0
1
2
4
-120
-80
-40
Inv
cm/s
-40
3.6sec


FR 21Hz 60°
R1
2D
58%
C 50
P Low
Gen
CF
83%
3000Hz
WF 165Hz
Med
Right Prox ICA
AGC
M2 M3
+28.9
PW
82%
WF 120Hz
SV1.5mm
M3
3.5MHz
2.7cm
-28.9
cm/s
PSV -373 cm/s
EDV -33.7 cm/s
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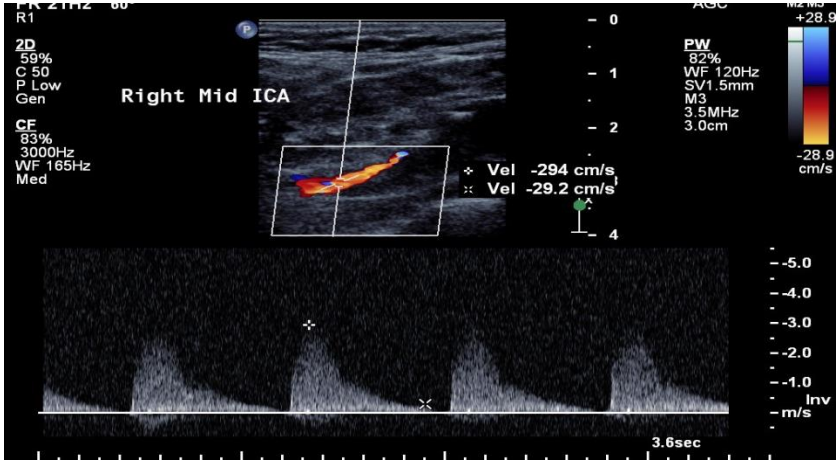
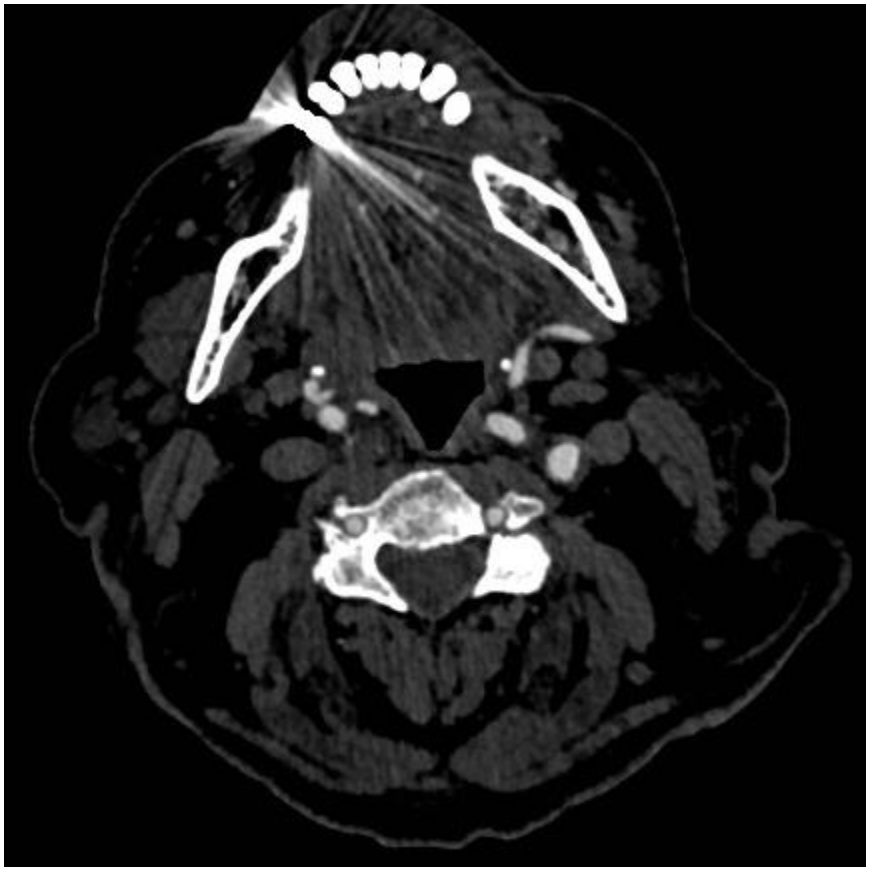

R1
2D
59%
C 50
P Low
Gen
CF
83%
3000Hz
WF 165Hz
Med
Right Mid ICA
PW
82%
WF 120Hz
SV1.5mm
M3
3.5MHz
3.0cm
+28.9
-28.9
cm/s
Vel -294 cm/s
Vel -29.2 cm/s
0
1
2
4
-5.0
-4.0
-3.0
-2.0
-1.0
Inv
m/s
3.6sec

Ultrasound Department

- Sonographers call the Brain Attack Team (BAT)
- Admitted under Stroke physicians

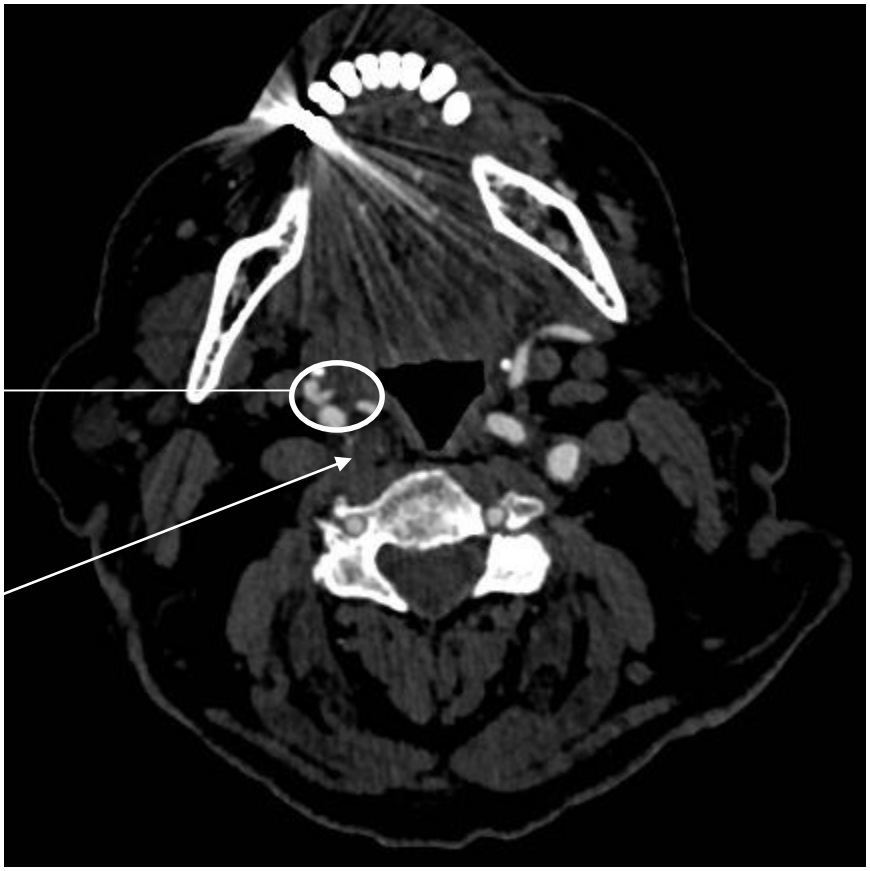
Question

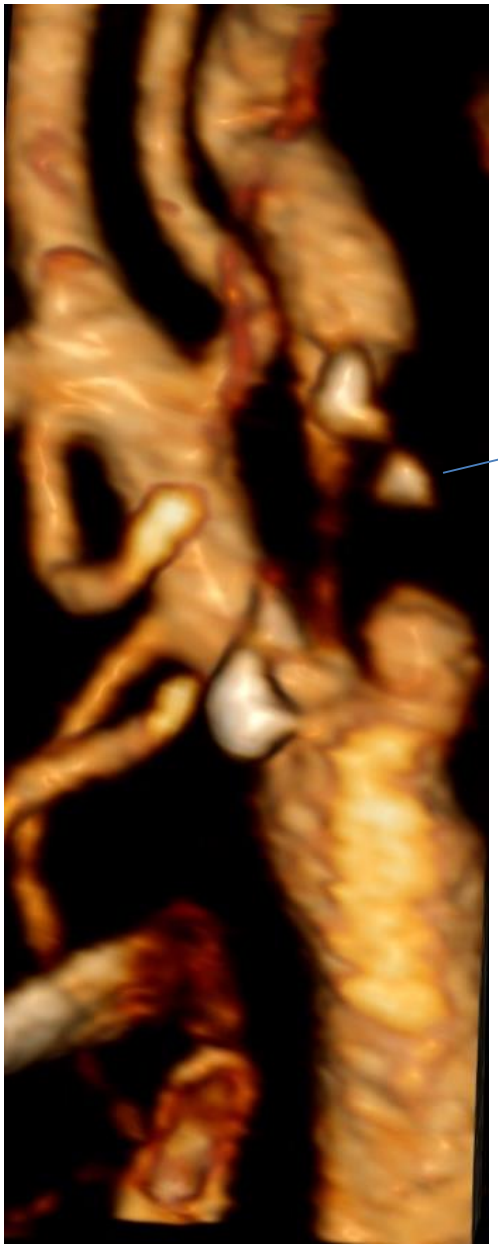
- Does the patient require secondary imaging ?



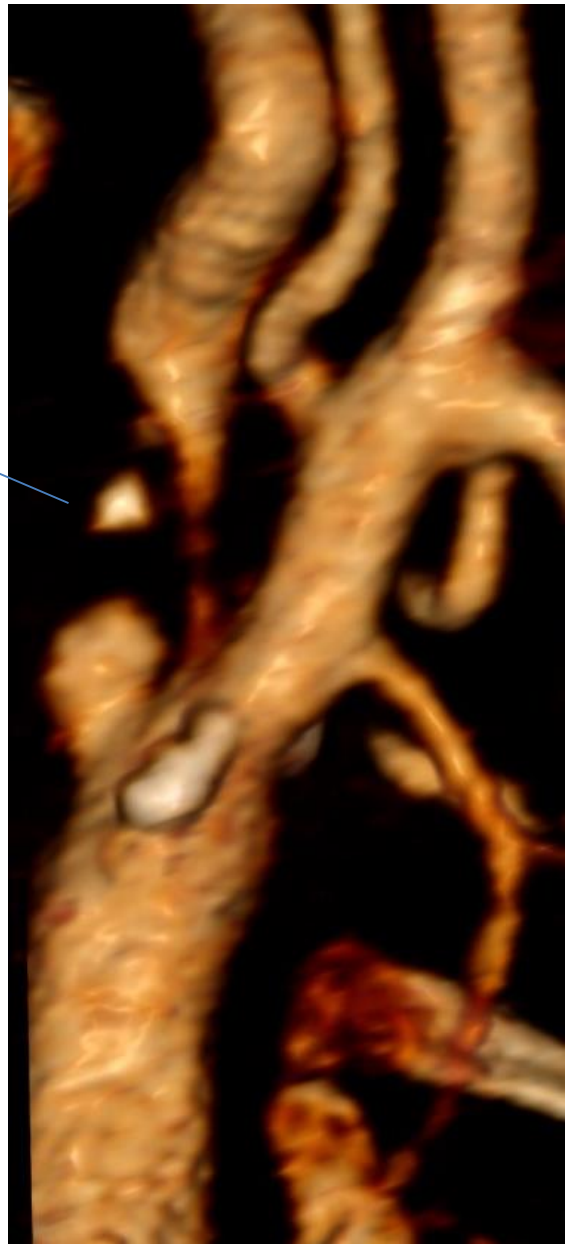
Right external carotid arteries and branches

Significantly stenosed right ICA





3D CTA reconstructions of the right common carotid artery bifurcation showing significant stenosis of the right ICA and several plaques of mural calcification



How would you manage?

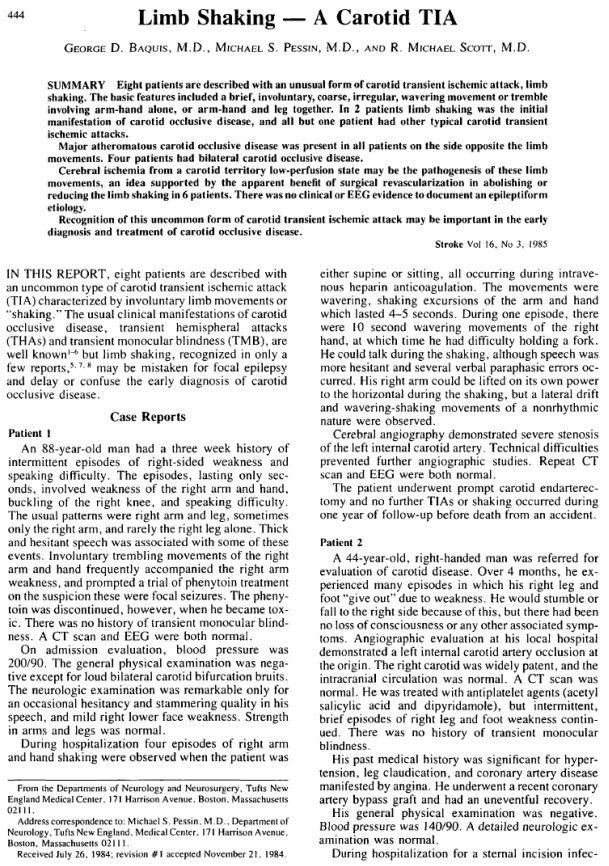
aphasia, visuospatial problems). Most symptoms are "negative" (i.e. loss of function), but occasionally a "limb-shaking" TIA can occur, characterised by involuntary limb movements caused by haemodynamic failure in patients with severe carotid stenoses (or occlusion). "Crescendo

Ophthalmology

- O/E mild non-proliferative diabetic retinopathy R > L no macular oedema. Significant arterial sclerosis R > L , but no obvious emboli.
- His retina is flat and he has early cataracts and mild vitreo-macular adhesion in both eyes

Stroke Physician Report

- Limb-Shaking-Syndrome TIA (LSS).
- *"Pathologically this is due to hypoperfusion, though there aren't huge descriptors of postural change and I note his BP+ and need for treatment and primary / secondary prevention for vascular disease. Withdrawing his medication alone would probably not be enough in this situation".*
- *"I feel that there should be a consideration given to undertaking a RCEA in the first instance".*



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Limb Shaking — A Carotid TIA

GEORGE D. BAQUIS, M.D., MICHAEL S. PESSIN, M.D., AND R. MICHAEL SCOTT, M.D.

SUMMARY **Eight patients are described with an unusual form of carotid transient ischemic attack, limb shaking. The basic features included a brief, involuntary, coarse, irregular, wavering movement or tremble involving arm-hand alone, or arm-hand and leg together. In 2 patients limb shaking was the initial manifestation of carotid occlusive disease, and all but one patient had other typical carotid transient ischemic attacks.**

Major atheromatous carotid occlusive disease was present in all patients on the side opposite the limb movements. Four patients had bilateral carotid occlusive disease.

Cerebral ischemia from a carotid territory low-perfusion state may be the pathogenesis of these limb movements, an idea supported by the apparent benefit of surgical revascularization in abolishing or reducing the limb shaking in 6 patients. There was no clinical or EEG evidence to document an epileptiform etiology.

Recognition of this uncommon form of carotid transient ischemic attack may be important in the early diagnosis and treatment of carotid occlusive disease.

Stroke Vol 16, No 3, 1985

IN THIS REPORT, eight patients are described with an uncommon type of carotid transient ischemic attack (TIA) characterized by involuntary limb movements or "shaking." The usual clinical manifestations of carotid occlusive disease, transient hemispheral attacks (THAs) and transient monocular blindness (TMB), are well known[1-6] but limb shaking, recognized in only a few reports,[5, 7, 8] may be mistaken for focal epilepsy and delay or confuse the early diagnosis of carotid occlusive disease.

Case Reports

Patient 1

An 88-year-old man had a three week history of intermittent episodes of right-sided weakness and speaking difficulty. The episodes, lasting only seconds, involved weakness of the right arm and hand, buckling of the right knee, and speaking difficulty. The usual patterns were right arm and leg, sometimes only the right arm, and rarely the right leg alone. Thick and hesitant speech was associated with some of these events. Involuntary trembling movements of the right arm and hand frequently accompanied the right arm weakness, and prompted a trial of phenytoin treatment on the suspicion these were focal seizures. The phenytoin was discontinued, however, when he became toxic. There was no history of transient monocular blindness. A CT scan and EEG were both normal.

On admission evaluation, blood pressure was 200/90. The general physical examination was negative except for loud bilateral carotid bifurcation bruits. The neurologic examination was remarkable only for an occasional hesitancy and stammering quality in his speech, and mild right lower face weakness. Strength in arms and legs was normal.

During hospitalization four episodes of right arm and hand shaking were observed when the patient was either supine or sitting, all occurring during intravenous heparin anticoagulation. The movements were wavering, shaking excursions of the arm and hand which lasted 4–5 seconds. During one episode, there were 10 second wavering movements of the right hand, at which time he had difficulty holding a fork. He could talk during the shaking, although speech was more hesitant and several verbal paraphasic errors occurred. His right arm could be lifted on its own power to the horizontal during the shaking, but a lateral drift and wavering-shaking movements of a nonrhythmic nature were observed.

Cerebral angiography demonstrated severe stenosis of the left internal carotid artery. Technical difficulties prevented further angiographic studies. Repeat CT scan and EEG were both normal.

The patient underwent prompt carotid endarterectomy and no further TIAs or shaking occurred during one year of follow-up before death from an accident.

Patient 2

A 44-year-old, right-handed man was referred for evaluation of carotid disease. Over 4 months, he experienced many episodes in which his right leg and foot "give out" due to weakness. He would stumble or fall to the right side because of this, but there had been no loss of consciousness or any other associated symptoms. Angiographic evaluation at his local hospital demonstrated a left internal carotid artery occlusion at the origin. The right carotid was widely patent, and the intracranial circulation was normal. A CT scan was normal. He was treated with antiplatelet agents (acetyl salicylic acid and dipyridamole), but intermittent, brief episodes of right leg and foot weakness continued. There was no history of transient monocular blindness.

His past medical history was significant for hypertension, leg claudication, and coronary artery disease manifested by angina. He underwent a recent coronary artery bypass graft and had an uneventful recovery.

His general physical examination was negative. Blood pressure was 140/90. A detailed neurologic examination was normal.

During hospitalization for a sternal incision infec-

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Are Limb-Shaking Transient Ischemic Attacks a Risk Factor for Postendarterectomy Hemorrhage? Case Report and Literature Review

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ABSTRACT

Postoperative intracerebral hemorrhage occurs in about 0.5% of all carotid endarterectomies. There are no recognized risk factors for this complication. We report on a 74-year-old woman with right sided limb-shaking transient ischemic attacks and severe stenosis of the left internal carotid artery. She suffered a fatal intracerebral hemorrhage 11 days after endarterectomy. This case prompted a review of the literature to determine if limb-shaking transient ischemic attacks might be a risk factor for postoperative intracerebral hemorrhage. We propose that patients with limb-shaking transient ischemic attacks have loss of vasomotor reactivity placing them at high risk for carotid reperfusion syndrome and hemorrhage into the revascularized territory.

Keywords: Transient ischemic attack, carotid artery stenosis, carotid endarterectomy.

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Introduction

We describe a case of right-sided limb-shaking transient ischemic attacks (LSTIAs) who developed hemorrhage into the left middle cerebral artery territory following a left carotid endarterectomy (CEA). This case served as an impetus for a review of the pathophysiology of limb-shaking cerebral ischemia and a possible connection with carotid reperfusion syndrome and postendarterectomy hemorrhage.

Case Report

A 74-year-old right-handed white female presented with a 3 month history of brief episodes of right arm and leg jerking that occurred on standing. Her past medical history was significant for hypertension, coronary artery disease, and emphysema. She had smoked one to two packs per day until quitting in 1984. Her medications included aspirin, hydrochlorothiazide, irbesartan, and pravistatin. She had up to three spells per day with occasional falls. The jerking movements usually abated spontaneously within less than a minute. No episodes of generalized convulsive activity, loss of consciousness, postictal confusion or bowel or bladder incontinence had occurred.

Her general examination was remarkable for bilateral carotid bruits. She had a mild right pronator drift but an otherwise unremarkable neurological exam.

Carotid duplex ultrasound demonstrated a greater than 90% stenosis of the left internal carotid artery (ICA) and 40–60% stenosis of the right ICA. Transcranial Doppler ultrasound (TCD) revealed high-velocity low-resistance waveforms in the left posterior cerebral artery suggesting collateral flow. These findings were confirmed by catheter angiography (Fig 1). In addition, very slow antegrade filling of the left hemisphere anterior circulation was demonstrated, with retention of contrast in the ICA siphon late into the angiographic sequence confirming prolonged circulation time (Fig 2). The anterior communicating artery was not patent and there was no cross filling (Fig 3). The left posterior communicating artery was also not

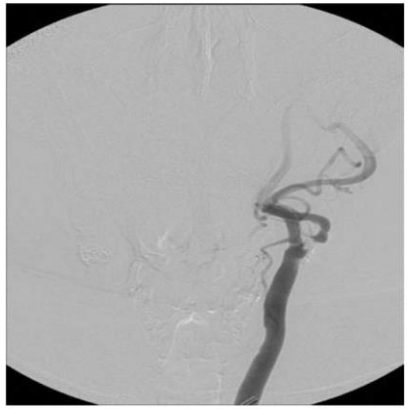


Fig 1. Left common carotid angiogram, arterial phase, AP view, shows a short segment atherosclerotic plaque with very high grade stenosis.

Table 1. ICH Postrevascularization for Limb-Shaking TIA

Treatment	# of Cases	ICH
CEA(3, 4, 8, 11, 14–16)	13	3
CAS(11)	1	0

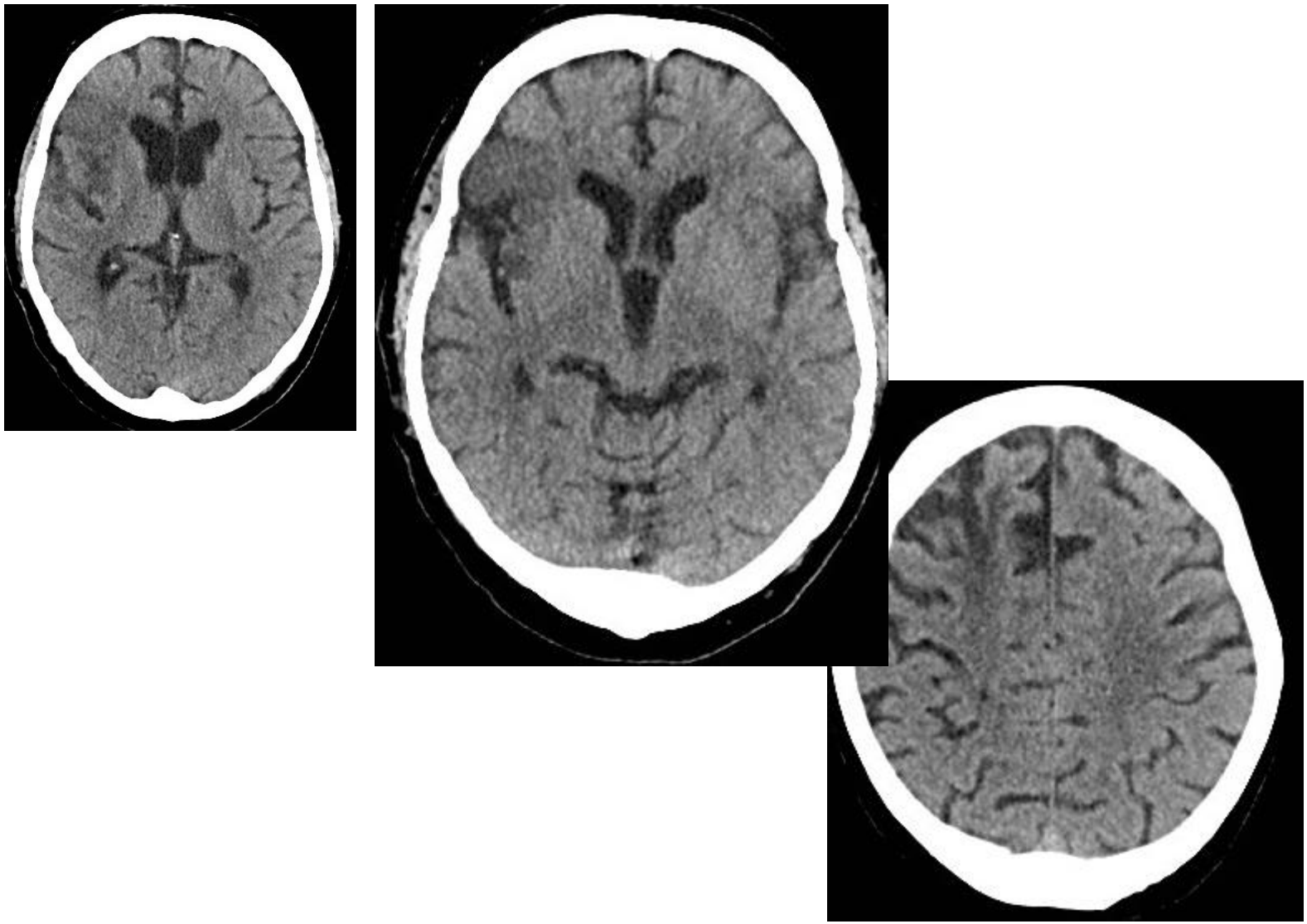
Operation

- Patient undergoes a CEA under LA
- Significant vagal response on dissection of CCA and ICA
- No Shunt
- Bovine Patch
- No complications

Case B

History

- Admitted 03/05/2018, 11:00 am
- Bladder biopsy on the 01/05/2018
- Stopped Rivoroxaban for 3 days as per request from urology.
- Left facial droop and slurred speech, unsteady on feet
- Type II DM
- Coronary Artery Disese, MI, Paroxysmal atrial flutter
- Hypertension
- MRSA + ve



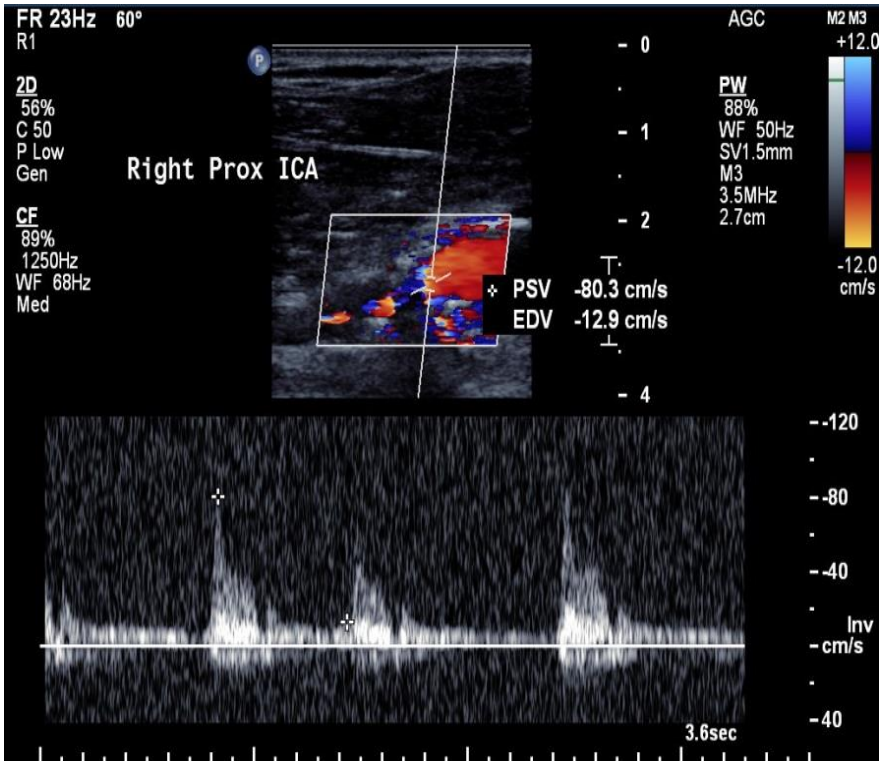
Day of admission under Stroke team
CT Head 03/05/2018

CT Head 03/05/2018

- There is an **subacute right MCA territory infarction** involving the right frontal lobe, and extending into the anterior right insula and lateral right temporal lobe.
- Right frontal watershed territory infarct appears more longstanding.
- Small established infarct at the head of the left caudate nucleus.

Carotid Ultrasound 04/05/2018

- • **Right Carotid:**
- CCA PSV 43 cm/s
- ICA PSV 80 cm/s (at origin, reduces to trickle flow before occluding)
- ICA EDV 13 cm/s
- ECA PSV 199 cm/s
- IC/CC ratio: 2:1
- **% ICA stenosis: 100 %**
- Vertebral direction: antegrade
- Plaque location: prox ICA
- Plaque classification: 5
- Comments: The right ICA reduces to trickle flow before occluding just beyond the origin.



Carotid Ultrasound 04/05/2018

- **Left Carotid:**
- CCA PSV 56 cm/s
- ICA PSV 370 cm/s
- ICA EDV 83 cm/s
- ECA PSV 197 cm/s
- IC/CC ratio: 6.6:1
- **% ICA stenosis: 70-99 %**
- Vertebral direction: antegrade
- Plaque location: prox ICA
- Plaque classification: 5
- There is a significant left ICA origin stenosis.

- Carotid Plaque 1- 5
- 1 - Uniform echolucent plaque
- 2 – Predominant echolucent plaques < 50% echogenic areas
- 3 – Predominantly echogenic plaques with < 50% echolucent areas
- 4 – Uniformly echogenic plaques
- 5 – Unable to classify due to calcification and acoustic shadow

How do you manage ?

Eur J Vasc Endovasc Surg (2018) 55, 3–81

Editor's Choice — Management of Atherosclerotic Carotid and Vertebral Artery Disease: 2017 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)

A.R. Naylor [a], J.-B. Ricco [a], G.J. de Borst [a], S. Debus [a], J. de Haro [a], A. Halliday [a], G. Hamilton [a], J. Kakisis [a], S. Kakkos [a], S. Lepidi [a], H.S. Markus [a], D.J. McCabe [a], J. Roy [a], H. Sillesen [a], J.C. van den Berg [a], F. Vermassen [a], ESVS Guidelines Committee [b], P. Kolh, N. Chakfe, R.J. Hinchliffe, I. Koncar, J.S. Lindholt, M. Vega de Ceniga, F. Verzini, ESVS Guideline Reviewers [c], J. Archie, S. Bellmunt, A. Chaudhuri, M. Koelemay, A.-K. Lindahl, F. Padberg, M. Venermo

Keywords: Carotid, Vertebral, Stroke, Transient ischaemic attack, Endarterectomy, Stenting, Medical therapy, Screening, Dementia, Asymptomatic, Symptomatic, Thrombolysis, Imaging, Bypass, Surgical techniques, Complications, Patch infection, Restenosis

TABLE OF CONTENTS

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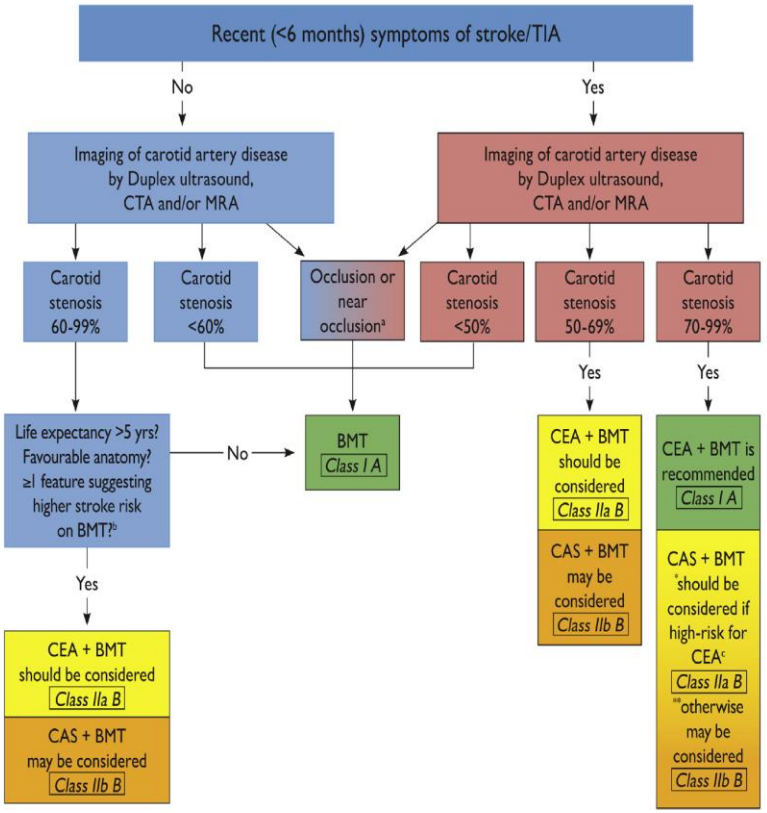
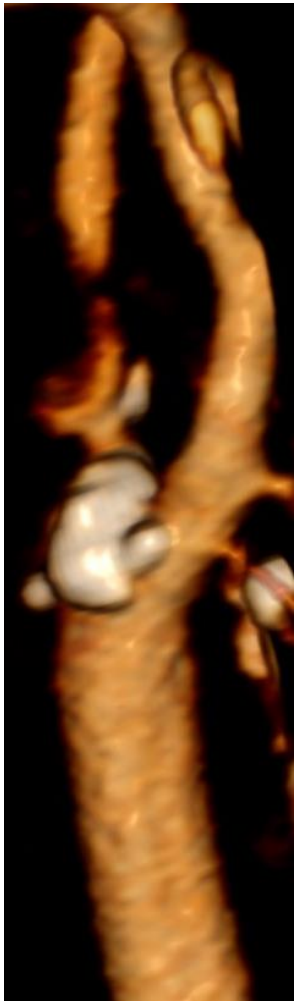
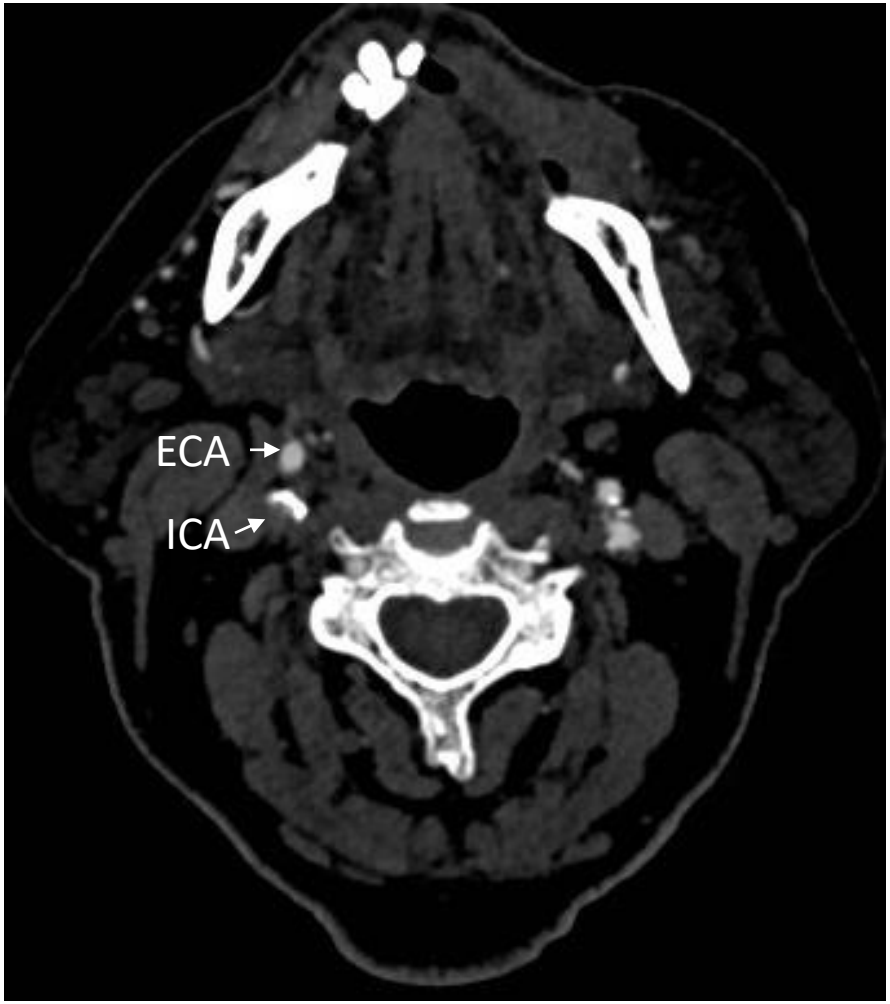


CTA significant right ICA stenosis with mural calcification

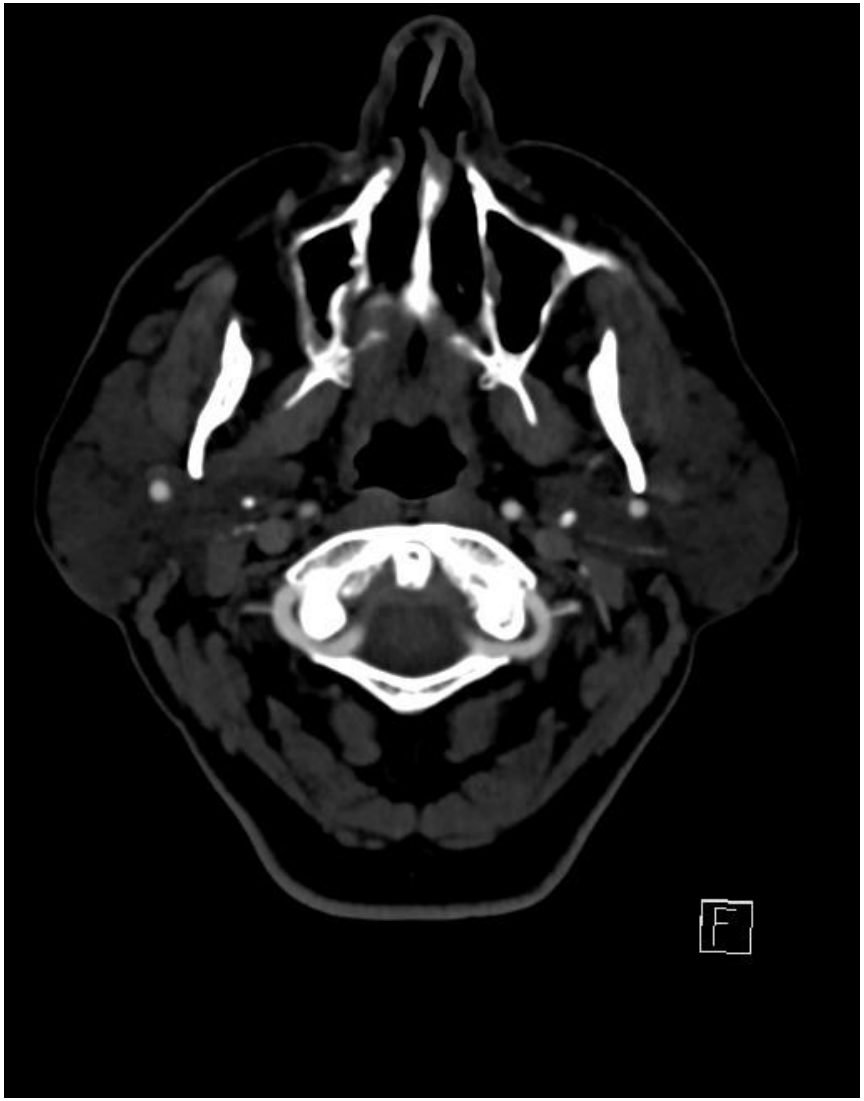


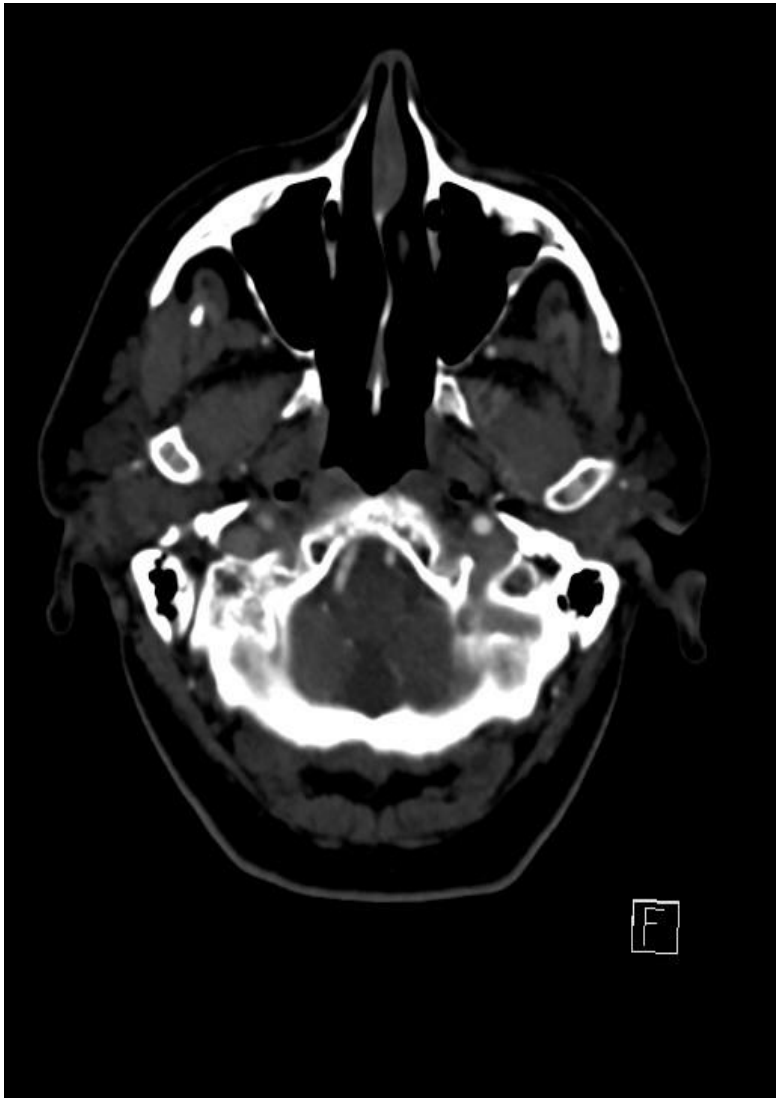
3D reconstruction



Axial CTA

4/5/2018





National Vascular Registry 2016

Grade of ipsilateral carotid stenosis* (n=4,620)				
<50%	68	1.5	2.0	0.9
50-69%	1,132	24.5	23.8	25.8
70-89%	1,982	42.9	45.0	44.1
90-99%	1,429	30.9	28.9	28.8
Occluded	9	0.2	0.3	0.5

“High Risk for Stroke” asymptomatic patients

Table 5. Clinical/imaging features associated with an increased risk of late stroke in patients with asymptomatic 50–99% stenoses treated medically.

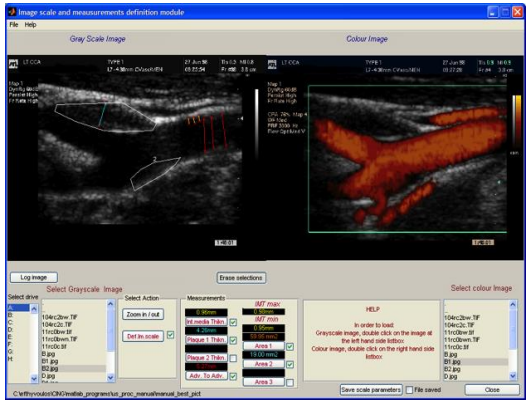
Imaging/clinical parameter and stenosis severity Type of study	Annual rate of ipsilateral stroke	OR/HR (95% CI) $p =$
Silent infarction on CT[84] 60–99% stenoses Multicentre, observational	Yes = 3.6% No = 1.0%	3.0 (1.46–6.29) $p = .002$
Stenosis progression[85] 50–99% stenoses Multicentre, observational	Regression = 0.0% Unchanged = 1.1% Progression = 2.0%	1.92 (1.14–3.25) $p = .05$
Stenosis progression[86] 70–99% stenoses Multicentre, RCT	Regression No change Progression 1 stenosis grade Progression 2 stenosis grades	0.7 (0.4–1.3) Comparator 1.6 (1.1–2.4) 4.7 (2.3–9.6)
Plaque area on computerised plaque analysis[87] 70–99% Multicentre, observational	<40 mm^2 = 1.0% 40–80 mm^2 = 1.4% >80 mm^2 = 4.6%	HR 1.0 2.08 (95% CI 1.05–4.12) 5.81 (95% CI 2.67–12.67)
JBA on computerised plaque analysis[88] 50–99% stenoses Multicentre, observational	<4 mm^2 = 0.4% 4–8 mm^2 = 1.4% 8–10 mm^2 = 3.2% >10 mm^2 = 5.0%	Trend $p < .001$
Intra-plaque haemorrhage on MRI[89] 50–99% stenoses Meta-analysis	Yes vs. no	OR 3.66 (2.77–4.95) $p < .01$
Impaired CVR[90] 70–99% stenoses Meta-analysis	Yes vs. no	OR 6.14 (95% CI 1.27–29.5) $p = .02$
Plaque lucency on Duplex US[91] 50–99% stenoses Meta-analysis	Predominantly echolucent 4.2% Predominantly echogenic 1.6%	OR 2.61 (95% CI 1.47–4.63) $p = .001$
Spontaneous embolisation on TCD[92] 50–99% stenoses Meta-analysis	Yes vs. no	OR 7.46 (95% CI 2.24–24.89) $p = .001$
Spontaneous embolisation plus uniformly or predominantly echolucent plaque[93] 70–99% stenoses Multicentre, observational	Yes = 8.9% No = 0.8%	OR 10.61 (95% CI 2.98–37.82) $p = .0003$
Contralateral TIA/stroke[94] 50–99% stenoses Multicentre, observational	Yes = 3.4% No = 1.2%	OR 3.0 (95% CI 1.9–4.73) $p = .0001$

Spontaneous embolisation plus uniformly or predominantly echolucent plaque[93] 70–99% stenoses Multicentre, observational — Yes = 8.9% No = 0.8% — OR 10.61 (95% CI 2.98–37.82) $p = .0003$

Contralateral TIA/stroke[94] 50–99% stenoses Multicentre, observational — Yes = 3.4% No = 1.2% — OR 3.0 (95% CI 1.9–4.73) $p = .0001$

Who pays?

- Imaging (Duplex/MRI)
- Transcranial Doppler
- Software
- Analysis



[Ann Neurol.](#) 2013 Jun;73(6):774-84.

ORIGINAL ARTICLE

Carotid Plaque Hemorrhage on Magnetic Resonance Imaging Strongly Predicts Recurrent Ischemia and Stroke

Akram A. Hosseini, MRCP, MD,[1] Neghal Kandiyil, MRCS,[1,2] Shane T. S. MacSweeney, FRCS, MChir,[2] Nishath Altaf, FRCS, PhD,[1,2] and Dorothee P. Auer, FRCR, PhD[1]

Objective: There is a recognized need to improve selection of patients with carotid artery stenosis for carotid endarterectomy (CEA). We assessed the value of magnetic resonance imaging (MRI)-defined carotid plaque hemorrhage (MRIPH) to predict recurrent ipsilateral cerebral ischemic events, and stroke in symptomatic carotid stenosis.
Methods: One hundred seventy-nine symptomatic patients with ≥50% stenosis were prospectively recruited, underwent carotid MRI, and were clinically followed up until CEA, death, or ischemic event. MRIPH was diagnosed if the plaque signal intensity was >150% that of the adjacent muscle. Event-free survival analysis was done using Kaplan–Meier plots and Cox regression models controlling for known vascular risk factors. We also undertook a meta-analysis of reported data on MRIPH and recurrent events.
Results: One hundred fourteen patients (63.7%) showed MRIPH, suffering 92% (57 of 62) of all recurrent ipsilateral events and all but 1 (25 of 26) future strokes. Patients without MRIPH had an estimated annual absolute stroke risk of only 0.6%. Cox multivariate regression analysis proved MRIPH as a strong predictor of recurrent ischemic events (hazard ratio [HR] = 12.0, 95% confidence interval [CI] = 4.8–30.1, $p < 0.001$) and stroke alone (HR = 35.0, 95% CI = 4.7–261.6, $p = 0.001$). Meta-analysis of published data confirmed this association between MRIPH and recurrent cerebral ischemic events in symptomatic carotid artery stenosis (odds ratio = 12.2, 95% CI = 5.5–27.1, $p < 0.00001$).
Interpretation: MRIPH independently and strongly predicts recurrent ipsilateral ischemic events, and stroke alone, in symptomatic ≥50% carotid artery stenosis. The very low stroke risk in patients without MRIPH puts into question current risk–benefit assessment for CEA in this subgroup.

ANN NEUROL 2013;73:774–784

The efficacy of carotid endarterectomy (CEA) in secondary prevention of stroke in patients with symptomatic severe carotid artery stenosis is well documented by pooled randomized controlled trial evidence.[1] Current guidelines recommend early surgical intervention for symptomatic individuals with 50 to 99% carotid stenosis as determined by angiographic or ultrasonographic measurement of the luminal diameter according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria.[2] However, 70 to 80% of symptomatic patients with ≥50% stenosis will not experience recurrent stroke at 5 years.[3,4] This group of patients at low risk of recurrent cerebral ischemic events routinely undergo potentially unnecessary surgical intervention, demonstrating the limitation of the current risk stratification model, based on degree of stenosis alone.[5]

Randomized controlled trials were mostly performed more than a decade ago, since which time there has been considerable progress in best medical management for secondary prevention of stroke. Since then, the EXPRESS study has successfully changed clinical practice to early initiation of medical therapy after transient ischemic attack (TIA) or minor stroke, lowering the risk of stroke recurrence.[6] This improvement in medical treatment may also reduce the additional benefit from surgery. Nonetheless, some recent guidelines recommend

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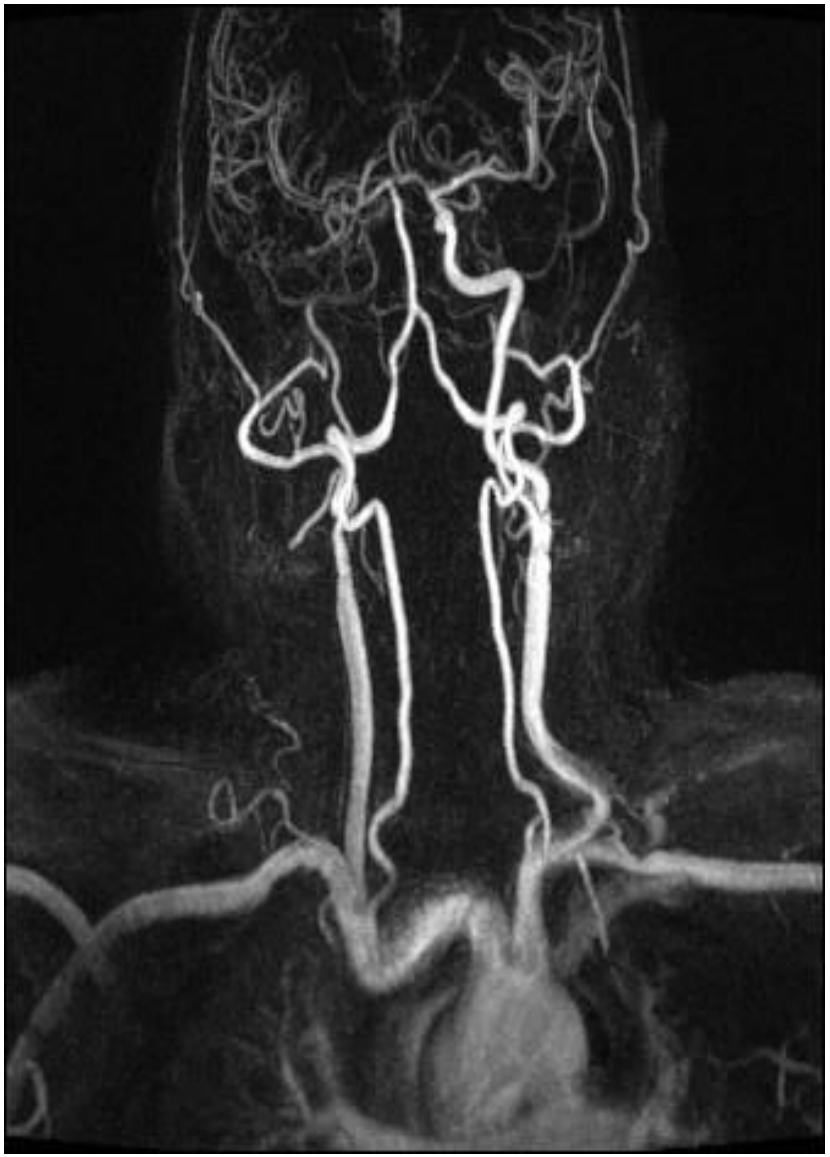
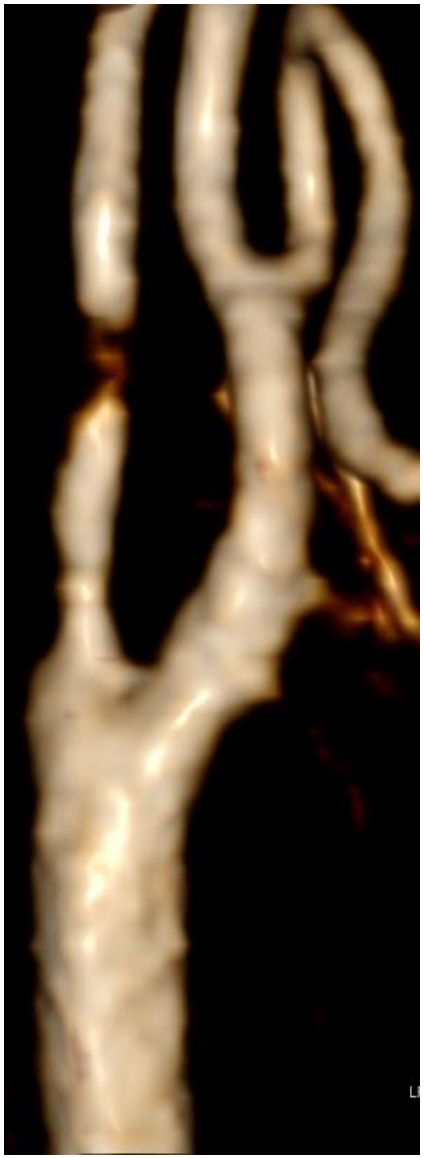
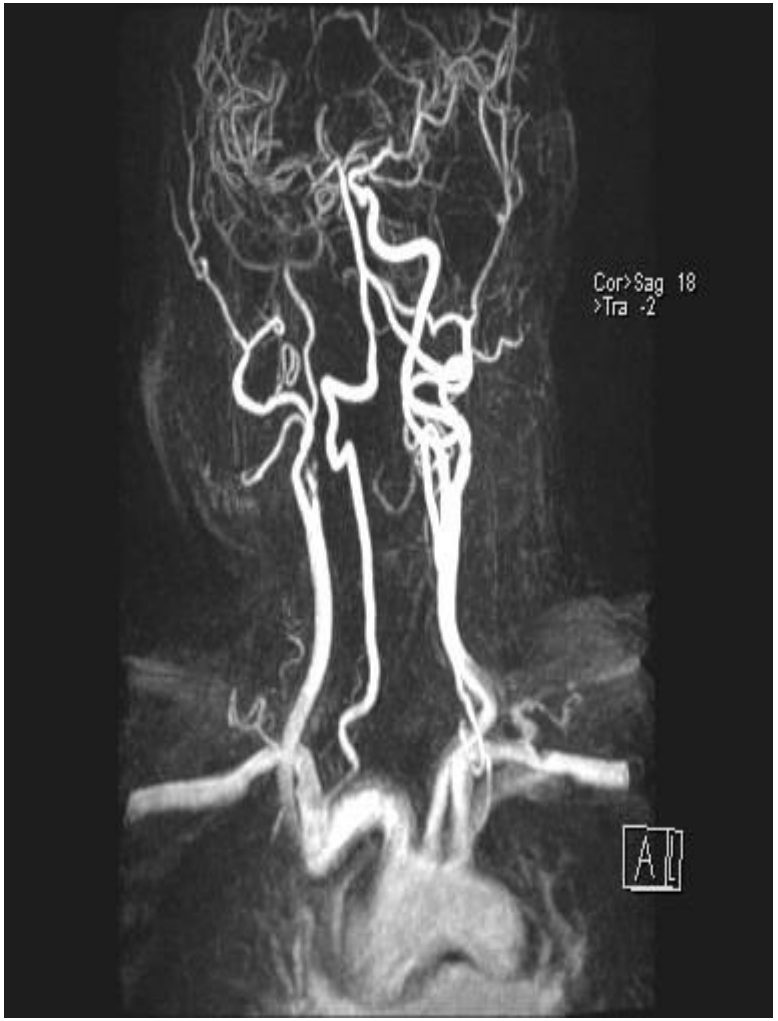
Additional Imaging; MRA

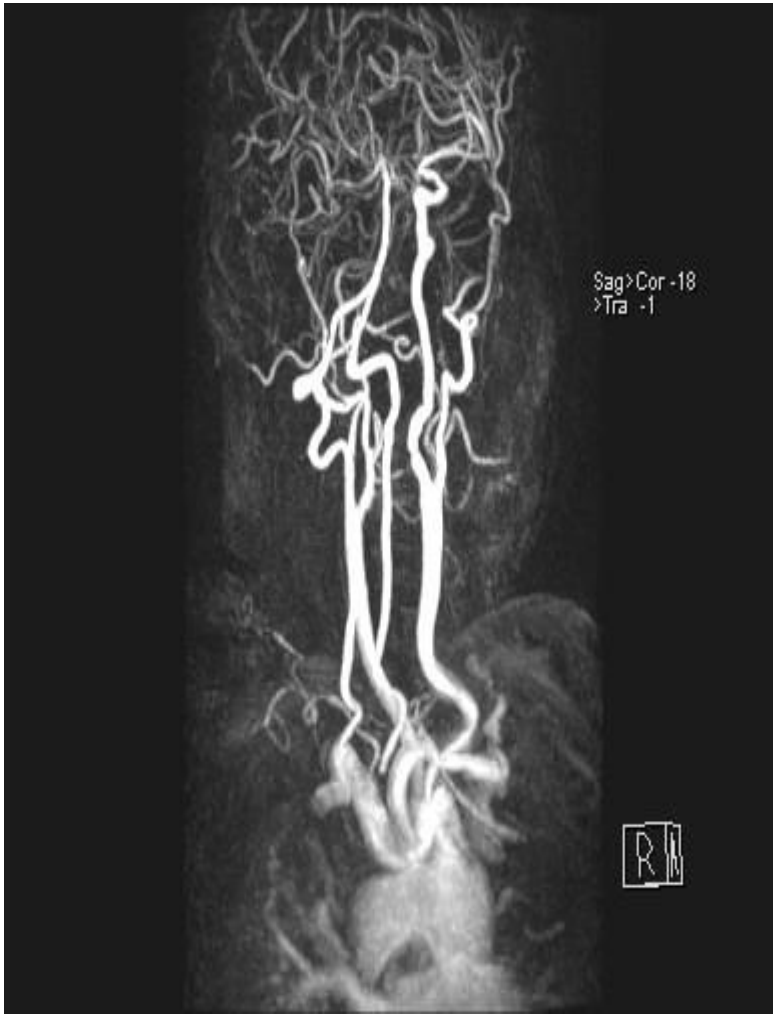




3D reconstruction of MRA showing significant stenosis of right ICA

MRA 10/05/2018



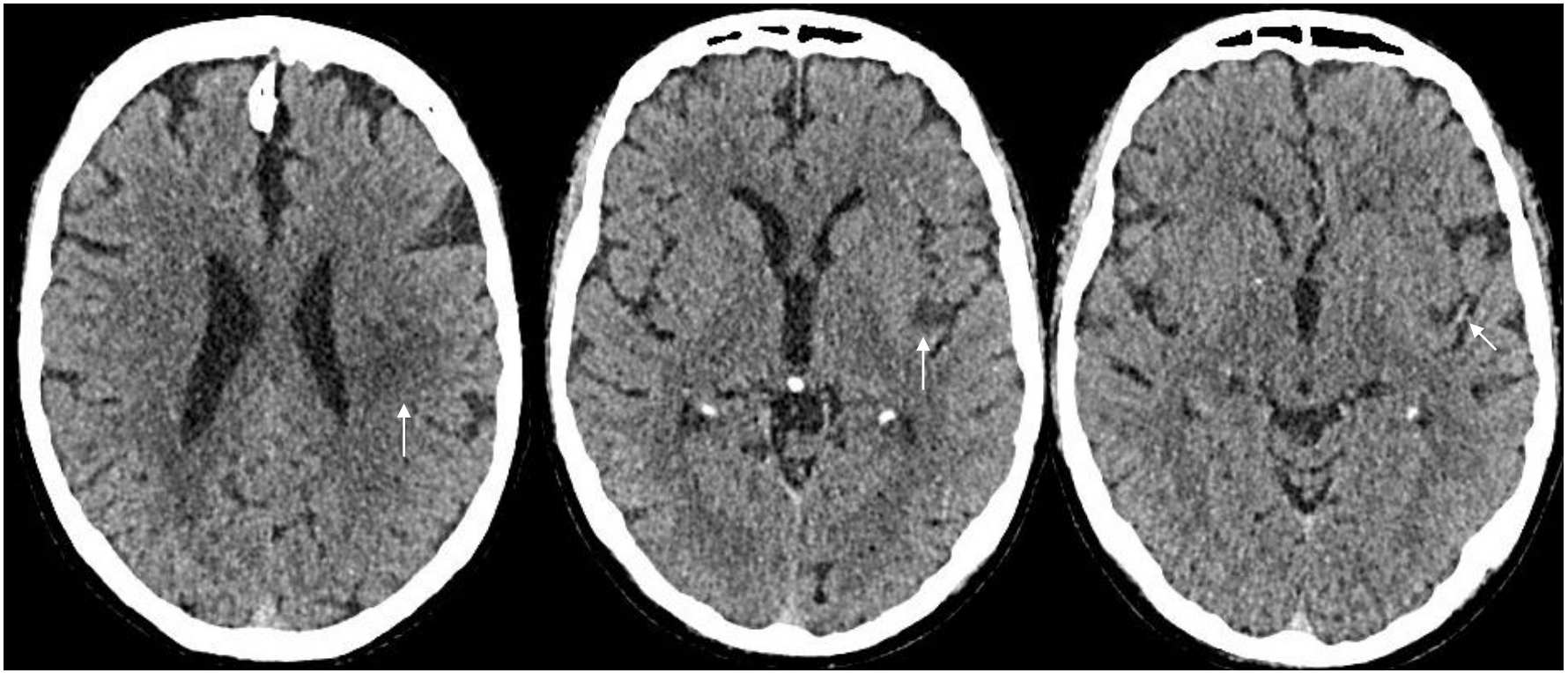


Case D

History

- 74 year still working as an engineer (wire spark erosion), previously ran 7 companies and employed 180 people.
- Saturday 5/5/2018 cleaning out the gutters , messing around with the ladder, right hand felt funny, had Brunch , feeling improved
- Sunday 6/5/2018 Family came for tea, Right hand symptoms returned, then developed expressive dysphasia, family suggested he go to the GP
- Tuesday 8/5/2018 Attended GP referred into hospital, saw BAT team had tests suggested come back Wednesday , but then the right arm and leg became progressively weaker.
- Currently being hoisted from chair.

Unenhanced CT
08/05/2018

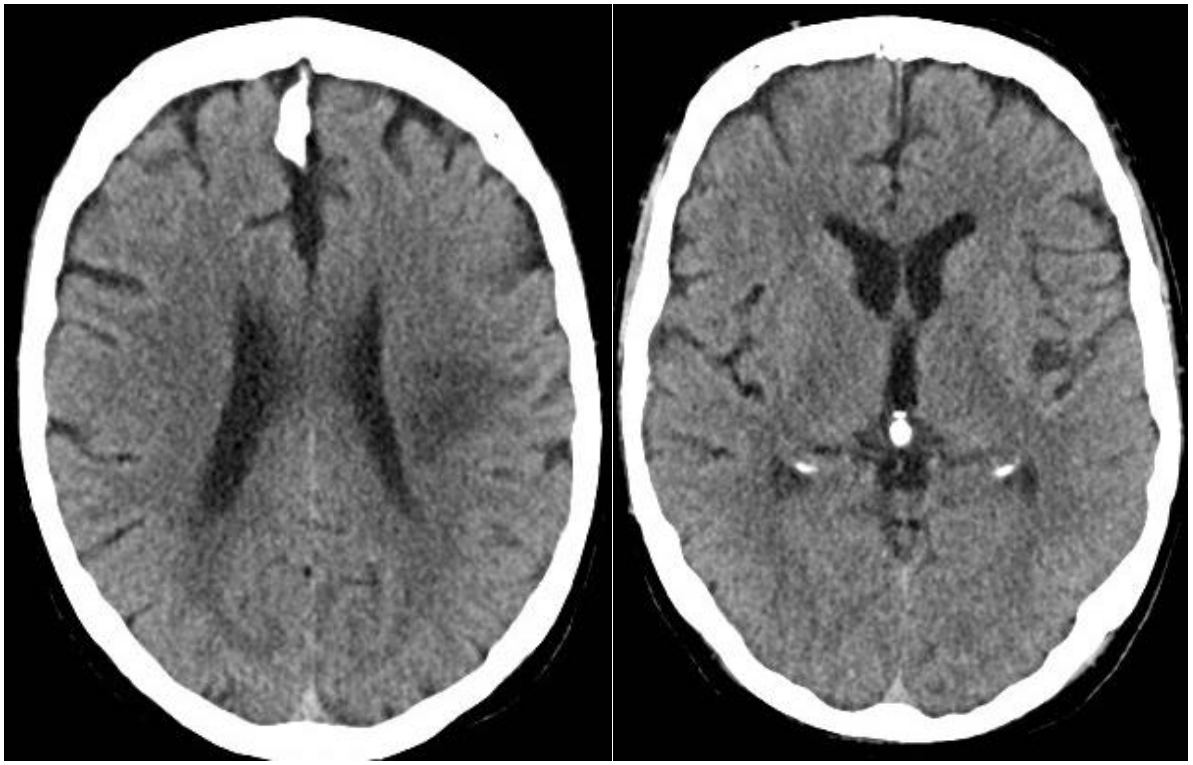


Acute infarction left corona radiata

Acute infarction left insula

Hyperdensity left middle cerebral artery indicating acute thrombus

Unenhanced CT
09/05/2018



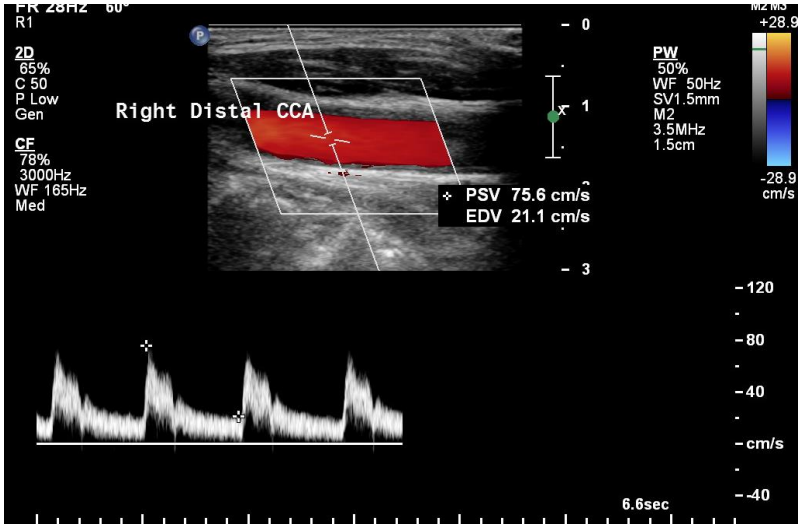
Maturation of left MCA infarction

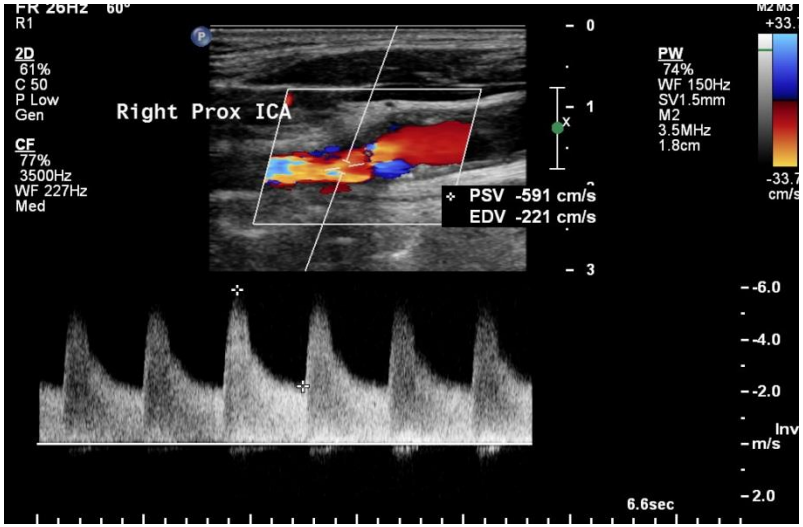
Carotid Duplex Scan 09/05/2018

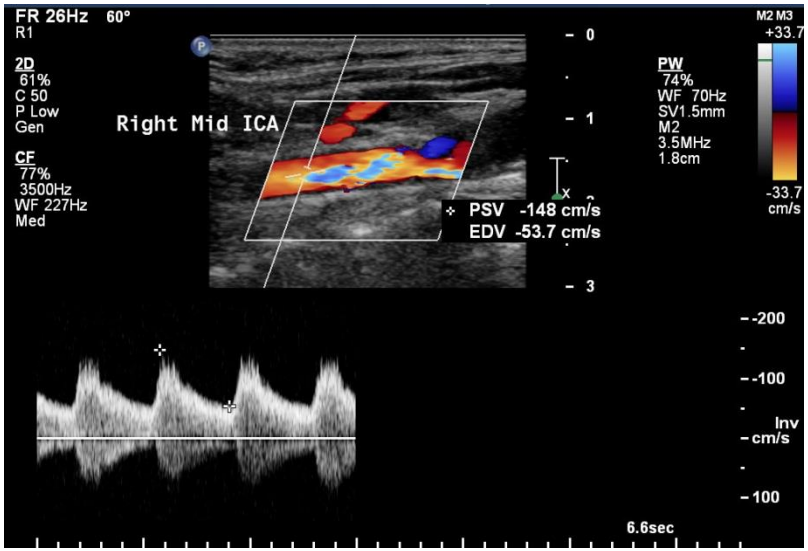
- Left Carotid:
- CCA PSV 61cm/s
- ICA PSV OCCLUDED
- ICA EDV OCCLUDED
- ECA PSV 138cm/s
- IC/CC ratio:N/A
- % ICA stenosis: N/A
- Vertebral direction: Antegrade
- Plaque location: Bulb
- Plaque classification: T3

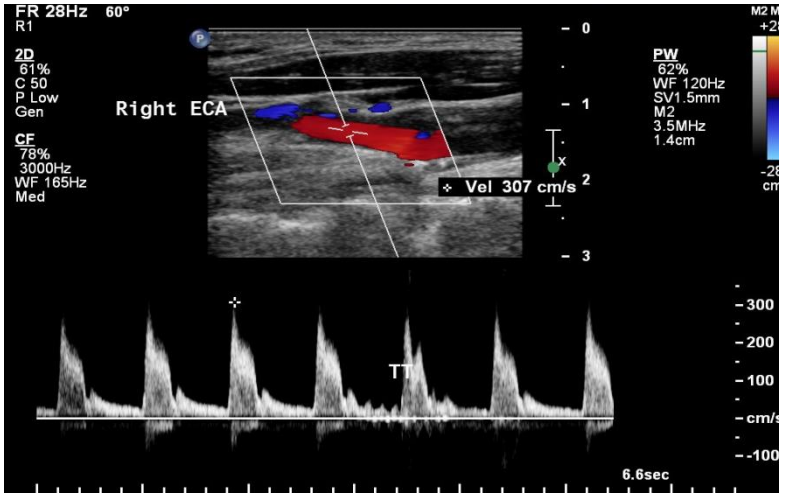
- Right Carotid:
- CCA PSV 76cm/s
- ICA PSV 591cm/s
- ICA EDV 221cm/s
- ECA PSV 307cm/s
- IC/CC ratio: 7.8:1
- % ICA stenosis: 0-49%
- Vertebral direction: Antegrade
- Plaque location: Bulb , ECA Prox ICA
- Plaque classification: T3

Carotid Duplex 09/05/2018









How do you manage ?

Eur J Vasc Endovasc Surg (2018) 55, 3–81

Editor's Choice — Management of Atherosclerotic Carotid and Vertebral Artery Disease: 2017 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)

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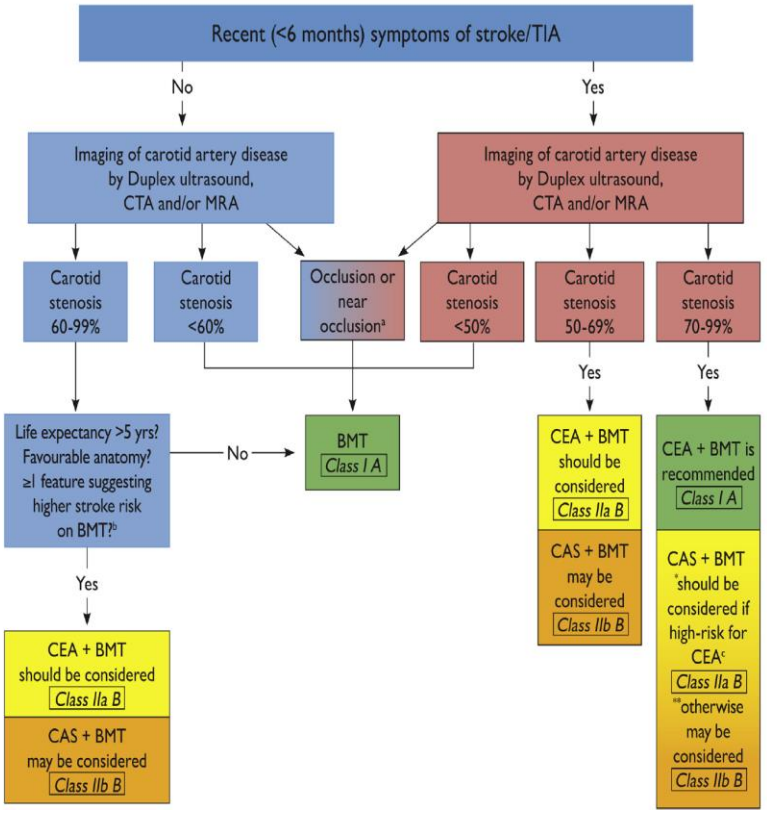

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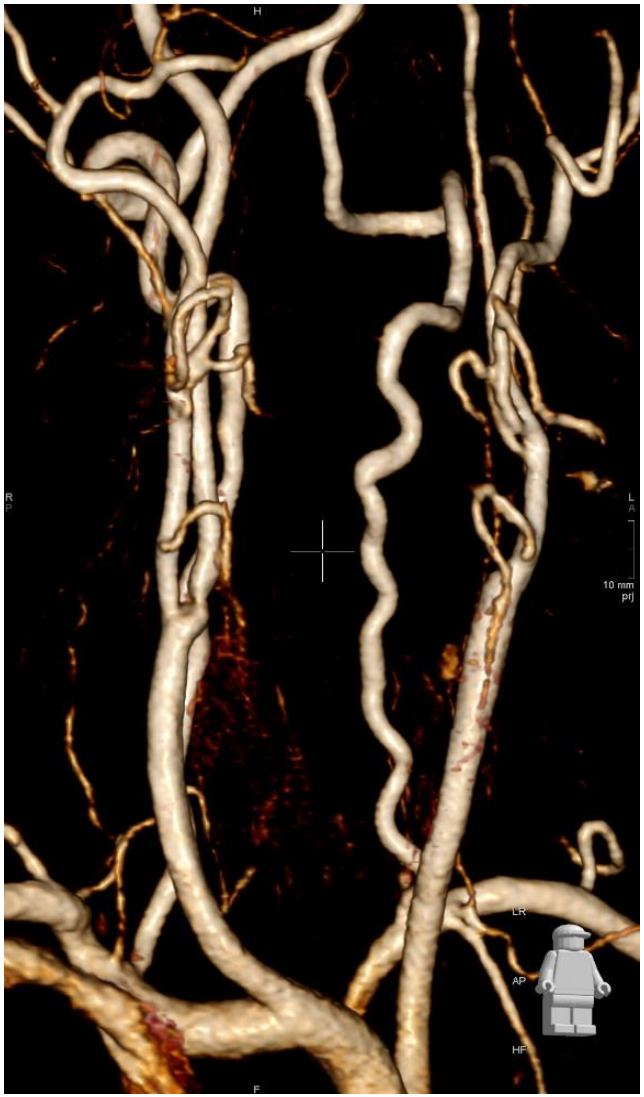

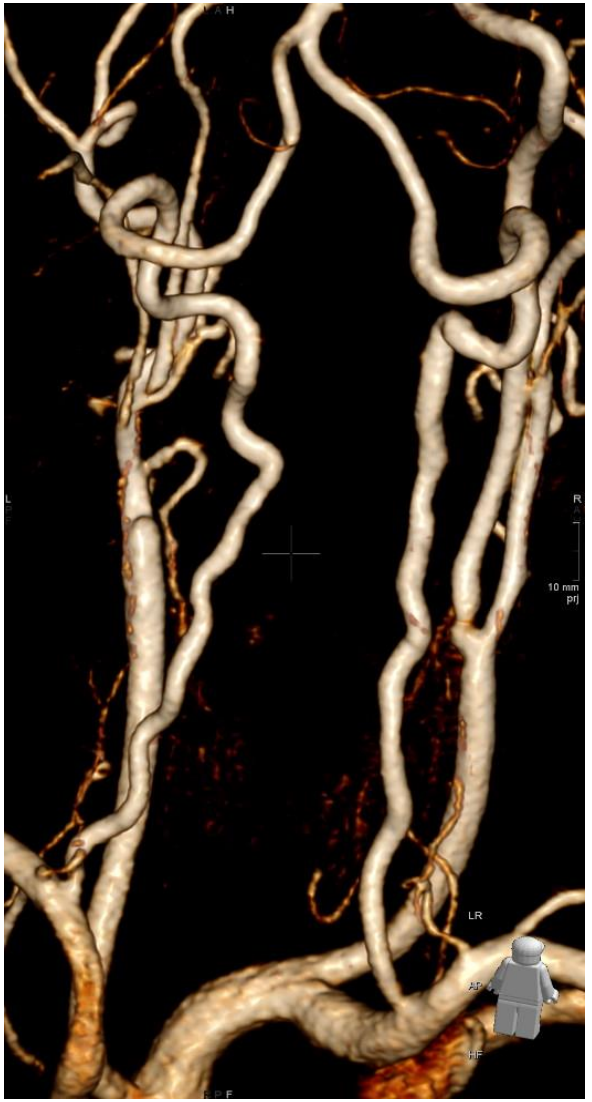

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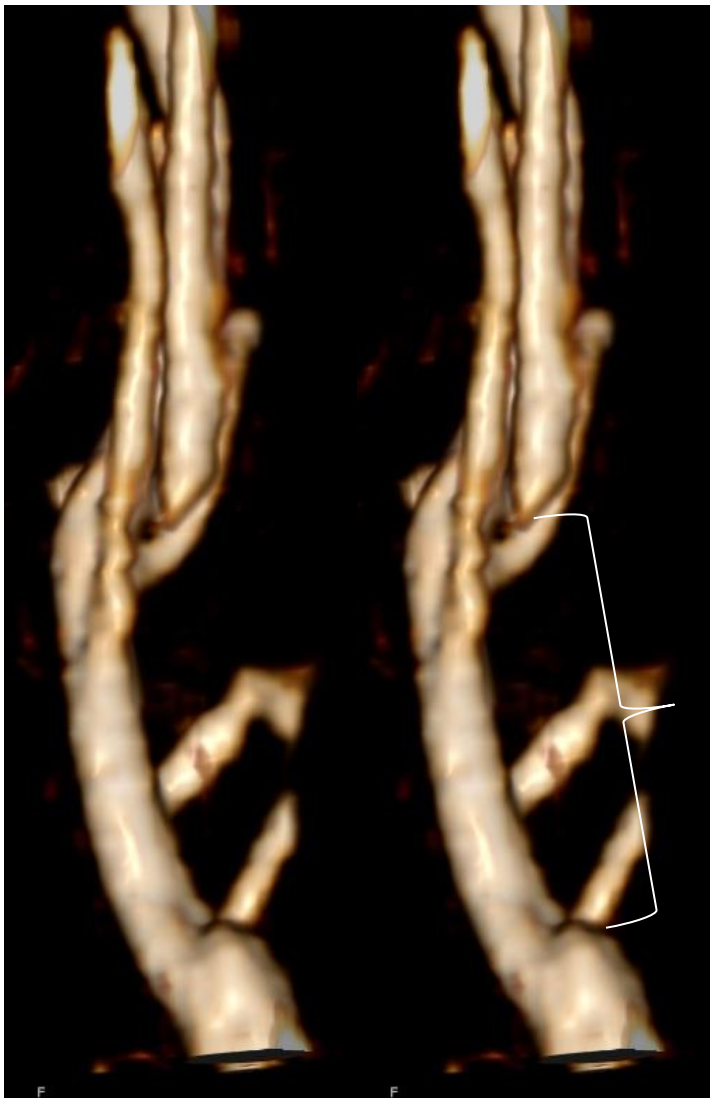
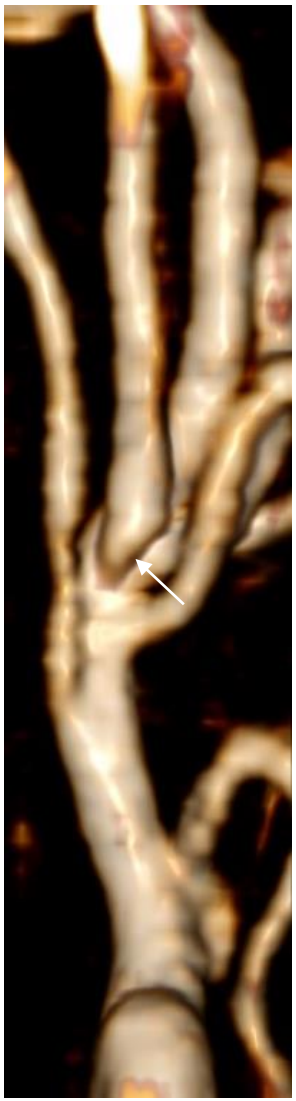
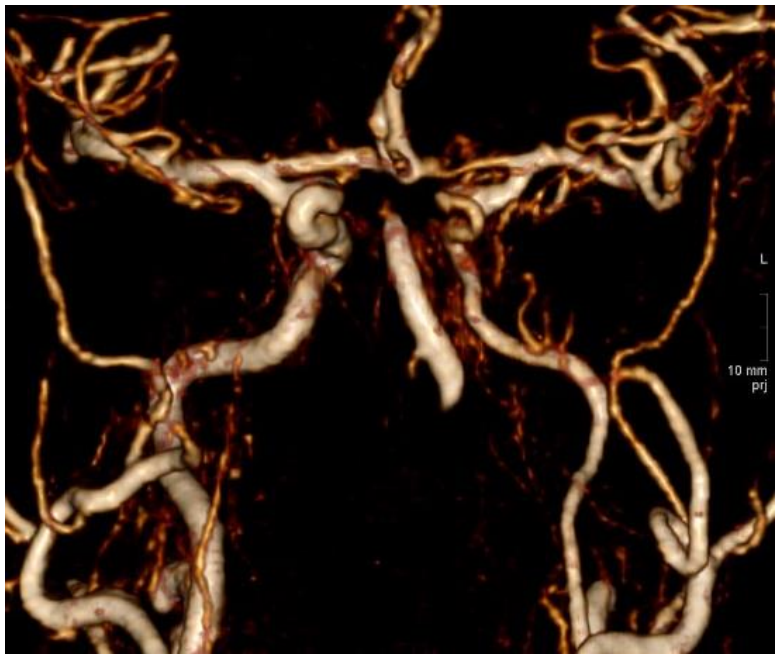




3D MRA: apparent occlusion of the proximal left ICA (depicted by length of bracket) with reconstitution distally (arrow)



3D MRA of the circle of Willis (basilar artery (centre) normal but truncated for imaging purposes).
Distal left ICA and its branches attenuated but patent

Question

- How would you manage the patient
- Left ICA
- Right ICA

CASE REPORT

Capsular warning syndrome

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SUMMARY

We present a case of a 72-year-old man who presented with fluctuating right-sided weakness and numbness. This was characterised by episodic sudden onset weakness with resolution of symptoms in between. His symptoms and signs were becoming persistent despite the addition of dual antiplatelet therapy. The history we describe is classical of capsular warning syndrome. The patient went on to have further definitive neuroimaging which revealed a pontine infarct, rather than the expected capsular infarct. We discuss the importance of capsular warning syndrome, the proposed pathophysiological mechanisms and different locations of infarction in previous cases of capsular warning syndrome.

We also discuss the lack of consensus (within the literature) in treatment options which are used to try and prevent a completed stroke occurring in cases of capsular warning syndrome.

BACKGROUND

Capsular warning syndrome (CWS) can be a dramatic occurrence in the clinical setting, which is important to recognise.

Despite its typical history and clinical course, the exact location of infarcts can vary from case to case. As in our case, instead of the internal capsule, the location of the infarct was in fact within the pons.

Our case highlights that CWS can be difficult to manage and try prevent a completed stroke. In the literature there is a lack of conclusive evidence on attempting to prevent a completed stroke in CWS. Our case highlights this further, and provides a case for further research that could provide specific treatment options for CWS.

CASE PRESENTATION

A 72-year-old right-handed man presented with fluctuating neurological signs. He had a medical history of hypertension, type 2 diabetes and he was an exsmoker of 35 pack-years.

He presented with right leg weakness and numbness, which he noticed on waking in the early hours of the morning. This initial episode lasted 10 min, before returning back to normal. He woke later in the morning to discover that he was unable to mobilise. He had developed weakness and numbness affecting both the right arm and leg. He had developed mild right-sided facial droop and dysarthria. The second episode lasted for 60 min before full resolution. Nine hours later after the onset of the first episode he developed further symptoms. He developed motor and sensory symptoms affecting the right arm and leg, he was again affected by facial droop and dysarthria. At the time of arrival to our department (after his third episode) his symptoms had improved considerably, and had resolved completely back to normal.

Examination

On examination, he had an elevated blood pressure (BP) of 198/86, the rest of his observations were within normal limits. He had a normal cranial nerve examination and there was no evidence of dysarthria. There was no evidence of cortical or any cerebellar signs.

Examination of the peripheral neurological system was normal. Examination of tone and power in all four limbs was normal. He had symmetrical reflexes and bilateral down going plantars. There was no evidence of limb ataxia and examination of the sensory system in all modalities was normal. His gait was normal; his National Institutes of Health Stroke Scale (NIHSS) was scored as 0.

Investigations

His investigations revealed that full blood count, urea and electrolytes and coagulation screen were all normal.

His ECG revealed sinus rhythm.

His initial CT of the head did not reveal any acute ischaemic changes. His initial CT angiogram was normal. It revealed normal carotids and vertebrobasilar system; it did show a left vertebral artery of reduced calibre. This is due to the vessel being congenitally smaller, suggesting a non-dominant vessel. This is a normal variant. The vessel itself was patent throughout its course. There was no evidence of intravascular thrombus.

Clinical progression/management

He was started with aspirin 300 mg once daily, simvastatin 40 mg once daily and was given a dose of amlodipine 5 mg (in view of his BP).

During the course of his admission he developed further symptoms the following morning, which were similar to his initial presentation. This episode lasted for approximately 30 min, with complete resolution again. Following this episode he was started on dual antiplatelet therapy and was given a stat dose of clopidogrel 300 mg.

In the early hours of the second day of his admission he developed further symptoms which were more severe in terms of deficit and duration. At this stage he developed weakness in the right face, arm and leg with profound dysarthria. He had 0/5 power in the right upper limb and 3+/5 in the right lower limb with a right upper motor neuron seventh cranial nerve weakness. Sensory

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Clinical characteristics and outcome of the capsular warning syndrome: a multicenter study

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Background **The capsular warning syndrome is defined as recurrent transient lacunar syndromes that usually precede a capsular infarction. Several aspects regarding the clinical management are controversial. We report the clinical and radiological characteristics of a multicenter series of patients with capsular warning syndrome, as well as their functional outcome during the follow-up.**

Aims **We sought to describe the clinico-radiological spectrum of the capsular warning syndrome and to report the functional outcomes and recurrences of these patients during the follow-up.**

Methods **We conducted a multicenter study that collected clinical and radiological data from patients with capsular warning syndrome during 2003–2013. Capsular warning syndrome was defined as the succession of three or more motor or sensory-motor lacunar syndromes within a period of 72 h, with complete recovery between them. We recorded the functional outcome (favorable when Rankin scale score ≤2) and recurrences during follow-up.**

Results **Our study included 42 patients whose mean age was 66·4 ± 10 years; 71·4% of them were men. The mean number of episodes before a permanent neurological impairment occurred or before a complete recovery of symptoms was 5·1 ± 2·3. Up to 30 patients (71·2%) had an acute infarct visible on the neuroimaging (computed tomography/magnetic resonance imaging). The internal capsule was the most frequent infarct location (50%), but other locations were noted. Twelve patients (28·6%) received thrombolysis in the acute phase. A favorable outcome was observed in 39 patients (92·9%). After a mean follow-up of 35 ± 29 months, only one patient suffered a recurrent ischemic stroke.**

Conclusions **Capsular warning syndrome preceded an ischemic infarction in 71·2% of patients. In addition to the internal capsule, other locations were noted. The most effective treatment remains unclear. The functional prognosis is favorable in most patients and recurrences are rare.**

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Key words: capsular warning syndrome, ischemic stroke, stroke warning syndrome, thrombolysis, transient ischemic attack, vascular warning syndrome

Introduction

The term 'Capsular Warning Syndrome' (CWS) was first proposed in 1993 by Donnan *et al*. They described 50 patients who presented with multiple stereotyped lacunar syndromes in a short period of time, with complete recovery between episodes (1). Using computed tomography (CT), Donnan *et al*. found that 50% of these patients had an acute infarct. Most of them were located in the clinically relevant internal capsule, which led to the term 'Capsular Warning Syndrome'. With the widespread use of magnetic resonance imaging (MRI), further studies and case reports identified other ischemic locations presenting also as CWS (2–4). Some of these locations prompted new terms like 'Pontine Warning Syndrome' (PWS) (3).

Because CWS is a rare clinical syndrome, there have not been many large studies. A prospective population-based study reported an incidence of only 1·5% among patients with transient ischemic attacks (TIA) (5). Thus, several aspects regarding the clinical management of these patients remain controversial. Additionally, whether the outcome of CWS is modified by thrombolysis is uncertain, and few studies have reported the short-term clinical outcomes of these patients who were treated with recombinant tissue plasminogen activator (rtPA) (4,6). Finally, there are no reports regarding long-term stroke and other vascular recurrences in CWS patients. This prompted us to conduct a multicenter study to collect data from a large series of patients.

Methods

We conducted a retrospective, multicenter study that collected clinical and radiological data from patients presenting with CWS. Four tertiary centers participated in our study. A database was designed that included clinical and radiological variables from patients diagnosed from 2003 to 2013. We defined CWS as the succession of at least three episodes of motor lacunar syndrome (MLS) or sensory-motor lacunar syndrome (SMLS) within a period of 72 h, with a complete resolution of symptoms between them. According to the Oxfordshire Classification of Stroke, we defined MLS and SMLS as a neurological deficit that affected at least two of the following locations: face, arm, or leg (7). We excluded patients who presented transient and repetitive neurological syndromes that were only sensitive and/or involved cortical structures. We also excluded patients with a previous score ≥3 in the modified Rankin Scale (mRS).