Stroke Mimics

A Series of Interactive Cases

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Foreword from Prof Michael Douglas:

The care pathways for patients experiencing an acute stroke have been revolutionised over the past three decades. Key therapeutic developments include the widespread approval of thrombolysis for acute ischaemic stroke, proven to improve outcomes, provided the agent is administered within a short time frame (typically within 4.5 hours of stroke onset), highlighting the need for prompt assessment and diagnosis of stroke. This has led to a key focus on efficient transport to a specialist centres, where urgent brain imaging (typically CT) provides important diagnostic information (particularly to exclude intracranial haemorrhage), with imaging interpreted by experienced clinicians so that thrombolysis can be administered in a safe and timely manner.

These patients are typically first evaluated in the acute setting, whereupon a significant proportion (around 1 in 5) are subsequently diagnosed with a range of alternative (non-stroke) diagnoses (often termed “stroke mimics”). These include presentations resulting from seizures, structural brain lesions (such as brain tumours), systemic infections or metabolic disturbance. Stroke likelihood is increased in the context of certain clinical features (visual field deficits for example) or cardiovascular risk factors (raised diastolic blood pressure and atrial fibrillation for example), but there remains a significant risk of misdiagnosis in this group of patients, estimated to be as high as 28%, particularly in situations where an neurological opinion is not available at the time of admission (Khawar 2025).

Although a stroke may have been excluded in a patient presenting with a stroke mimic, they remain unwell and the underlying condition needs prompt recognition and management. The publication of this interactive case series of 36 stroke mimics, encountered in the clinical practice of my experienced colleagues should therefore present a timely and important educational resource for both medical and non-medical practitioners who are likely to be encounter these conditions in their daily work.

Michael R Douglas, Consultant Neurologist, Dudley Group NHS Foundation Trust, January 2026.

Khawar DO et al. Stroke mimics at 30 years: where we have been, where we are now and where we are going. Stroke 56 (4): 1061-1068.

Preface from the Authors:

Dr Abdul Salam FRCP Consultant Physician in Stroke and General Internal Medicine
Dudley Group of Hospitals NHS FT,
Honorary Senior Clinical Lecturer at the University of Birmingham

As a Consultant Physician in Stroke and General Medicine at the Dudley Group of Hospitals NHS Foundation Trust, my clinical work has repeatedly shown how challenging it can be to distinguish true strokes from conditions that closely mimic them. My training, including CCTs in General Internal Medicine, Geriatric Medicine, and Stroke Medicine from UCL and the National Hospital for Neurology and Neurosurgery, Queen Square, has reinforced the value of careful assessment and diagnostic accuracy.

In addition to my clinical role, I serve as an Honorary Senior Clinical Lecturer at the University of Birmingham, have authored several peer-reviewed papers, and act as Editor-in-Chief of the *Journal of Case Reports for Medical Students and Trainees* (www.jcrmst.com).

This book brings together more than a decade of noteworthy stroke mimics encountered at the Dudley Group NHS Foundation Trust. Each case illustrates key learning points and aims to support clinicians in refining their diagnostic approach to acute neurological presentations.

Dr Shams Ud Duja FRCP Consultant Physician and Geriatrician

Honorary Associate Professor of Medicine University of Birmingham,

Dr Shams Ud Duja is a Consultant Physician and Geriatrician, and the Clinical Director for Geriatrics and Stroke Medicine. He is a Fellow of the Royal College of Physicians, an international PACES examiner, and a college representative on Consultant Appointments Committees. He is honorary Associate Professor of Medicine at University of Birmingham Medical School. He serves as Principal Investigator for multiple national research projects and has published many papers and presented both nationally and internationally. Dr Duja has held several leadership roles, including Treasurer of the West Midlands British Geriatrics Society and Chair of the West Midlands Parkinson's Excellence Network.

This book is the outcome of our efforts of collecting and putting together real cases to improve the knowledge of stroke and stroke mimics.

Dr Muqeet Enver Internal Medicine Trainee, Dudley Group of Hospitals NHS FT

Dr Muqeet Enver is an Internal Medicine Trainee working in the West Midlands, UK. He graduated with an MBChB in Medicine from the University of Leicester and holds an MSc in Cognitive Neuroscience from Aston University, reflecting a strong academic interest in the interface between brain function and clinical neurology. He has completed the MRCP(UK) examinations and is currently undertaking a Postgraduate Certificate in Medical Education. Dr Enver has

a developing specialist interest in neurology, with particular focus on stroke and stroke mimics, and plans to pursue higher specialty training in neurology.

Dr Zerlene Lim Consultant Radiologist, Dudley Group of Hospitals NHS FT

Dr Zerlene Lim is currently a radiologist at Dudley Group and UHB NHS Trusts. She completed her medical and radiology training in Sydney, Australia, and her fellowship training in Canada and the UK. She is the author and contributor of several papers predominantly in body imaging and musculoskeletal radiology.

This is a good review of stroke mimics, with relevant clinical and radiological information.

Acknowledgements:

We are extremely grateful to several colleagues, without whose generosity and expertise the development of this book would not have been possible.

We are grateful to Consultant Stroke Physicians Dr Ashim Banerjee, Dr Ragu Durairajan and Dr Snigdhendhu Mandal for their careful review of the clinical cases and for ensuring clinical accuracy and consistency. We would also wish to acknowledge the contributions of resident doctors and medical students: Dr Samaah Fathima, Asma Fathima, Dr Lousie Pollard, Dr Sadia Faisal, Dr Bindu Kesarmal and Dr Urooj Saeed who assisted with the preparation, review, and proofreading of case material.

All contributors provided input limited to case preparation, proofreading, and verification of clinical accuracy. Responsibility for the final content, interpretation, and any remaining errors rests solely with the authors.

All individuals acknowledged here reviewed this section and approved inclusion.

Contents

STROKE MIMICS: 36 CASES

Case 1

A 46-year-old male presented to the emergency department with recurrent episodes of right sided weakness, right facial paraesthesia and headaches. On further enquiry, he was found to have a family history of suspected migraines with aura as well as strokes. The patient was a non-smoker and had no other risk factors. A CT head scan (Image 1) was performed, followed by an MRI scan of the brain (Image 2).

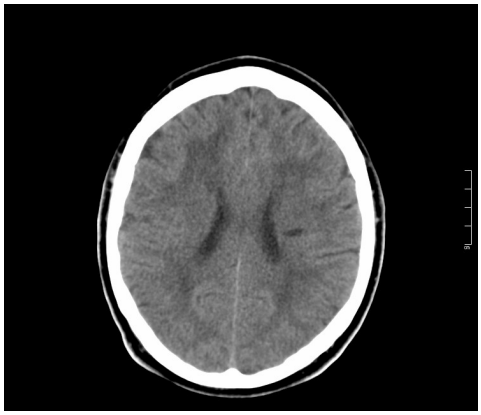

Image 1: CT Head

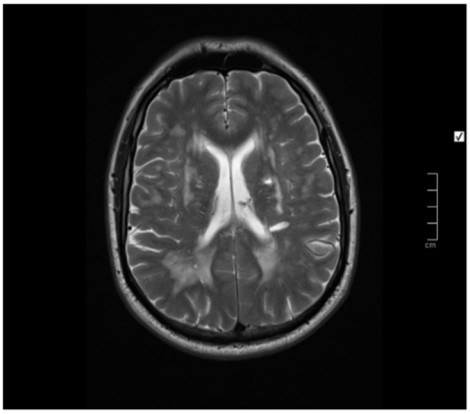

Image 2: MRI Head

Question 1: What are the positive CT and MRI scan findings?

Question 2: What other investigations would you recommend to confirm the diagnosis?

Question 3: What is the diagnosis in this case?

Question 4: Based on the diagnosis, what should the management plan be for this patient?

Answer 1:

(a) The CT head shows extensive small vessel ischaemic changes throughout the deep white matter of both cerebral hemispheres. There is a basal ganglia infarct on the left and a previous infarct in the left corona radiata.

(b) The MRI head shows symmetrical abnormal signal throughout the cerebral white matter bilaterally, particularly in the occipital lobes. Differential diagnosis of these periventricular hyperintense plaque-like lesions would include multiple sclerosis, cerebral autosomal dominant arteriopathy with subcortical infarcts (CADASIL), leukoencephalopathy and vasculitis.

Answer 2: Genetic analysis for the NOTCH3 mutation on chromosome 19 would confirm a diagnosis of CADASIL. Misfolding of the NOTCH3 extracellular domain (N3ECD) protein associated with this gene leads to aggregations in the walls of small perforating arteries, resulting in progressive occlusion[1].

Answer 3: CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy)

Answer 4: There is no specific treatment for CADASIL other than managing risk factors.

Ref

1. Chabriat H, Vahedi K, Iba-Zizen MT et al. Clinical Spectrum of CADASIL: a study of 7 families. CADASIL. Lancet 1995;346:934

Diagnosis: Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)

Case 2

A 59-year-old patient was admitted to the stroke unit with a history of acute onset right sided weakness with a score of 2/5 on the MRC Muscle Strength Scale, as well as progressively worsening expressive dysphasia. A CT head scan was done on admission (Images 1 and 2).

His past medical history included a significant head injury sustained 40 years ago. At the time, this injury had not required any neurosurgical intervention. Upon further questioning, he also reported that he had had a prolonged episode of sneezing 2 days before admission.

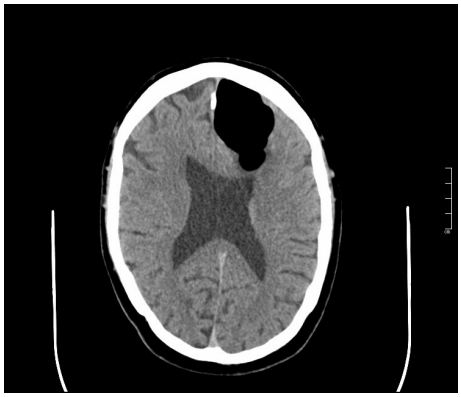

Image 1 CT Head

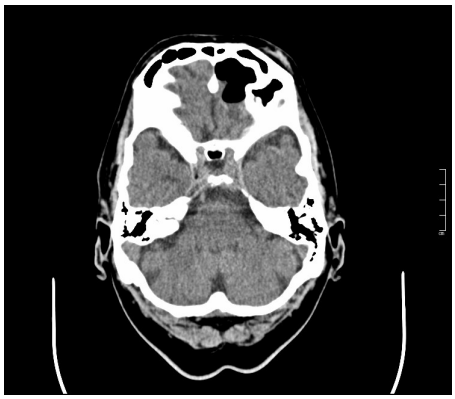

Image 2 CT head

Question 1: Describe the findings seen in the CT head scan.

Question 2: Based on these findings, what is the most likely diagnosis?

Question 3: What are the possible causes for this diagnosis, and what are the major complications which can occur secondary to this?

Question 4: What should this patient's management plan be?

Answer 1: The initial CT head scan shows a large amount of air within the left frontal lobe with associated mass effect on the frontal parenchyma and effacement of the left lateral ventricular frontal horn.

Answer 2: The diagnosis in this case is a spontaneous left frontal pneumoencephalocele.

Answer 3: Pneumoencephalocele can occur following craniofacial trauma and post neurosurgical procedures. It rarely occurs spontaneously. If there is a valve-like mechanism for air entry into the skull, there tension pneumocephalus may occur.

Answer 4: The patient was transferred to the neurosurgical unit. Aspiration of the air pocket in the left frontal lobe was carried out using a left frontal twist drill. Significant improvement in expressive dysphasia was seen, and right-sided weakness improved to a score of 4/5 on the MRC Muscle Strength Scale.

Ref:

1. Schirmer CM[1], Heilman CB, Bhardwaj A. Pneumocephalus: Case illustrations and review
Neurocrit Care. 2010 Aug;13(1):152-8. doi: 10.1007/s12028-010-9363-0

Diagnosis: Pneumocranium

Case 3

A 35-year-old female was admitted with a 5-week history of headaches which were worse in the morning. She also had associated neck stiffness, nausea and photophobia. She developed left sided weakness and altered speech and was admitted to the stroke ward.

Past medical history included IV drug abuse. Her initial blood test showed raised white blood cell count, raised neutrophil count and raised CRP. She also had a few spikes in temperature.

A CT head scan was carried out (Images 1 and 2)

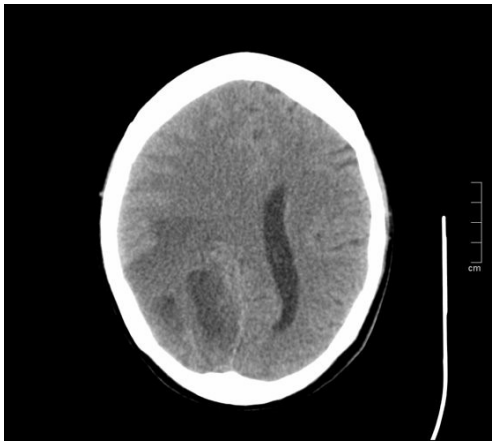

Image 1: CT head

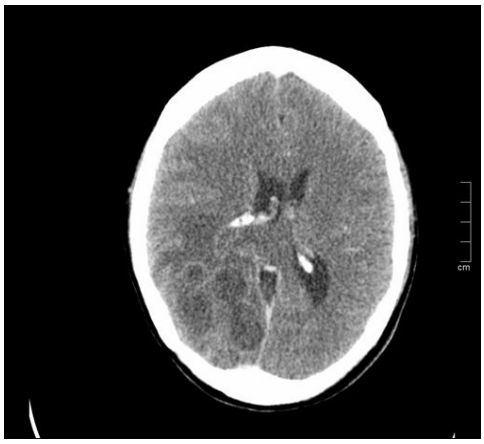

Image 2: CT head

Question 1: Describe 3 positive findings which are visible from the CT head scan.

Question 2: What are the differential diagnoses for these findings?

Question 3: What is the diagnosis?

Question 4: What should the management plan be for this patient?

Answer 1:

A. There is a large 4 cm x2cm thick-walled ring enhancing lesion in the right parieto-occipital temporal lobe

B. There are several septations with in the lesion.

C. There is a local mass effect with effacement of the right lateral ventricle, overlying sulci and midline shift.

Answer 2: Differential diagnoses would be a primary malignancy or solitary metastases in the brain

Answer 3: The findings are consistent with cerebral abscess

Answer 4: Start this patient on broad spectrum antibiotics which would eventually need changing based on the blood culture results and sensitivity. This patient would also warrant urgent neurosurgical input for surgical intervention.

Diagnosis: Cerebral Abscess

Case 4

A 39-year-old lady was admitted with left sided weakness and altered speech. She worked as a sewing machine mechanist. There was a significant family history of strokes at a young age. She did not have any typical risk factors which would have predisposed her to early cerebrovascular accident. She had multiple investigations including a carotid duplex of the neck, an echocardiogram, transoesophageal ECHO and a 48- hour Holter monitor. There were no significant abnormalities detected in these.

To identify other causes for the stroke, she was screened for autoantibodies, thrombophilia and vasculitis. These also came back as negative. She had a CT head following her initial presentation (Image 1). Following this, Magnetic Resonance Angiogram of the head (MRA Head) (Image 2) and a Digital Subtraction Angiogram (Image 3) were also performed.

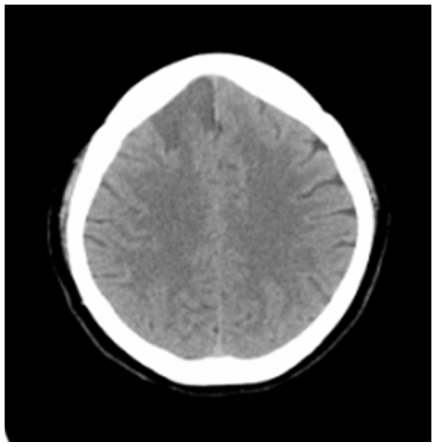

Image 1: CT Head

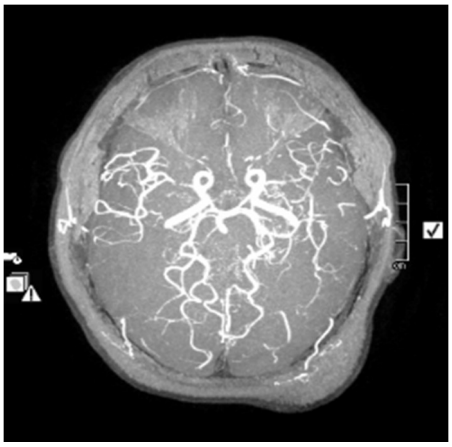

Image 2: MRA Head

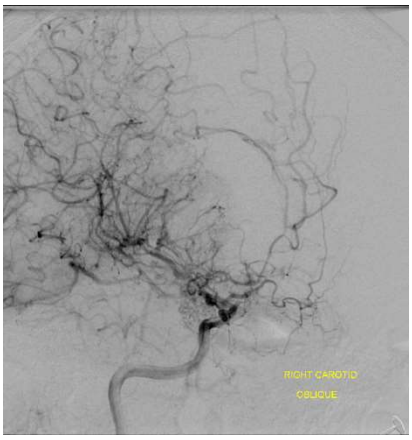

Image 3: DSA Head

Question 1: What are the CT head findings?

Question 2: What are the MRA findings?

Question 3: What is the probable diagnosis?

Question 4: What is the classical finding of this disease on the (DSA) carotid angiogram?

Question 5: What are the treatment options for this disease?

Answer 1: The CT head demonstrates a right frontal lobe infarct.

Answer 2: On the MR angiogram, middle cerebral arteries and anterior cerebral arteries bilaterally demonstrate diffuse luminal narrowing. The internal carotid arteries are of a normal calibre.

Answer 3: The diagnosis is Moyamoya disease. This is a rare and progressive cerebrovascular disorder in which the internal carotid arteries and their main branches gradually narrow and can eventually become blocked. The arteries typically involved are the internal carotid arteries, the anterior cerebral artery or the middle cerebral artery. The cause for this disease is unknown.

Answer 4: A carotid angiogram will demonstrate a tight middle cerebral artery stenosis and new vessel formation, classically described as a "puff of smoke" as shown in image 3 DSA head.

Answer 5: There is no specific treatment for Moyamoya disease. Extracranial to intracranial (EC-IC) bypass is recommended by some authorities to improve collateral circulation. The role of antithrombotic agents is uncertain, especially after the formation of a new vessel, due to the risk of haemorrhage. Antihypertensives are recommended to reduce the risk of bleeding.

Diagnosis: Moyamoya Disease

Case 5

42-year-old gentleman was brought to the Emergency department after he was found unresponsive. The history taken from the family revealed that he had gone to bed at 3pm in the afternoon and his daughter was unable to wake him up in the evening. The family reported no significant past medical history, and he did not take any regular medications. He worked in a warehouse and did not smoke, drink alcohol or use any recreational drugs.

On initial examination, his score on the Glasgow Coma Scale (GCS) was 6/15 (E1, V2 and M3) and he was able to maintain his airway. He had bilateral pinpoint pupils which were unreactive to light. His plantars were down-going with no obvious wasting of the upper/lower limb muscles. His chest was clear with normal heart sounds, and the abdomen was soft and non-tender.

His blood tests revealed normal renal function, liver function and bone profile. He had a mildly raised Creatine Kinase 281 with a normal lactate and CRP. Paracetamol levels in the blood were insignificant, and he had a normal full blood count. His toxicology screen was normal. An initial CT scan of the head did not reveal any acute intracranial pathology.

He was started on treatment for encephalitis and meningitis with strong IV antibiotics (ceftriaxone) and IV antivirals (acyclovir). He had an echocardiogram with saline bubble contrast which demonstrated a patent foramen ovale (PFO).

During his admission he was found to be in an altered state of consciousness and well as fluctuating mental states with periods of

confusion when he was awake. The images from his MRI head scan are seen below.

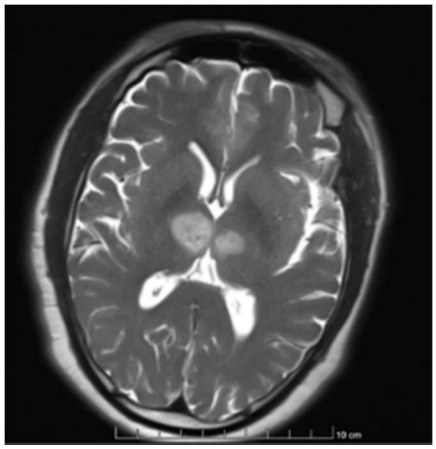

Image 1 MRI Head

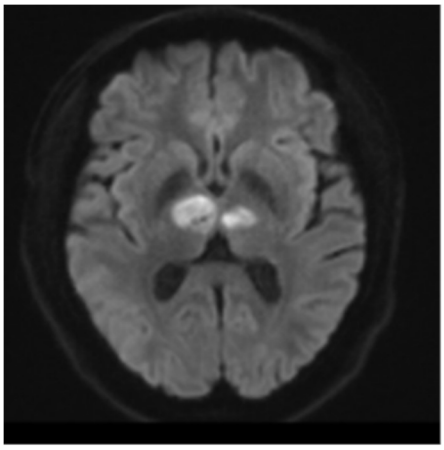

Image 2 MRI Head

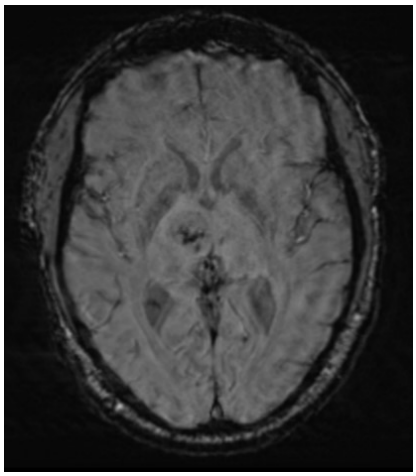

Image 3 MRI Head

Question 1: What are the findings in the MRI head?

Question 2: What diagnosis are these findings associated with?

Question 3: What other common clinical findings are associated with this diagnosis?

Question 4: What should the management plan be for this patient?

Answer 1: The MRI head shows bilateral thalamic restriction of diffusion which is consistent with infarcts. Blooming artefact in both thalami are consistent with micro-haemorrhages.

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Answer 2: Bilateral thalamic infarcts are associated with an Artery of Percheron occlusion. The Artery of Percheron is an anatomical variation which supplies both thalami and sometimes the midbrain from a single trunk.

Answer 3: The common clinical findings associated with an artery of Percheron stroke are: a sudden loss of consciousness, altered mental state, coma, confusion and memory problems.

Answer 4: The patient was referred to an adult congenital heart disease specialist for consideration of PFO closure. He was started on aspirin and had a vasculitis screening which was normal. He was admitted to a neuro-rehabilitation bed at the nearest rehabilitation facility for further management.

Diagnosis: Artery of Percheron related infarct with Stroke mimic

Case 6

A 56-year-old lady was referred by her GP to the Emergency Department due to an unsteady gait and blurred vision. The instability in her gait had come on suddenly 4 weeks prior to her admission. Initially, this instability was transient in nature but became constant in the 2 weeks leading up to her admission, requiring her to walk with support. She described the unsteadiness as being worse when she changed her posture and when walking. She also reported associated dizziness and bilateral blurring of her vision over the same period. She denied any headache, loss of vision, loss of consciousness, facial weakness, muscle weakness or changes in her speech. Furthermore, she did not report new memory problems, cognitive decline, seizures or loss of consciousness.

Her past medical history included hypertension, for which she was on doxazosin, losartan and amlodipine. There was no significant family history of neurological disorders other than a maternal aunt with Charcot-Marie-Tooth disease.

Neurological examination found no ophthalmoplegia or cranial nerve abnormalities. Her GCS score was 15/15 and she had no bulbar weakness. She had preserved power (5/5) in all 4 limbs and preserved reflexes (with the exception of the ankle jerk reflex, which was present bilaterally with reinforcement). She had normal vibration and position sense with no evidence of myoclonus. There was mild ataxia in heel-to-shin and face-to-nose face testing on the right side with no associated dysdiadochokinesia.

She also complained of diplopia which was worse on vertical gaze bilaterally.

Her initial blood tests revealed no significant abnormalities. A CT head scan showed no new intracranial abnormalities.

Patient had an urgent MRI head followed by lumbar puncture and EEG. The MRI images are as seen below.

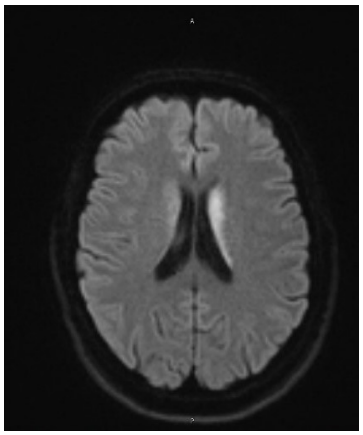


Image 1:MRI Head

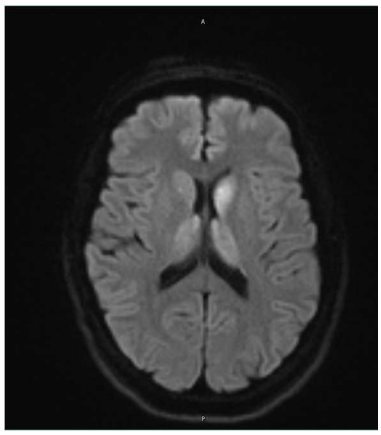


Image 2: MRI Head

Question 1: What are the positive findings seen on the MRI head scan?

Question 2: What differentials should be considered in light of these MRI findings, and what is the most likely diagnosis?

Question 3: What specific test would you request on the lumbar puncture sample?

Question 4: What should the management plan be for this patient?

Answer 1: The MRI scan shows restricted diffusion in both thalami and left caudate nucleus. There is also restricted diffusion in the right caudate nucleus, but to a lesser extent.

Answer 2: The MRI findings are most consistent with a diagnosis of Creutzfeldt-Jacob Disease (CJD). Other differentials to consider in this case are recent seizure activity and previous hypoxic or metabolic encephalopathy.

Answer 3: If considering an underlying diagnosis of CJD, a CSF sample must be sent for a RT-QuIC test (Real-Time Quaking-Induced Conversion). This test involves adding a patient sample (usually CSF but can use nasal brushing) to a solution with normal prion protein substrate. If abnormal prions are present, they "seed" the misfolding of the normal prion proteins. The misfolding process is amplified and detected using a fluorescent dye that binds to the aggregated proteins.

Answer 4: Unfortunately, there is no current cure for CJD. Management involves early involvement of palliative care teams and CJD clinical nurses who can offer support. Patients can be redirected to the CJD support network which is a charity that offers emotional and practical support to those diagnosed with CJD or other prion diseases.

Diagnosis: Creutzfeldt-Jacob Disease

Case 7

A 53-year-old female presented to the emergency department following a collapse and inability to speak. Her daughter reported that she had been alert and talking as normal 30 minutes prior to this episode. She had found the patient on the floor, unable to speak and with bleeding around her left eye.

There was no significant past medical history and the patient was not on any regular medications. She had recently recovered from a COVID-19 infection approximately 3 weeks before the current presentation. There was no family history of strokes or other neurological disorders.

On initial examination, she was found to be aphasic. She was alert with both pupils equal and reactive to light. She had no visual deficit, no nystagmus, no facial droop, no upper/lower limb weakness or sensory loss. There was an evident left eye subconjunctival and periorbital haemorrhage.

Her initial blood tests revealed no significant abnormalities in the renal, bone or liver function tests and a normal full blood count. She had raised triglyceride levels (2.5 mmol/L) but normal cholesterol levels. Her ESR levels were raised (112) and she had a weakly positive ANA titre. A COVID-19 swab done at the time of presentation was positive. The initial CT head did not show any acute intracranial haemorrhage or acute infarct.

The next day, she was re-examined and scored 12/15 on the GCS. She also continued to present with aphasia. She had reduced power on the right side (0/5 in both upper and lower limbs on the right side) and upward going plantars on the right. She also demonstrated right-sided neglect and audible

upper airway noises. A repeat urgent CT head scan was performed in view of these findings, as well as a chest x ray (Image 3). The images for this can be found below (Images 1 and 2).

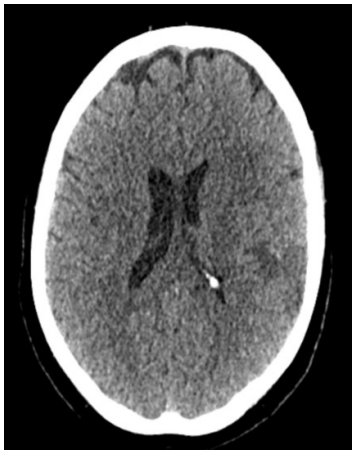


Image 1: CT Head

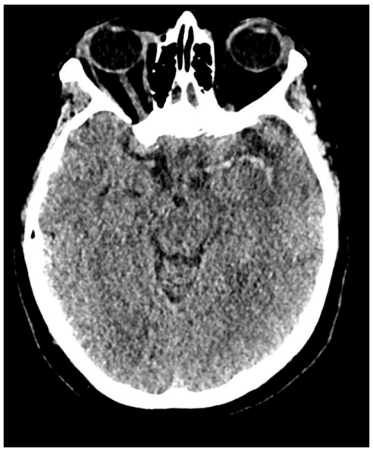


Image 2: CT head

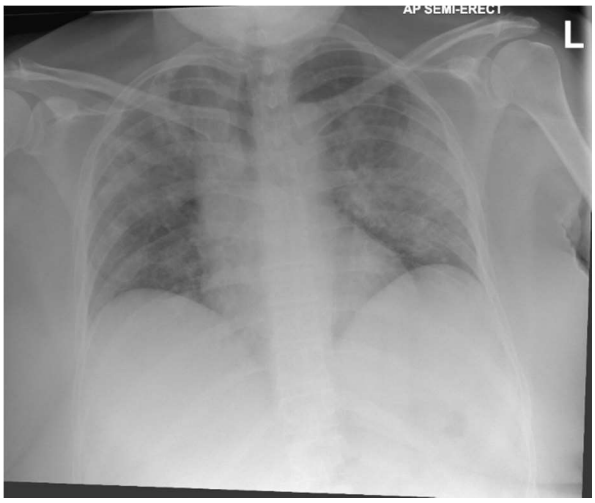


Image 3: Chest X Ray

Question 1: What are the findings visible on the CXR given this patient's recent exposure to COVID-19?

Question 2: What are the findings from the repeat CT head scan?

Question 3: Given the above findings, what is the likely diagnosis for this patient?

Question 4: What would be the long-term management plan for this patient?

Answer 1: Widespread airspace opacity is seen in both lungs. Given the positive COVID-19 PCR result, this is the likely cause of this finding. There is no pleural effusion.

Answer 2: The CT head scan demonstrates a wedge-shaped area of hypoattenuation within the left parietal lobe with some patchy attenuation change in the ipsilateral temporal lobe.

Answer 3: The likely diagnosis for this patient was a significant large left MCA territory infarct secondary to COVID associated vasculitis.

Answer 4: Due to significant periorbital bleeding, the patient was not considered for thrombolysis. She was managed medically with treatment for COVID –19 and was started on anti-platelet therapy. She was referred to a rehabilitation facility to receive neurological rehab following a large stroke.

Diagnosis: COVID Vasculitis

Case 8

27-year-old male who was diagnosed with a rare lung condition called plastic bronchitis. His other past medical history included asthma, ADHD and high BMI. He underwent iatrogenic injection of glue into the venous system following an experimental treatment for plastic bronchitis. However, the patient did not wake up following this treatment and the team were unable to extubate him.

A CT head scan (Image 1) was performed which revealed infarcts in multiple territories and some brainstem hyper densities. He was subsequently transferred to the nearest hospital for thrombectomy. He underwent a successful removal of glue/thrombus from the left MCA territory under the interventional radiology team. An Intracranial Pressure (ICP) bolt was inserted which was subsequently removed 10 days post-thrombectomy. He was subsequently transferred to his nearest District General Hospital (DGH) for further monitoring and rehab.

Interestingly, a CT Abdomen and Pelvis with contrast revealed infarcts at the upper pole of the left kidney and multiple wedge-shaped splenic infarcts secondary to glue embolization. He showed good progress during rehabilitation and was subsequently discharged from the ward.

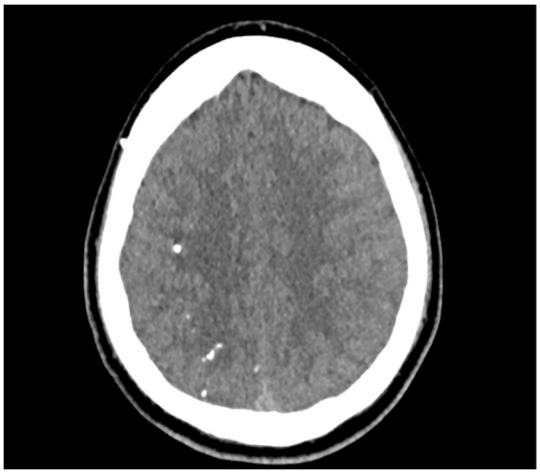


Image 1: CT head

Question 1: What are the findings from the CT head scan?

Question 2: What is plastic bronchitis?

Question 3: What is an ICP bolt?

Question 4: What do you think is the best discharge destination for this patient before returning to his home?

Answer 1: Punctate hyperdense foci seen at the grey-white matter junction of the posterior right frontal, parietal and occipital lobes consistent with glue embolus.

Answer 2: Plastic bronchitis is a rare lung condition where thick, rubbery mucus casts form in the bronchi and block airflow. This can lead to severe breathing difficulty and even respiratory failure. Plastic bronchitis is diagnosed using bronchoscopy and removal of these casts to confirm diagnosis.

Answer 3: An ICP bolt is a device used to monitor intracranial pressure. It is crucial in patients after mechanical thrombectomy, subarachnoid haemorrhage or traumatic brain injury to monitor for brain swelling, haemorrhagic transformation or increased pressure that could potentially worsen the clinical outcomes.

Answer 4: The best destination for this patient before discharge home would be a neuro-rehabilitation centre where he could undergo daily physiotherapy and assistance from occupational therapists as well as speech/language therapists in order to achieve the best outcomes post-admission.

Diagnosis: Glue Embolus

Case 9

A 69-year-old lady who presented to the Emergency department with a 4-day history of intermittently "finding it difficult to find the words she wanted to say". She also described feeling confused and disorientated. She denied any cough, shortness of breath, chest pain, abdominal pain, headache, visual disturbance or limb weakness. She had a past medical history of hypothyroidism, sciatica and osteoarthritis. She was on Levothyroxine 75 microgram daily. She was non-smoker and there was no history of excessive alcohol intake.

On arrival, her blood pressure was 126/68, pulse rate 87/minute, temp 37.4 °C, oxygen saturations 94% on air, and respiratory rate of 18/minute. Her Glasgow Coma Scale (GCS) was 14/15 (E4, V4, M6). An ejection systolic murmur was heard on the aortic area radiating to both carotids, otherwise her cardiac, respiratory and gastro-intestinal systems examination was unremarkable. Her cranial nerves were intact, and motor and sensory nervous system examination was unremarkable. She scored 1 out of 4 on the abbreviated mental test (AMT). Her blood tests showed slightly low sodium (127 mmol/l), otherwise her renal, hepatic, bone profiles were all normal. Her full blood count, glucose, INR and C-reactive protein were also normal. Her ECG and blood gas analysis was normal.

A consultant physician reviewed the patient and made a diagnosis of stroke. She was given Aspirin 300mg stat, stroke team review and urgent CT head were requested. The stroke team noticed severe expressive dysphasia (she was unable to describe a pen, cup, water or book). Patient went for CT head

and had a generalized tonic-clonic seizure before her scan and one immediately after her scan (both seizures were terminated by intravenous benzodiazepines). The images from her CT scan are as shown below.

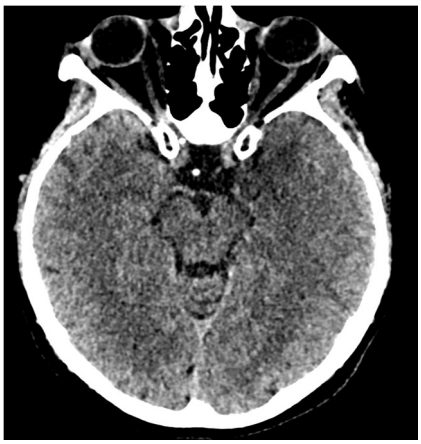


Image 1: CT Head

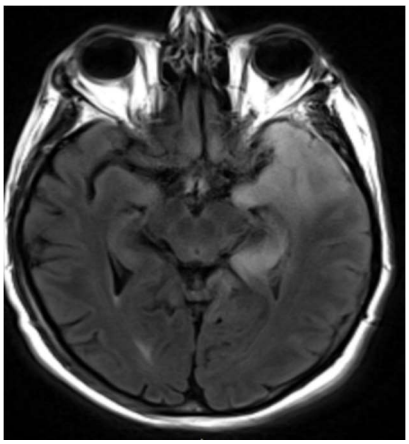


Image 2: MRI Head

Question 1: What are CT scan and MRI findings?

Question 2: What is your diagnosis?

Question 3: What other investigations will you do?

Question 4: How do you think this patient was managed?

Answer 1: Asymmetrical low density in the left temporal lobe on CT. Corresponding signal abnormality and oedema in the left temporal lobe on MRI.

Answer 2: The likely diagnosis in this case is acute encephalitis. Other differentials could include an infarct and post seizure changes.

Answer 3: Other investigations to consider in this patient would be a lumbar puncture and EEG.

Answer 4: Patient was transferred to ITU where she was intubated and ventilated. She was started on acyclovir 600mg TDS, phenytoin 100mg TDS, amoxicillin 2g QDS, cefotaxime 2g QDS and levetiracetam 500mg BD. Lumbar puncture was performed. Lumbar puncture showed CSF protein: 0.80 (high), CSF glucose: 4.1 (high), NMDA Receptor Antibodies-negative, HSV1- detected by PCR while HSV2- not detected by PCR. EEG showed frequent runs of sharp activity predominantly over left frontal and temporal regions. Findings supported mild to moderate encephalopathy. A diagnosis of Herpes Simplex Encephalitis was made, and Acyclovir was continued for 21 days. Patient made remarkable recovery and was left with mild expressive dysphasia.

Diagnosis: HSV Encephalitis

Case 10

A 78-year-old male was brought to the emergency department following a fall in the street. He was known to have atrial fibrillation. A drug history was not available at the time of admission. He was taking three different types of tablets but could not tell the team the names of these medications. He was an ex-smoker, consumed alcohol occasionally and was a retired builder and lived with his wife.

On arrival, his pulse was 82bpm, blood pressure was 145/87 and oxygen saturation was 99% on air. He appeared clinically well hydrated. He scored 14/15 on Glasgow Coma Scale (GCS). Neurological and other systemic examinations (respiratory, cardiac and gastro-intestinal) were normal, except for rate controlled atrial fibrillation. His sodium was borderline low, and the patient had chronic kidney disease stage 3. His full blood count, liver function and bone profile were normal. His urinalysis was negative. He had an urgent CT scan of his head.

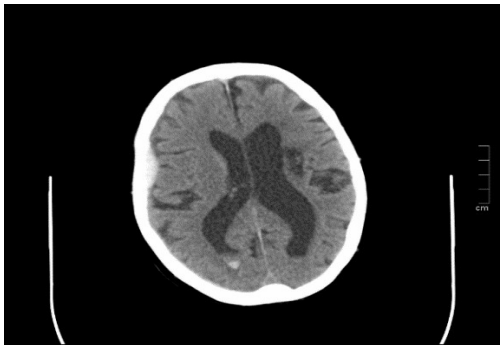


Image 1: CT Head

Question 1: What are the positive findings on the CT scan?

Question 2: What would be the best way to manage this patient?

Answer 1: Small extra dural haematoma in right parietal area. Furthermore, there is blood in both lateral ventricles.

Answer 2: Start neuro-observation every 15 minutes. Obtain drug history (as patient is known AF, he may well be on anti-coagulation). If he is on anti-coagulation, check INR and reverse the anti-coagulation. Maintain intra-venous access and perform a swallowing assessment. If the GCS deteriorates, get a repeat CT scan and speak to the neurosurgery team. If GCS maintains or improves, continue neuro-observations hourly for 12 hours and then 2-hourly. If no deterioration in 24 hours, observe patient for couple of days in hospital before discharge.

Diagnosis: Extradural Bleed

Case 11

A 63-year-old Caucasian male was referred to the movement disorder clinic with a slow, shuffling gait and a provisional diagnosis of Parkinson's disease. He attended in a wheelchair, accompanied by his son. He gave a history of gradual deterioration over 5 to 6 months. He described losing his balance often, walking with the aid of furniture inside his house and had stopped going out over the last couple of months. He also reported occasional freezing and stated no problems using his arms. He could feed and dress himself, use a knife and fork and had no difficulty in doing his buttons or shoelaces. Furthermore, he was able to swallow his food without any difficulty. He denied any dribbling of saliva. He was sleeping well and was able to turn himself in the bed. On further questioning, he gave a history of occasional urinary incontinence and forgetfulness.

His past medical history included hypertension, abdominal aortic aneurysm repair and osteoarthritis. He was taking furosemide, ramipril, lansoprazole, quinine sulphate and simvastatin. He was a non-smoker and consumed alcohol mainly on the weekend. His facial examination was unremarkable. There was no tremor, rigidity or bradykinesia of upper limbs. There were no tremors but moderate rigidity and bradykinesia of both legs. He could not stand properly and had very poor balance to walk. He was shuffling and had lost his confidence. There was no sensory-motor deficit and cerebellar examination was normal. He scored 24/30 on Mini Mental State Examination (MMSE). His CT scan and MRI scan images are as shown below.

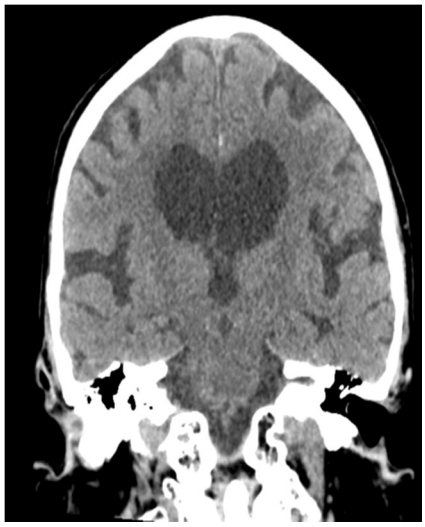


Image 1: CT Head

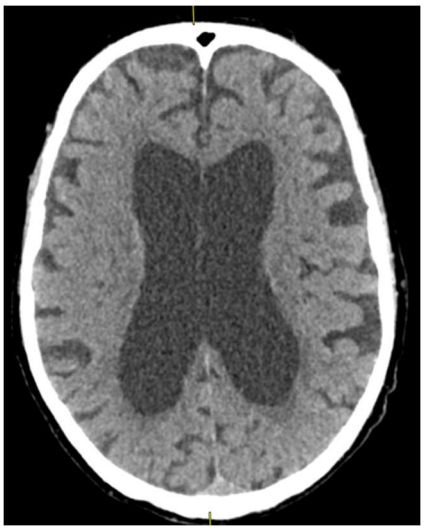


Image 2: CT Head

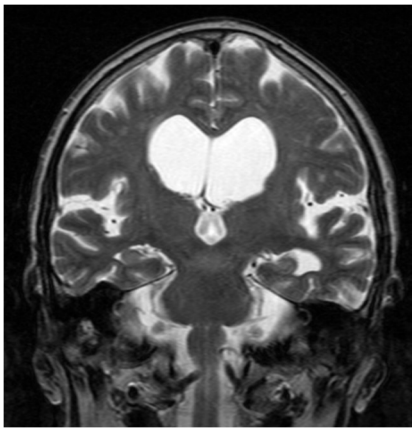


Image 3: MRI Head

Question 1: Describe the main finding seen in both CT and MRI scans?

Question 2: What other investigations, if any, would you like to do?

Question 3: What is the most likely diagnosis in this patient?

Question 4: What would be an appropriate management plan for this patient?

Answer 1: There is disproportionate enlargement of the lateral and third ventricles relative to the overall degree of cerebral atrophy.

Answer 2: Large-volume lumbar puncture (spinal or CSF tap test)- CSF pressure will be normal or intermittently raised. Furthermore, the effect of the lumbar puncture is assessed looking at improvement of the patient's symptoms which can last for a period of days to weeks. The value of this test is limited in diagnosing normal pressure hydrocephalus (NPH) but may be useful in narrowing the differential diagnosis. If it is positive and symptoms do improve, it can be used as a predictor of positive operative outcome (see below).
Intraventricular monitoring - in NPH, this may show a particular pattern characterised by beta waves. Lumbar infusion test (intrathecal infusion test) where the CSF absorptive capacity is tested with a fluid challenge. An abnormal, sustained rise in CSF suggests NPH.

Answer 3: Normal Pressure Hydrocephalus (diagnosis is based on the clinical triad in the absence of papilloedema, backed by neuroimaging)

Answer 4:
Medical: Medical treatment of NPH includes acetazolamide and repeated lumbar puncture. These methods are rarely successful long-term and are usually used as temporary deferment measures or in patients who are too ill for surgery.
Surgical: The mainstay of treatment is surgical insertion of a CSF shunt. This could be to the peritoneum, the right atrium or, more recently, via

external lumbar drainage. Selection of patients for surgery is important, as exposing patients to shunt-related complications such as mechanical failure or infection is unwarranted, unless a good clinical outcome is expected. Various parameters are used to predict which patients will benefit from surgery, but there is insufficient evidence for their efficacy. Features suggesting a good outcome include:

- Presence of a clearly identified aetiology.
- Predominant gait difficulties with mild cognitive impairment.
- Normal-sized or occluded Sylvian fissures and cortical sulci on CT or MRI.
- Absent or moderate white matter lesions on MRI.

Insertion of a ventriculoperitoneal shunt is the first-line procedure, with ventriculo-atrial shunting being used as an alternative.

Further reading: Dalvi A et al, Normal Pressure Hydrocephalus, Medscape, Oct 2010

Diagnosis: Normal Pressure Hydrocephalus

Case 12

42-year-old female who was brought to the Emergency department due to being found unresponsive. Her husband found her to be staring at him, but she was unable to speak. The ambulance crew reported a low GCS and on-going unresponsiveness.

Her husband reported that she is normally fit and well but had been complaining of a headache for one week. She had also recently been more stressed due to her work. She had no significant past medical history. She worked as a teacher and did not have a significant smoking or alcohol history. Her only regular medication was the oral contraceptive pill.

The examination performed by the medical team was limited and found her GCS 8/15 (M4, V1, E3). Her blood tests at admission were as follows; sodium 137, eGFR 70, ALT 63, calcium 2.19, Hb 141 and WCC 8.10. Her ECG showed normal sinus rhythm. A CT head and CT Venogram were performed urgently

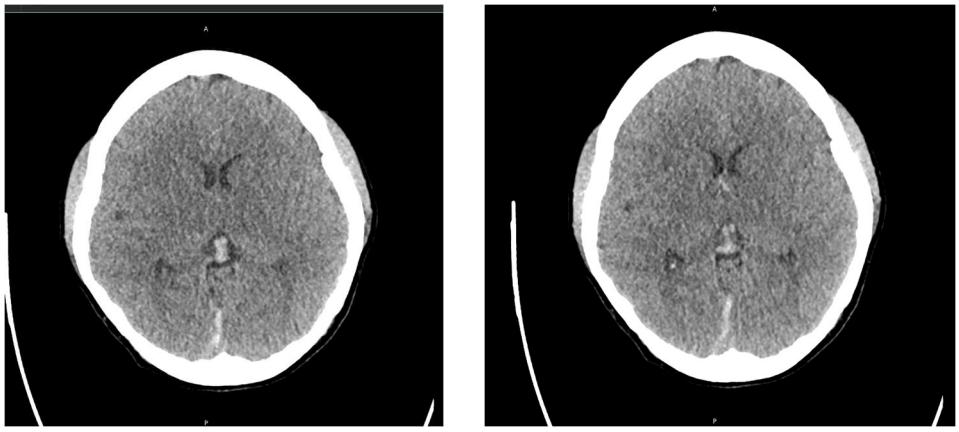


Image 1: CT Head

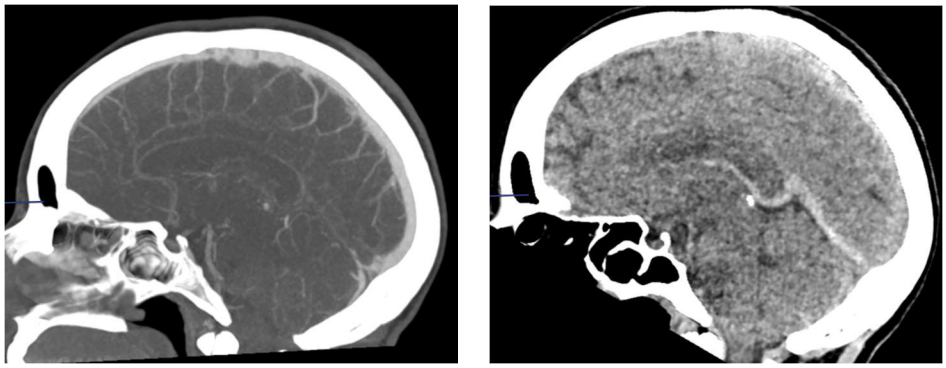


Image 2: CT venogram

Question 1: What are the findings evidenced on the CT head scan?

Question 2: What are the important findings in the images from the CT Venogram?

Question 3: What is the management for the underlying diagnosis?

Question 4: What is the prognosis for this diagnosis?

Answer 1: There is high density within the internal cerebral vein, straight sinus and extending towards the torcula.

Answer 2: The CT venogram confirms the presence of venous sinus thrombosis with occlusion of the inferior sagittal and straight sinuses extending to the torcula.

Answer 3: First line of treatment includes anticoagulation with Low Molecular Weight Heparin (LMWH) or unfractionated heparin. Patients will then be started on long-term anticoagulation in the form of warfarin or Direct Oral Anticoagulants (DOACs). The underlying cause must be addressed i.e. infection (with antibiotics), dehydration (with IV fluids) or managing provoking factors (such as oral contraceptive pill). Mechanical thrombectomy may be considered if there is clinical deterioration despite anticoagulation or extensive clot burden.

Answer 4: The prognosis is generally good with early treatment and most patients recover fully.

Diagnosis: Cerebral Venous Thrombosis

Case 13

A 43-year-old gentleman was brought into the emergency department after suffering a seizure and right sided facial droop. The ambulance crew reported reduced movements in the right upper and lower limbs. As per the ambulance notes, the patient had been complaining of worsening headaches for 2 weeks. He experienced right sided stiffness followed by shaking of the whole body which lasted for a few minutes. Since then, he had been drowsy and producing incomprehensible speech.

He had a past medical history of a cerebral abscess after which he developed seizures. He underwent surgery for this abscess after which a part of the left frontal lobe was resected. Furthermore, he had a part of his left frontal bone removed. He had developed epilepsy post procedure and so was started on regular anti-epileptics and had been seizure free for nearly a year prior to admission.

Examination by the stroke team revealed no evidence of right sided facial droop. He had decreased movements in the right upper limb, compared to the left upper limb. It was difficult to assess power as the GCS was 11 at presentation (E3, V2, M6). Blood tests showed a lactate of 5.0, CRP 3, WCC 16.70 and renal function was at baseline.

Once the patient became more alert with GCS 15, we noted that the right sided weakness had resolved, and patient had restored function in right upper and lower limbs. The lactate levels reduced to <1.0, WCC normalised and renal function stabilised.

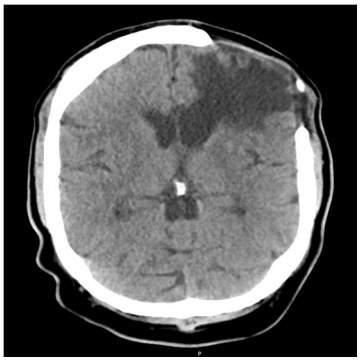


Image 1: CT Head

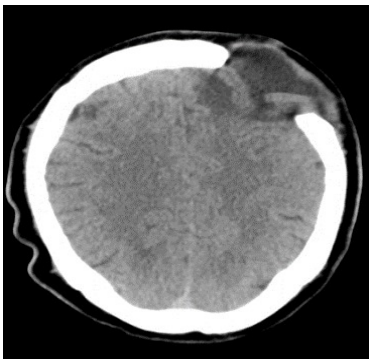


Image 2: CT Head

Question 1: What is the diagnosis for the pattern of weakness observed in this patient?

Question 2: What are the findings from the CT head scan?

Question 3: What would be the management plan for this patient?

Answer 1: This patient had a diagnosis of Todd's paresis. Todd's paresis (also called Todd's paralysis or postictal paresis) is a temporary neurological condition characterised by weakness or paralysis in a part of the body following a seizure. This can mimic a stroke and must be considered in patients presenting with weakness after a seizure. Todd's paresis usually affects one side of the body (hemiparesis), and the weakness can last from minutes to hours.

Answer 2: The CT head shows that at the site of the previous craniectomy- there is loss of cerebral parenchyma and increase in volume and herniation of the surrounding CSF and dura beyond the craniectomy defect. There is no evidence of raised ICP and so this herniation is chronic.

Answer 3: The management plan for this patient would involve admission to hospital for 24-48 hours to monitor for further seizure activity. If seizure activity was not controlled by the current anti-epileptic medications, it would be warranted to seek neurology opinion to optimise anti-epileptic medications. Neurological function should be reassessed and if returned to baseline, no further intervention would be required.

Diagnosis: Todds Paresis

Case 14

A 60-year-old retired lady was referred to the Rapid Access Transient Ischaemic Attack (TIA) Clinic by her GP due to having difficulty in holding a pen with her right hand and difficulty doing up buttons and zips. She could not straighten the fingers of her right hand. These symptoms started three months ago. Her husband reported increased salivation for the past two weeks, intermittent slurred speech and "tightening" of the calf muscles for two months.

She did not suffer from migraines and was not on any regular medication. There was no reported family history of stroke or migraines. She was being investigated for familial hypercholesterolemia. She lived in a house with her husband and was independent with her Activities of Daily Living (ADL). She did not smoke and drank alcohol socially. She did not have a low mood and was not under stress. She did complain of shortness of breath on climbing the stairs.

On examination her pulse was 74 beats/min (regular), Body Mass Index (BMI) 29.9, sitting blood pressure 155/77 mmHg and standing blood pressure was 150/77. There was a soft ejection systolic murmur best heard in the aortic area. Her speech was mildly dysarthric (score of 1 in the articulation section of NIHSS), postural tremor in the upper limbs (right greater than left), wasting of the dorsal interossei muscle of her right hand, with clawing of the fingers. The power was 4/5 in the distal muscles of the right upper limb. Reflexes were brisk throughout. There was no sensory

loss and the right plantar was up-going. Her gait was normal. Romberg's sign was negative, and she was able to perform tandem gait.

On fundoscopy the discs appeared slightly pale. She complained of pain in the anterior thigh muscles on performing straight leg raise test. Her bloods including thyroid function, creatinine kinase, B12, folate, plasma glucose and Angiotensin Converting Enzyme levels were normal. Syphilis and autoantibody screen were negative. Cholesterol 7.7 (high) and Vitamin D level 30 (suggestive of Vitamin D insufficiency). ECG showed normal sinus rhythm. Chest X Ray was reported as normal. Carotid Doppler showed 25% stenosis of the right internal carotid artery. Transthoracic echocardiogram showed mildly thickened and calcified aortic and mitral valve.

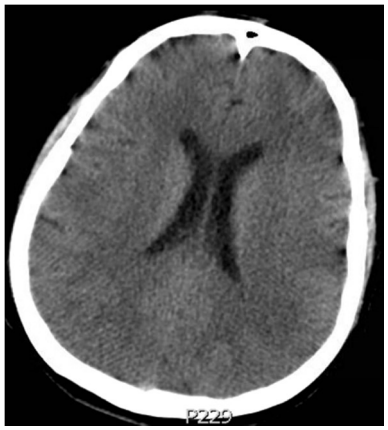


Image 1: CT Head

Question 1: What are the findings as seen in the CT head scan?

Question 2: What additional investigations would you consider, and what is the most likely differential diagnosis?

Question 3: Describe two changes that can be seen in the MRI head of patients with motor neurone disease?

Question 4: What type of motor neurone disease does this patient have?

Question 5: Name the drug that has been recommended by the National Institute of Clinical Excellence (NICE) for the treatment of motor neurone disease.

Answer 1: The CT head scan is normal with no acute changes.

Answer 2: EMG/NCS, MRI Head

Answer 3: Hyperintensity on T2 sequences in regions of the pyramidal tracts e.g. the internal capsule and the motor strip (cell bodies of the pyramidal tract are found on this gyrus). MRI head also shows hyperintensity on SWI (Susceptibility Weighted Imaging) sequence in regions where pyramidal tracts are present. This is due to the loss of upper motor neurons and deposition of iron.

Answer 4: Amyotrophic Lateral Sclerosis (ALS) due to combination of upper motor neurone signs (tightening of the calf muscles due to spasticity, brisk reflexes and up going planter) and lower motor neurone signs (wasting of the small muscles of the hand). The other types are primary lateral sclerosis (PLS) and progressive muscular atrophy (PMA).

Answer 5: The only drug licensed and approved by NICE for the treatment of MND is Riluzole which works by suppressing glutamate activity. In two large, randomised trials of patients with the ALS form of MND, Riluzole was shown to prolong tracheostomy free survival by 3–6 months.

Progress of the case: She was seen by a consultant neurologist and a diagnosis of "Likely motor neurone disease" was made. The patient and her daughter were told that her symptoms were due to motor neurone disease. Her anti- ganglioside antibodies (to exclude multifocal motor neuropathy

with conduction block) were checked and referred to the multidisciplinary Motor Neurone Disease Team in a tertiary centre. She was also referred to a community speech and language therapist for dysarthria and dysphagia management.

Review article: Diagnosis and management of motor neurone disease. BMJ 2008; 336:658

<u>Diagnosis</u>: Motor Neurone Disease

Case 15

A 82-year-old gentleman presented to the emergency department with left sided weakness. He described sitting in his garden and when he tried to stand up, he felt weak in his legs with left arm weakness and left-sided facial droop. His past medical history included CKD stage 5, hypercholesterolaemia, recent diagnosis of lung cancer, previous NSTEMI, peripheral vascular disease, Left Ventricular Systolic Dysfunction (LVSD), Chronic Obstructive Pulmonary Disease (COPD) and type 2 diabetes. He was an ex-smoker, who had recently stopped smoking.

On examination by the stroke team, he was found to have left sided facial droop, dysarthria with normal visual fields. He had reduced power in the left upper limb (score 4/5) and left lower limb (score 3/5). There was no evidence of inattention, ataxia or sensory impairment.

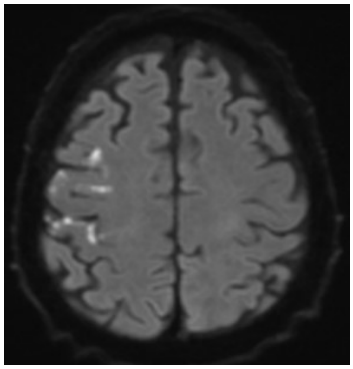


Image 1- MRI Head

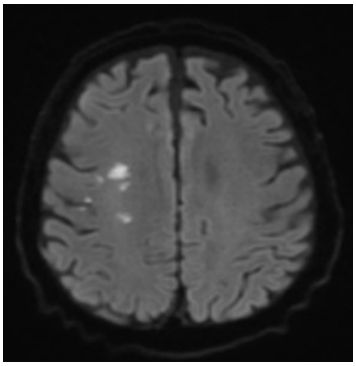


Image 2- MRI Head

His blood tests on admission were as follows; Hb 88, WCC 12.80, Platelets 274, CRP 56 and eGFR 10. A recent CT thorax confirmed the presence of a malignant mass in the left upper lobe of the lung.

Question 1: What are the findings from the MRI scans?

Question 2: What is the definition of a watershed territory?

Question 3: From this patient's history, what may predispose him to these findings?

Question 4: How should this patient be managed?

Answer 1: Multiple foci of acute infarction within the right MCA territory, however, there is also a single focus within the left MCA/PCA watershed territory.

Answer 2: A watershed territory is the area that lies at the junction between two major cerebral arteries. These areas are especially vulnerable to ischaemia because they are the furthest away from the direct arterial supply.

Answer 3: The multiple foci of acute infarction including the area in the watershed territory were suggestive of an embolic phenomenon. This is likely due to the hypercoagulable state created by his lung cancer, predisposing him to embolic infarcts.

Answer 4: The treatment for such infarcts remains the same as other ischaemic strokes. The patient was started on high-dose aspirin and moved to the stroke ward for rehabilitation.

Diagnosis: Watershed Infarct

Case 16

85-year-old gentleman presented to the emergency department after his wife found him to be more confused. His wife also noted that patient had a seizure following which he developed transient right-sided weakness. The patient denied any recent night sweats, weight loss or other significant symptoms. His past medical history included osteoarthritis and previous retinal artery occlusion. He lived with his wife and was completely independent with both mobility and ADLs.

He was examined by the stroke team which found no inattention, normal speech, laceration to the left of the tongue due to biting, no facial droop and no evidence of atrial fibrillation. He had reduced power in the right upper limb (4/5) and right lower limb (3/5).

Initial blood tests were as follows; Na 141, eGFR 73, Glucose 8.7, HbA1c 41, cholesterol 3.1, LDL 1.6, TSH 1.92, Hb 137, WCC 13.20 and neutrophils 11.11.

A CT and MRI head with contrast scan was performed in the emergency department for which the images are as follows:

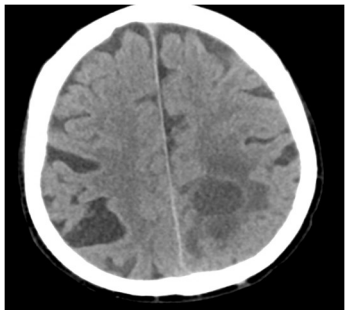


Image 1: CT Head

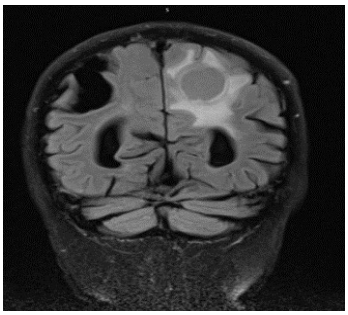


Image 2: MRI Head

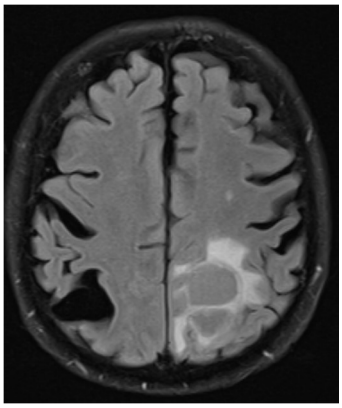


Image 3: MRI Head

Question 1: What are the findings seen on the CT and MRI head scans?

Question 2: What is the possible differential diagnosis for the above findings?

Question 3: What is the management of this condition?

Answer 1: There is a hypodense mass in the left parieto-occipital lobe, with surrounding oedema on CT. MRI confirms the presence of the intra-axial mass, with localised surrounding oedema and mass effect. No other intracranial mass seen on this study.

Answer 2: The most likely differentials would be a primary brain tumour (i.e. glioma), brain metastasis or lymphoma. Other less likely differentials would include a cerebral abscess, however there are no infective signs or symptoms.

Answer 3: Treatment depends on various factors but can include:
Surgical resection: if operable and the patient is fit. This aims to reduce mass effect and obtain tissue for diagnosis.
Radiotherapy: Adjuvant therapy.
Chemotherapy: Temozolomide is standard for glioblastoma.
Symptom management: Steroids, anticonvulsants, analgesia.
Palliative care: Early involvement for symptom control.

Diagnosis: Left Parietal Intra-Axial Tumour

Case 17

A 20-year-old gentleman presented to the Emergency Department with a two-day history of weakness, tingling sensations and pins and needles in the right hand. He also described unsteadiness when he walked and had an episode of slurring of his speech in the two days prior to admission. He had a past medical history of iron deficiency anaemia and learning difficulties.
He was seen initially by the stroke team who examined him and found no evidence of slurred speech, facial droop, inattention or sensory loss. However, he did have evidence of right-hand weakness (power 3/5) and pronator drift. There was also evidence of right upper limb reflexes being brisk including positive Hoffman's sign. Furthermore, there was right lateral gaze nystagmus but no evidence of internuclear ophthalmoplegia.
His blood tests at admission were as follows – Sodium 141, eGFR >90, CRP 9, calcium 2.40, HbA1c 33, cholesterol 5.9, LDL 4.4, Hb 162 and WCC 7.00.
A CT head and MRI scan of the head and spine were performed.

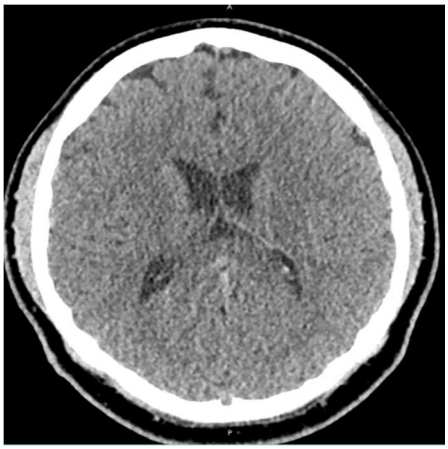


Image 1: CT Head

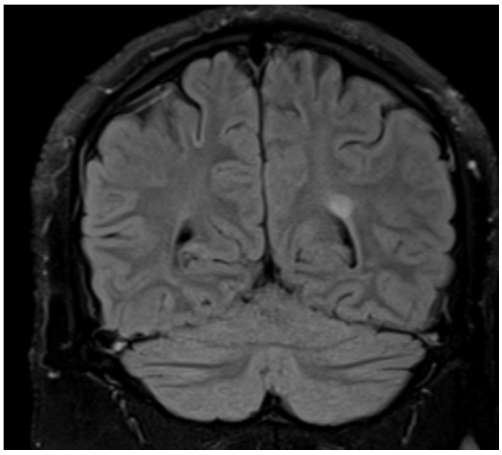


Image 2: MRI Head

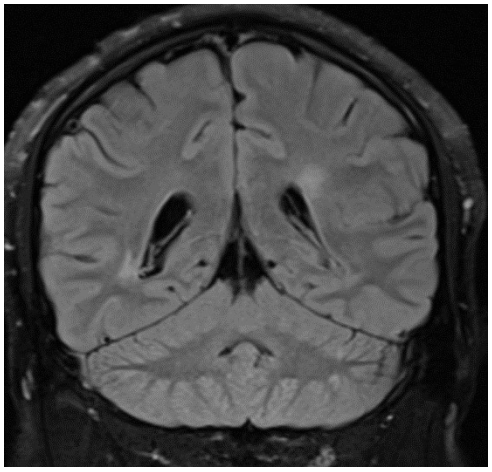


Image 3: MRI Head

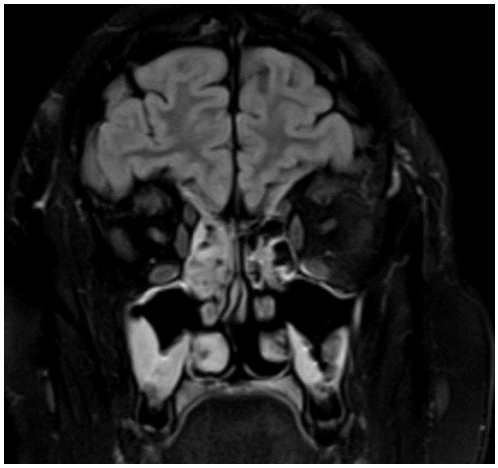


Image 4: MRI Head

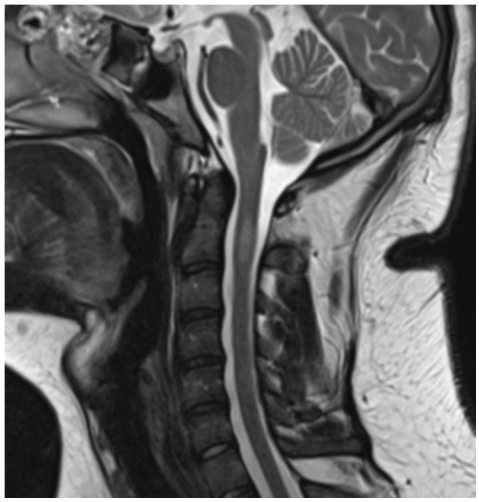


Image 5: MRI Spine

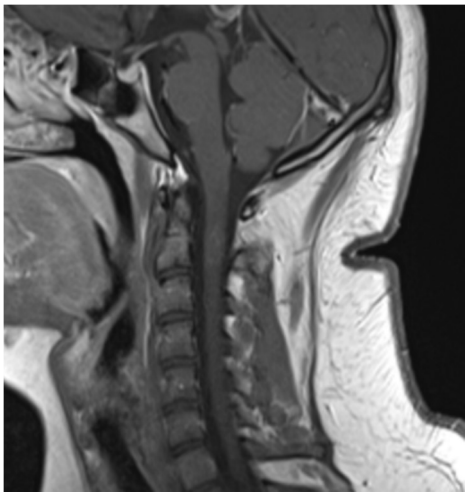


Image 6: MRI Spine

Question 1: What are the positive findings seen in the images above?

Question 2: What is the most likely diagnosis for this patient?

Question 3: What features in this case supports the above diagnosis?

Question 4: What is the acute and chronic management of this condition?

Answer 1: The CT demonstrated no abnormality. The MRI head shows FLAIR hyperintensity in white matter bilaterally and within the right optic nerve concerning for optic neuritis. The MRI spine shows that there is a single hyperintense focus in the cervical cord at the level of C2, measuring up to 16 mm, with spinal expansion and mild enhancement.

Answer 2: The diagnosis in this case was Multiple Sclerosis.

Answer 3: Corticospinal tract involvement explains right-hand weakness, pronator drift, brisk reflexes. Brainstem involvement accounts for right gaze-evoked nystagmus and possibly slurred speech. Cerebellar pathway involvement explains unsteadiness. This multifocal pattern is characteristic of disseminated CNS demyelination seen in Multiple sclerosis.

Answer 4: Initial acute management would involve high dose IV steroids (i.e. methylprednisolone) and then switch to a tapering dose of oral prednisolone. Chronic management involves treatment with disease modifying drugs such as beta-interferons and ocrelizumab.

Diagnosis: Multiple Sclerosis

Case 18

A 62-year-old female presented to the emergency department after suffering a 'funny turn' during which she remained conscious but had difficulty getting her words out. She was able to understand what was being said to her, however, she had difficulty remembering the names of the individuals that she was talking to. She also reported auditory hallucinations. This episode lasted for a few minutes and resolved spontaneously.

She also reported having several vacant episodes, lasting a few seconds, which were witnessed by others around her. Furthermore, she reported a left-sided pulsatile tinnitus which she did not find to be particularly intrusive. She had a past medical history of hypertension and hypothyroidism. She worked in a care home and was independent with her mobility and ADLs.

Her bloods on admission were as follows; Sodium 134, eGFR 82, calcium 2.44, cholesterol 4.8, LDL 2.9, Hb 128 and WCC 12.00. On examination by the stroke team, her pupils were found to be equal and reactive, no signs of inattention, no cerebellar signs and motor function was 5/5 in all four limbs. Her MRI scan is shown below after her initial presentation.

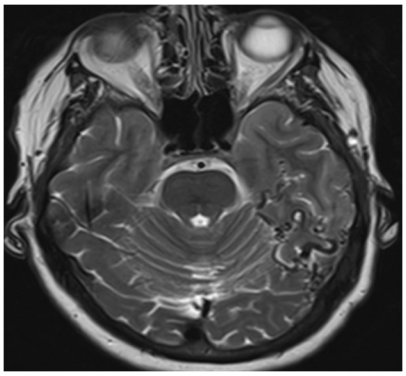


Image 1: MRI Head

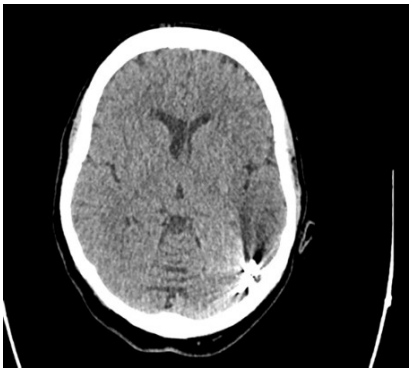


Image 2: CT Head

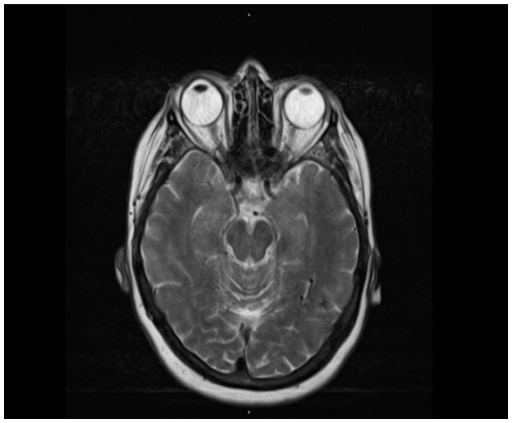


Image 3: MRI Head

Question 1: What is the important finding on the MRI head scan?

Question 2: The patient was referred for neuroradiological intervention. What are the findings in CT head scan and the MRI (T2 sequences) post intervention?

Question 3: What is a dural arteriovenous fistula (dAVF) and what is the best investigation for diagnosis?

Question 4: What are the treatment options for dural arteriovenous fistulas?

Answer 1: The MRI head scan shows prominent blood vessels in the left temporal lobe. These findings are in keeping with a left sided dural arteriovenous fistula (dAVF).

Answer 2: CT scan shows a hyperdense metallic coil mass with artefact in the left temporal lobe at the site of the previously treated dAVF. MRI (T2 sequence) demonstrated resolution of venous congestion, with some gliosis and a small void at the site of the coil.

Answer 3: A dAVF is an abnormal connection between an artery and a vein located in the dura mater. They involve direct shunting of blood from dural arteries into dural venous sinuses or cortical veins, bypassing the normal capillary network. The gold-standard investigation for diagnosis is a digital subtraction angiography.

Answer 4: Depends on symptoms and haemorrhage risk. Options include:

- Endovascular embolisation (common approach)
- Surgical disconnection
- Stereotactic radiosurgery

Some low-risk, asymptomatic dural arteriovenous fistulas may be monitored without immediate treatment.

Diagnosis: Dural Arteriovenous Fistula

Case 19

A 64-year-old female who presented to the Emergency department following a one-day history of sudden onset slurring of speech, confusion, left-sided weakness and paraesthesia. She denied any tonic-clonic seizures, photophobia or neck stiffness. She had no significant past medical history. She worked in an office and was independent with her mobility and ADLs. She had no significant smoking or alcohol history.

She was reviewed by the stroke team who found that her power (MRC scale) was 4/5 in the left upper limb and 3/5 in the left lower limb. There was no evidence of facial drooping, pronator drift or any other neurological signs.

Her bloods on admission were unremarkable. A CT head scan was done which was reported as normal. She was treated with high dose aspirin initially while she awaited an MRI head scan. The images for the MRI head scan are as seen below.

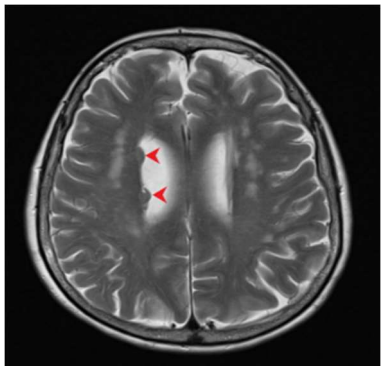


Image 1: MRI Head

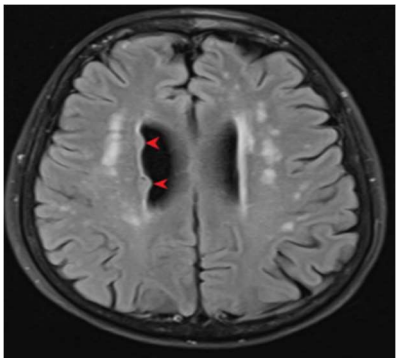


Image 2: MRI Head

Question 1: What are the positive findings seen on the MRI head?

Question 2: What is grey matter heterotropia (GMH)?

Question 3: What is the aetiology of GMH?

Question 4: What is the most common presentation for patients with GMH?

Question 5: What are the treatment options for GMH?

Answer 1: Axial section at the level of lateral ventricles shows sub ependymal nodules (red arrows). These incidental small nodules in the right periventricular deep white matter have appearances of grey matter heterotopia (GMH).

Answer 2: GMH are developmental disorders characterised by interruption of normal neuronal migration to the cortex thus normal neurones are located in abnormal areas. These foci of heterotopia therefore demonstrate signal identical to grey matter on all MRI sequences. They are classified as periventricular nodular heterotopia (PNH), subcortical heterotopia (SCH), subcortical band heterotopia (SBH), and mixed type.

Answer 3: Their aetiology is attributed to both genetic and epigenetic factors. The likely cause is abnormal migration of neuronal cells, during the developmental stage.

Answer 4: GMH commonly presents with either epilepsy or a range of psychiatric manifestations, which include delirium, cognitive decline, and schizophrenia to name a few. GMH can present early with intellectual disability, epilepsy and speech disorder. Adult-onset GMH usually presents with mild cognitive decline, and neurological manifestations are exceedingly rare.

Answer 5: There is no specific treatment for patients with grey matter heterotopia. GMH needs to be identified by a physician and differentiated from other lesions, as due to its rare presentation it may be missed. Other

medical management involves anti-epileptic medications and surgical intervention in those with drug-resistant epilepsy.

Diagnosis: Grey Matter Heterotropia

Case 20

A 42-year-old gentleman of Afro-Caribbean origin presented to the emergency department with a gradual onset headache, dizziness, double vision and difficulty in maintaining his balance. He reported the onset of these symptoms to be over three days prior to admission. He had no significant past medical or surgical history. He worked as a builder and lived with his family. He did not smoke and drank alcohol socially.

He was reviewed by the stroke team in the emergency department. There was no evidence of loss of power in both upper and lower limbs. There was no pronator drift or pupillary abnormalities. His gait was ataxic, and he reported double vision on lateral gaze.

His blood tests appeared grossly normal on admission with Hb 120, sodium 130, eGFR 90, cholesterol 4.6, LDL 2.5, and WCC 10.00. An initial CT head was done which reported bilateral cerebellar infarcts. The MRI head scan showed the findings presented below.

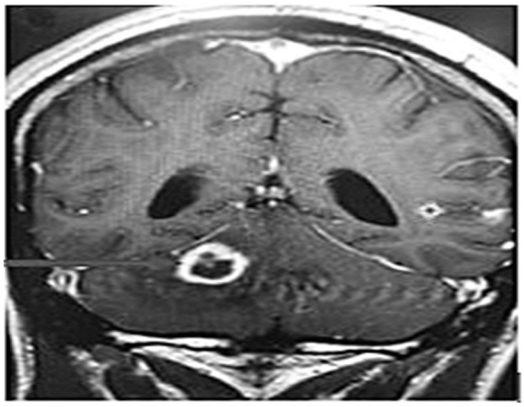


Image 1: MRI Head

Question 1: What is the positive finding seen in the MRI head scan?

Question 2: What is the above finding indicative of?

Question 3: What other underlying condition must be tested for in view of these MRI findings?

Question 4: How should this patient be managed?

Answer 1: The MRI head scan demonstrates a few thick-walled ring-enhancing lesions, the largest in the right cerebellum, with an eccentric nodule.

Answer 2: The above-mentioned ring-enhancing lesions are commonly associated with a diagnosis of cerebral toxoplasmosis.

Answer 3: It is important to screen these patients for HIV infection. In this patient, the CD4 count was low (59), CD8 was 440 and serology for HIV was positive.

Answer 4: This patient was managed by a multidisciplinary team including neuro-oncologists, infectious disease and genitourinary medicine specialists. He was treated for HIV related cerebral toxoplasmosis with sulfadiazine, pyrimethamine, folinic acid along with anti-retrovirals. He responded well to this treatment and was left with no residual neurological symptoms.

Diagnosis: Cerebral Toxoplasmosis with HIV

Case 21

A 94-year-old gentleman presented to the emergency department with numbness in his left hand. He described feeling a sensation of pins and needles involving the lateral 3 fingers as well as the left perioral region. He also described weakness in the left upper limb which lasted approximately 30 minutes. He denied any speech difficulty or visual symptoms. He mentioned sustaining a head injury 2 months before his current presentation. He had a past medical history of atrial fibrillation, and he was an ex-smoker.

His medications included digoxin, simvastatin, warfarin and omeprazole.

He was seen by the stroke team and examination revealed that his cranial nerves were intact, normal visual fields, no signs of focal neurological deficit, no residual sensory deficit and no hemi spatial neglect. His blood tests showed a normal full blood count and renal function. His CRP was 18, INR 2.83 and lipids, cholesterol and LDL were in normal range. His ECG showed long-standing rate-controlled AF.

This patient underwent a CT head and MRI scan which are shown as below.

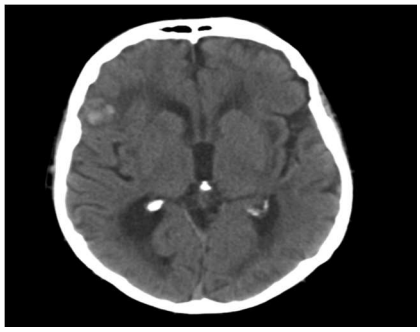


Image 1: CT Head

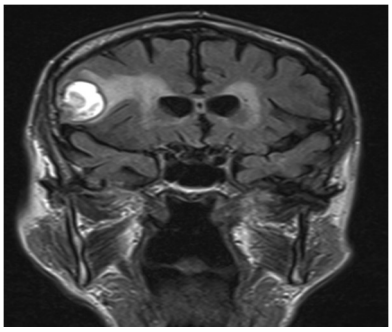


Image 2: MRI Head

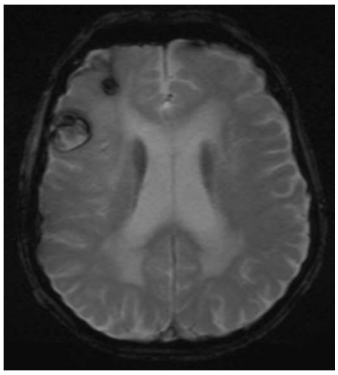


Image 3: MRI Head

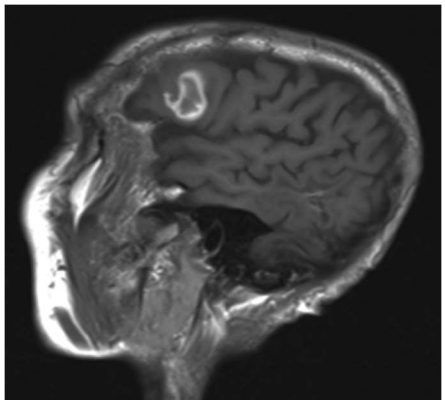


Image 4: MRI Head

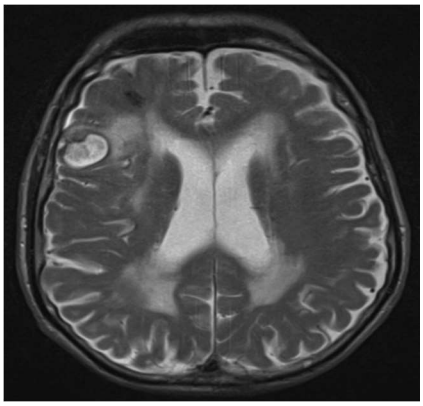


Image 5: MRI Head

Question 1: What are the findings as seen in the CT and MRI head scans above?

Question 2: What do you think is the diagnosis in this case?

Question 3: What is cerebral amyloid angiopathy?

Question 4: How do we obtain a definitive diagnosis for cerebral amyloid angiopathy?

Answer 1: The CT demonstrates an area of high density in the periphery of the right frontal lobe, suggestive of haemorrhage. The MRI confirms the presence of two haematomas and evidence of previous haemorrhage in the right frontal lobe, with surrounding oedema.

Answer 2: The diagnosis in this patient was cerebral amyloid angiopathy as demonstrated in the scans above.

Answer 3: Cerebral amyloid angiopathy is a condition where amyloid proteins (usually amyloid- B) are deposited into the walls of small-to-medium sized blood vessel in the brain. The deposition usually occurs in the cerebral cortex or in the leptomeninges.

Answer 4: The histopathological diagnosis of this condition can be obtained via a brain biopsy. Congo-red staining reveals apple-green birefringence under polarised light.

Diagnosis: Amyloid Angiopathy

Case 22

A 44-year-old gentleman presented to the emergency department with left arm and leg weakness, and slurred speech. He described a generalised headache, a 'tingling' sensation in his left hand. He described the symptoms being present for at least four days. He had no significant past medical history and there was no family history of stroke.

He was seen by the stroke team in the emergency department. He had weakness in the left upper and lower limb weakness (MRC grade 3/5). He also demonstrated left sided facial droop. There were no visual field defects and no signs of any cerebellar pathology. An ECG showed rate controlled atrial fibrillation. A chest X-ray was done which was normal. Blood tests were normal including vasculitis and thrombophilia screens. The patient's symptoms resolved within 24 hours of admission.

A CT head scan was done which did not show any acute infarct or bleeding and no midline shift. An MRI head scan was arranged and the images for this are as follows. This patient also had a transthoracic echocardiogram followed by a transoesophageal echocardiogram (TOE), and the image from the TOE are as seen below.

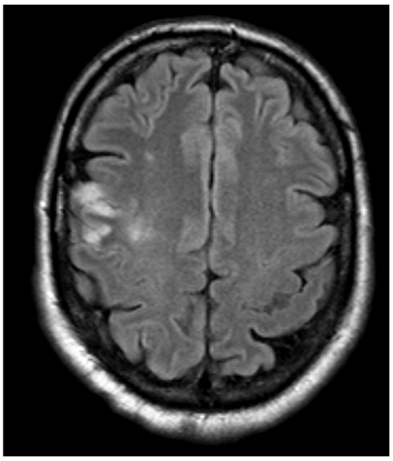


Image 1: MRI Head

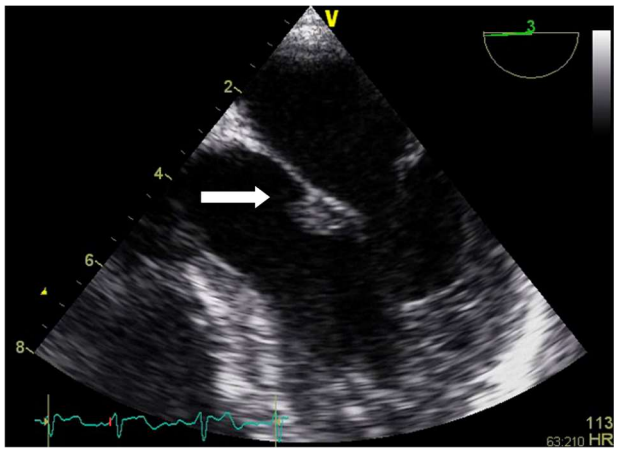


Image 2: Transoesophageal echocardiogram

Question 1: What are the findings seen in the MRI head scan?

Question 2: What are the findings seen on the TOE?

Question 3: How do you think that this patient was managed?

Question 4: What are the common types of primary heart tumours?

Answer 1: The MRI scan shows an area of increased T2 hyperintensity in the posterior part of the right frontal lobe, other sequences confirmed that this was in keeping with an acute infarct.

Answer 2: The TOE shows two masses attached to the left ventricular side of the anterior leaflet, one measuring 0.6 x 1cm and a smaller one measuring 0.4 x 0.5cm. Both masses were echo dense, pedunculated, mobile and stippled in appearance. The left atrial appendage showed no evidence of a thrombus and there was a normal flow velocity. Trivial central mitral regurgitation was also seen. A diagnosis of a mitral valve papillary fibroelastoma was made. This was the cause of the embolic stroke that was seen on the MRI head scan.

Answer 3: This patient was started on a direct oral anticoagulant called dabigatran- 150mg twice daily. Advice was sought from the cardiothoracic surgeons who suggested to manage this patient medically in the first instance. If this patient were to have further embolic events, they would consider surgery to prevent further thromboembolic events.

Answer 4: Myxomas are the most common of the cardiac tumours. These are predominantly left sided tumours which are attached to the atrial septum by a stalk. After a myxoma and lipoma, the third most common cardiac tumour is a papillary fibroelastoma (PFE)

Diagnosis: Mitral Valve Fibroelastoma

Case 23

A 49-year-old gentleman presented to the emergency department with a 3-day history of generalised abdominal pain and ongoing pyrexia. He also reported feeling lethargic with poor appetite and associated nausea. He had no significant past medical history and no recent travel or surgical procedures. There was no significant family history of autoimmune conditions or strokes.

He was noted to be pyrexial with a temperature of 38.5°C, with localised tenderness in the right lower quadrant on abdominal examination. Initial inflammatory markers were raised, with a CRP of 125 and WCC of 14.3. A working diagnosis of acute intra-abdominal sepsis was made, and he was admitted under the surgical team for further evaluation and management.

On day 2 of admission, the patient developed sudden onset left-sided weakness affecting both the upper and lower limbs, along with right-sided facial droop and slurred speech. Neurological examination revealed left-sided hemiparesis (MRC grade 2/5) and dysarthria. His NIH Stroke Scale (NIHSS) score was 10.

An urgent CT head scan was arranged for this patient, and he had urgent blood cultures taken. He also underwent an echocardiogram for which the images are as seen below.

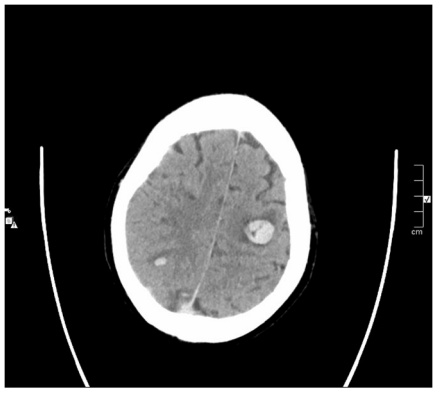


Image 1: CT Head

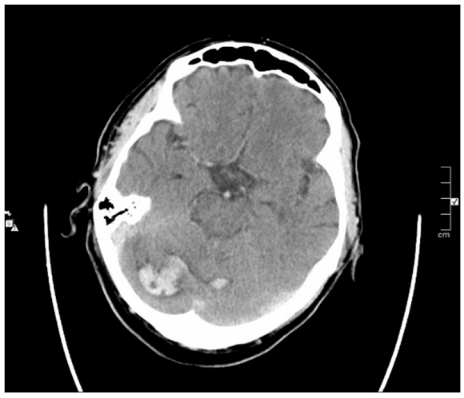


Image 2: CT Head

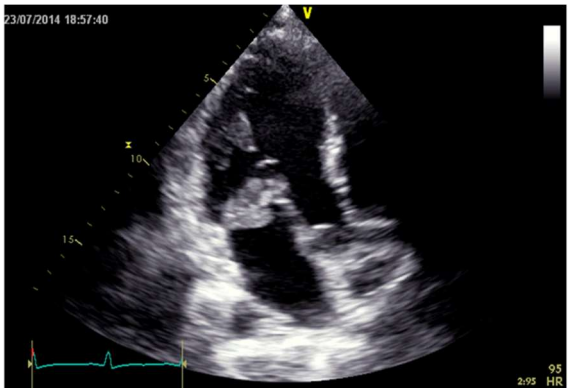


Image 3: Echocardiogram

Question 1: What are the findings as seen on the CT scan?

Question 2: What are the findings as seen on the echocardiogram?

Question 3: What would be the management for this patient?

Answer 1: There are several, peripheral, bilateral areas of high density in the cerebral parenchyma and cerebellum.

Answer 2: The echocardiogram shows a mobile vegetation on the mitral valve which confirms the diagnosis of infective endocarditis as the cause for embolic stroke.

Answer 3: The patient was started on IV antibiotics with microbiology guidance and was transferred to the stroke unit, with joint care of the cardiology team. Anticoagulation was deferred in the acute phase due to the risk of haemorrhagic transformation. A repeat MRI was planned, and he remained under close neurological and cardiac monitoring.

Diagnosis: Thromboembolic Stroke secondary to Infective Endocarditis

Case 24

A 86-year-old female was admitted to the emergency department as she was found to be unconscious. Her daughter was able to give a brief history and explained that she found her mother unconscious on the sofa. Her daughter also mentioned that the patient had a cough for the three days prior to admission, with associated confusion and drowsiness. The patient had recently been diagnosed with oesophageal cancer and had a stent inserted under the gastroenterology team. Other past medical history included congestive cardiac failure, hypertension and osteoarthritis.

Her current medications included omeprazole, buprenorphine, domperidone, furosemide, levothyroxine and paracetamol. She lived alone and was independent with her activities of daily living and mobility.

She was assessed by the stroke team who found her GCS to be 3/15. Her initial blood pressure was recorded as 152/80 with HR of 92 bpm. She had a nasopharyngeal tube inserted by the emergency department and was requiring 15L of oxygen to maintain saturations above 92%. Her blood tests showed a slightly raised urea at 9.1 otherwise the renal profile was unremarkable. Her ALP was raised at 159 but had a normal liver and bone profile. Her Hb was 111, WCC 12.9, platelets 431 and CRP 132.

An urgent CT head scan was performed, and the images are as seen below.

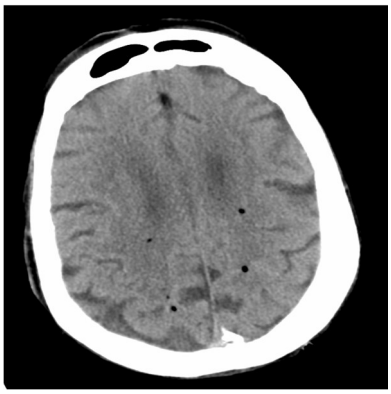


Image 1: CT Head

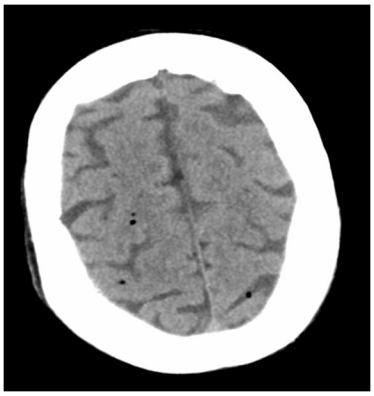


Image 2: CT Head

Question 1: What are the positive findings seen in the CT head scan?

Question 2: What is the most likely diagnosis and what is a less likely differential diagnosis?

Question 3: What would be the important aspects in history, examination and investigations when investigating the above finding?

Answer 1: There are several, scattered, small (1-6mm) peripheral locules of intracranial fat density globules in both cerebral hemispheres.

Answer 2: The most likely diagnosis based on the findings in the CT head scan is that of fat embolism. Another less likely differential diagnosis would be a simple (non-tension) pneumocephalus.

Answer 3: It is important to ascertain any recent history of falls or new fractures. It would be essential to assess this patient for fractures of the long bones or clavicles. X-ray would be the first line investigation if there is any suspicion of fracture.

Diagnosis: Fat Embolism Syndrome

Case 25

A 54-year-old female presented to the emergency department following a sudden onset headache and collapse while at work. According to her colleagues, she had been well earlier in the day when she suddenly developed a severe headache and subsequently collapsed. She experienced a brief loss of consciousness, lasting approximately 2 minutes.

By the time she arrived in hospital, she had regained full consciousness (GCS 15) but continued to complain of a persistent, dull headache, which was less severe than at the time of onset. The pain was frontal in location, with no associated photophobia, nausea, vomiting, or neck stiffness. She denied any limb weakness, visual changes, or sensory disturbance. There was no significant past medical history, and she was not taking any anticoagulants or antiplatelet agents. There was no family history of aneurysms or early cerebrovascular disease.

On examination, she was neurologically intact with no focal neurological deficits. There was no neck stiffness, and fundoscopy revealed no papilloedema. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

An urgent CT head scan was performed and the images for this are as seen below.

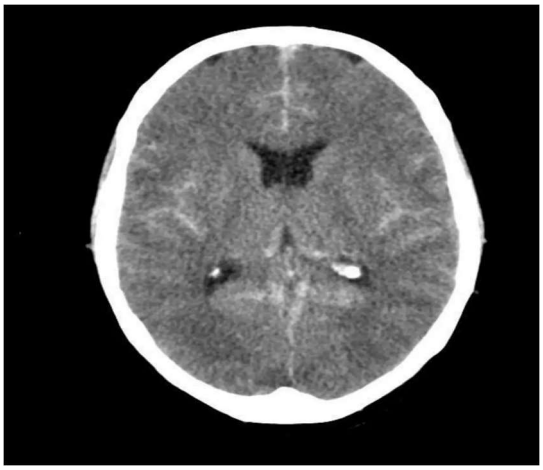


Image 1: CT Head

Question 1: What are the findings seen on the CT head scan?

Question 2: What was the most appropriate management plan for this patient?

Answer 1: There is widespread subarachnoid blood. Investigations for an underlying cause for this (e.g. aneurysm) should be performed. There is an intra-axial haemorrhage located between the frontal lobes, consistent with a haemorrhage in the interhemispheric fissure.

Answer 2: She was admitted under the care of the stroke and neurology teams, with early input from neurosurgery. She remained neurologically stable throughout her admission and was managed conservatively with close monitoring, analgesia, and blood pressure control.

Diagnosis: Intra-axial Haemorrhage

Case 26

A 52-year-old male was admitted to the emergency department (ED) after sustaining a fall at home three days ago. At the time of the fall, he had no apparent loss of consciousness and did not seek any medical attention immediately. However, over the past 48 hours preceding his admission, his family noticed that he had become increasingly confused, with slurred speech and progressive weakness on the right side.

He was known to have a long-standing history of alcohol abuse, with previous hospital admissions for alcohol-related complications, including withdrawal seizures and decompensated liver disease. He was not on any anticoagulant or antiplatelet therapy, and there was no known history of epilepsy, stroke, or traumatic brain injury.

On arrival to ED, he was drowsy but rousable (GCS 13) and disoriented to time and place. Neurological examination revealed right-sided hemiparesis (MRC grade 3/5) and dysarthria. Cranial nerve examination showed no visual field defects or gaze abnormalities. His cardiovascular and respiratory examinations were unremarkable, and there were no signs of neck stiffness or photophobia. An urgent CT head scan was arranged and the images for this are as seen below.

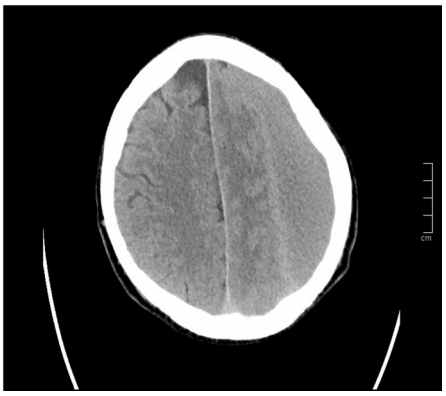


Image 1: CT Head

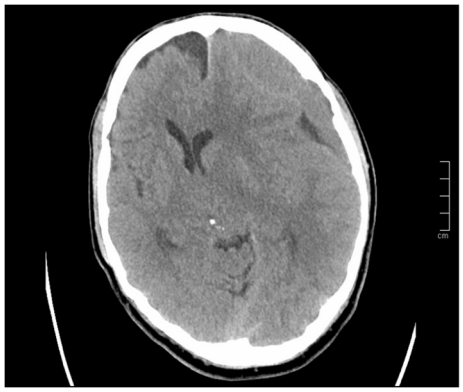


Image 2: CT Head

Question 1: What are the findings seen on the CT head scan?

Question 2: Why is a subdural haemorrhage particularly associated with alcohol abuse?

Question 3: What complications should we be mindful of in this patient?

Question 4: What findings may require urgent neurosurgical intervention in this patient?

Answer 1: Large left subdural haematomas causing effacement of the underlying parenchyma and midline shift.

Answer 2: Patients who are known to have a history of alcohol misuse are likely to have cerebral atrophy, increased risk of falls and evidence of coagulopathy. These factors increase the risk of developing a subdural haemorrhage, even with a low-impact head injury.

Answer 3: It is important to monitor for any seizure activity secondary to the haemorrhage. It is also important to monitor for any signs of raised intracranial pressure, alcohol withdrawal and aspiration of gastric contents.

Answer 4: If the patient has a midline shift of >5mm, thickness of haemorrhage of >10mm or sudden neurological deterioration, this would warrant urgent neurosurgical intervention.

Diagnosis: Subdural Haemorrhage

Case 27

A 40-year-old male patient presented to the emergency department with difficulty in speech. His parents found the patient in bed with a bruise around his left eye. He was found to have both receptive and expressive dysphasia. His parents reported that the patient had a fall twelve days prior to admission, but there were no acute medical concerns at the time. He had a past medical history of excess alcohol consumption (drinking 200 units of alcohol per week), portal hypertension and oesophageal varices (banded after recent upper GI bleed).

His regular medications included vitamin B supplements, thiamine and furosemide. He lived with his parents, was unemployed and smoked 10 cigarettes a day. Initial examination by the stroke team did not reveal any focal deficits. His GCS was 13/15 and he was found to have both expressive and receptive dysphasia. His blood tests were normal.

During his time in the emergency department, he became more disruptive and aggressive. He attacked a member of staff and was sedated with haloperidol.

He had an urgent CT scan followed by an MRI scan. The images for these scans are seen below.

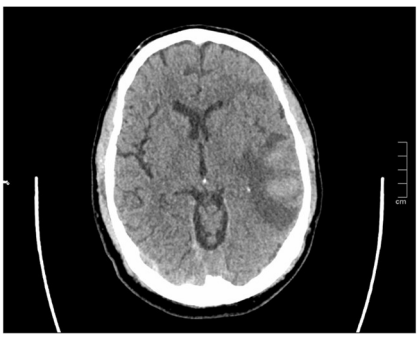


Image 1: CT Head

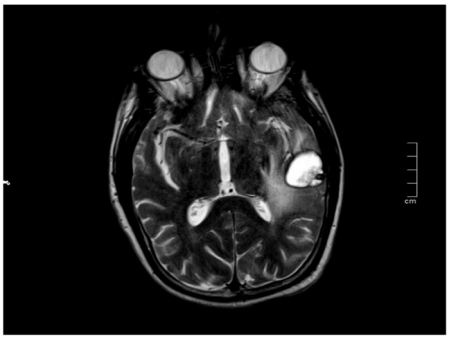


Image 2: MRI head

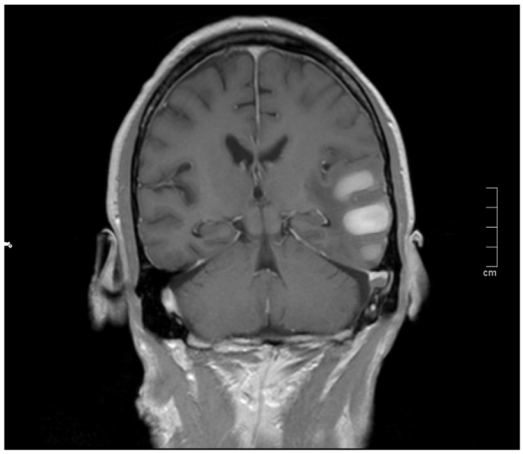


Image 3: MRI head

Question 1: What is the finding seen on the CT head scan?

Question 2: What are the findings seen on the MRI head scans?

Question 3: Based on the findings mentioned from the MRI head scan, what do you think is the diagnosis in this patient?

Question 4: What are the common causes and treatment of CVST?

Answer 1: The CT head scan shows increased density in the left temporal lobe in keeping with haemorrhage, with surrounding oedema.

Answer 2: The MRI head scan shows corresponding areas in the periphery of the left temporal lobe. There is surrounding oedema in the left temporal lobe. Further contrast studies were done which showed large volume filling defect in the left transverse and sigmoid sinus extending into the jugular foramen. There was also absence of venous flow in these areas.

Answer 3: This patient has a diagnosis of cerebral venous sinus thrombosis (CVST) with evidence of haemorrhagic infarction in the left temporal lobe. This resulted from the left transverse and sigmoid sinus thrombosis.

Answer 4: Some of the common causes for CVST are thrombophilia, trauma, surgery, pregnancy, IBD and meningitis. The mainstay of management for CVST is anticoagulation therapy using heparin or warfarin to prevent further clot propagation.

Diagnosis: CVST with haemorrhagic venous infarction

Case 28

A 72-year-old male was admitted to the emergency department due to a 2-week history of increasing confusion. He also reported difficulty in producing coherent speech and excessive drowsiness. Five days prior to admission, he had returned from holiday abroad. His family described him to be completely off his baseline - he was fit and well prior to this episode. The patient had a 2-day history of fever, for which the GP started him on oral antibiotics to cover for a urinary tract infection.

He had a past medical history of hypertension, raised cholesterol, overactive bladder, migraines, benign prostatic hypertrophy, migraine and right shoulder replacement. His current medications included aspirin, atorvastatin, solifenacin and candesartan. His mother had a stroke at the age of 74 and there was no other family history of strokes or intracranial haemorrhage. He was independent with his mobility and activities of daily living. He did not smoke but did drink 20 units of alcohol per week.

He was examined by the medical team in the emergency department. His cranial nerves were intact, no cerebellar signs and no sensory deficits. He had reduced power in his right leg (MRC score 4/5) and no other focal neurological deficits were noted. His initial blood tests shoed no significant abnormalities: Na+ 133, eGFR 79, CRP 2, Hb 131 and WCC 6.40.

An urgent CT head and subsequently MRI head scans were performed.

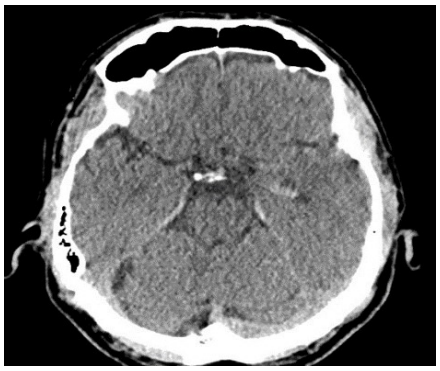


Image 1: CT Head

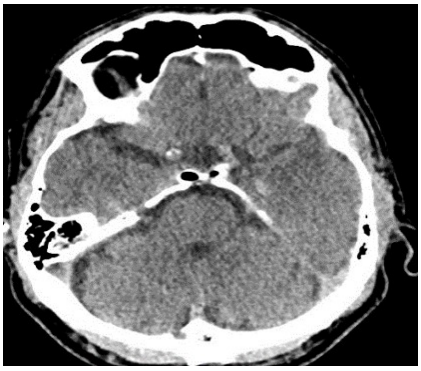


Image 2: CT Head

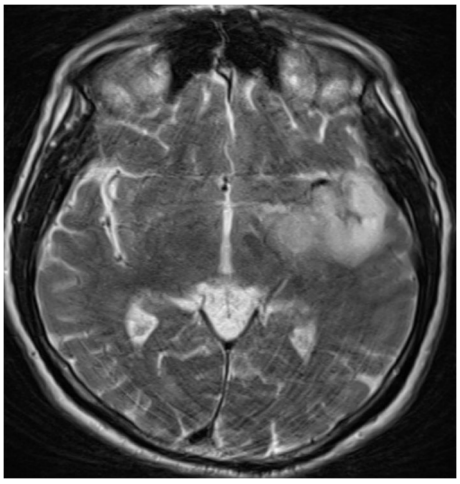


Image 3: MRI Head

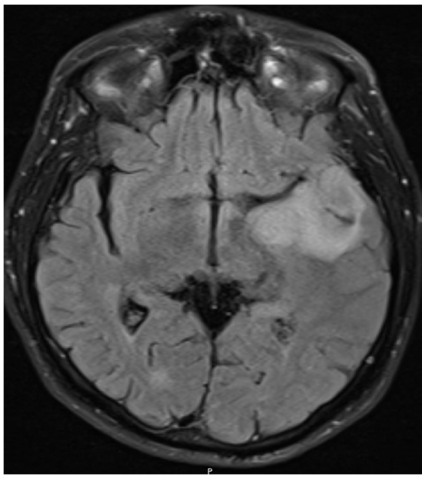


Image 4: MRI Head

Question 1: What the findings seen in the CT head scans?

Question 2: What are the findings seen in the MRI head scan?

Question 3: In a patient presenting with the history above, what is the most important differential diagnosis?

Question 4: What is the best management plan for this patient?

Answer 1: The CT head scan demonstrates a small intraparenchymal haematoma in the medial aspect of the left temporal lobe. No evidence of midline shift.

Answer 2: There is extensive oedema seen at the left temporal lobe.

Answer 3: Given the history of fever and confusion, the most likely diagnosis in this patient is acute encephalitis with haemorrhagic transformation. Another possible differential diagnosis for this patient is an underlying space-occupying lesion.

Answer 4: This patient was started on IV dexamethasone to reduce oedema surrounding the haemorrhage. He was started on a 21-day course of IV Acyclovir to cover for encephalitis. Advise was sought from the neurosurgery team who advised a contrast MRI scan in 6 weeks to assess for underlying space-occupying lesion. Antiplatelet medication was held during admission and the patient recovered fully after treatment with acyclovir and steroids. An MRI contrast was done 6 weeks post admission which showed no signs of an underlying space occupying lesion.

Diagnosis: Haemorrhagic Encephalitis

Case 29

A 51-year-old gentleman presented to the emergency department from the neurological rehabilitation facility. His history included a fall, after which he developed acute right subdural hematoma and intracerebral haemorrhage. He was seen by the neurosurgical team who performed embolization, Arteriovenous Malformation (AVM) excision, and a right frontal craniectomy. Postoperatively, the patient experienced prolonged agitation. His neurological examination showed power of 4/5 on left side, prompting discharge to a secondary centre for rehabilitation, where he wore a helmet during therapy sessions. Initially responding well to therapy, the patient later developed left-sided weakness, visual inattention and postural instability.

He was transferred to the emergency department following these findings at the rehabilitation facility. He was assessed by the stroke team. Their examination findings revealed reduced power in left upper and lower limbs (MRC score 4/5), left sided visual inattention, no sensory deficits, no dysarthria or facial drooping. His blood tests were initially normal with a Na 130, eGFR 90, Hb 110, WCC 10.10 and CRP 3.

He underwent an urgent CT head scan in the view of these findings.

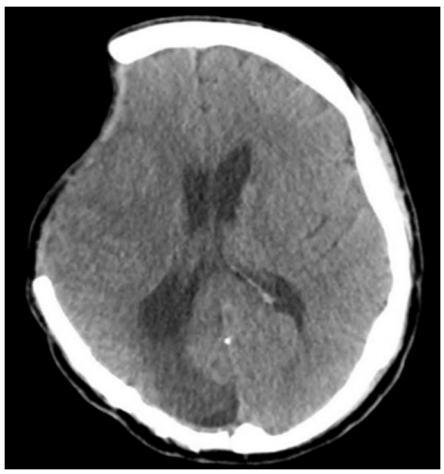


Image 1: CT Head

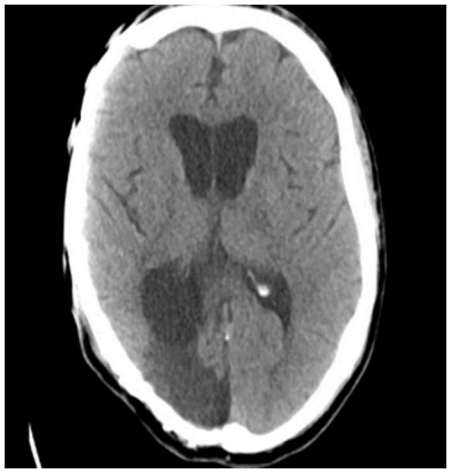


Image 2: CT Head

Question 1: What are the CT head scan findings?

Question 2: How do patients with this diagnosis present?

Question 3: What is the management of the above diagnosis?

Answer 1: A concave appearance of the craniectomy site and a sunken skin/scalp flap, confirming the diagnosis of sinking skin flap syndrome.

Answer 2: Sinking skin flap syndrome manifests through distinctive clinical features; most patients present with orthostatic symptoms such as headache, nausea and vomiting. Some patients present with focal neurological deficit, which is a rare but recognised manifestation of sinking skin flap syndrome and often manifesting weeks to months post-operatively. Patients may present with symptoms such as seizures, agitation, visual inattention, and motor weakness, which can progress rapidly if left unaddressed.

Answer 3: The cornerstone of sinking skin flap syndrome management lies in a multidisciplinary approach. Neurosurgical consultation is imperative, with cranioplasty emerging as a key therapeutic intervention. Elective titanium cranioplasty has shown significant success in restoring the structural integrity of the skull and reversing neurological deficits.
The patient underwent elective titanium cranioplasty (CT head Image 2) at a tertiary centre, resulting in significant recovery.

<u>Diagnosis</u>: Sinking Skin Flap Syndrome

Case 30

A 55-year-old male was admitted to the emergency department due to sudden-onset left-sided weakness. He reported that his symptoms started abruptly whilst watching television earlier that afternoon. He described a heavy sensation in his left arm and leg, accompanied by numbness and difficulty gripping objects. He denied any headache, visual disturbances, speech difficulties, or loss of consciousness. The symptoms improved partially when he was distracted and worsened when attention was drawn to them. His wife, who was present at the time of symptom onset, noted that he appeared distressed but did not exhibit facial drooping or slurred speech.

He had a past medical history of type 2 diabetes mellitus, hypertension, depression, and chronic lower back pain. His current medications included metformin, amlodipine, sertraline, and paracetamol as needed. There was no personal or family history of stroke or transient ischaemic attacks. He was independent with his mobility and activities of daily living. He was a non-smoker and reported occasional alcohol consumption (4–6 units per week). He was examined by the stroke team in the emergency department. His cranial nerves were intact, with no evidence of facial weakness or dysarthria. Tone was normal throughout. Power testing revealed variable weakness in the left arm and leg, with inconsistent effort and a “give-way” quality (MRC score ranging from 2/5 to 4/5). Sensation was intact, and there were no cerebellar signs. Reflexes were symmetrical, and plantar responses were flexor bilaterally. His initial blood tests did not show any

significant abnormalities: Na 138, eGFR 85, CRP 1, Hb 147, WCC 6.1. He underwent an urgent CT head followed by an MRI head scan in the view of these findings.

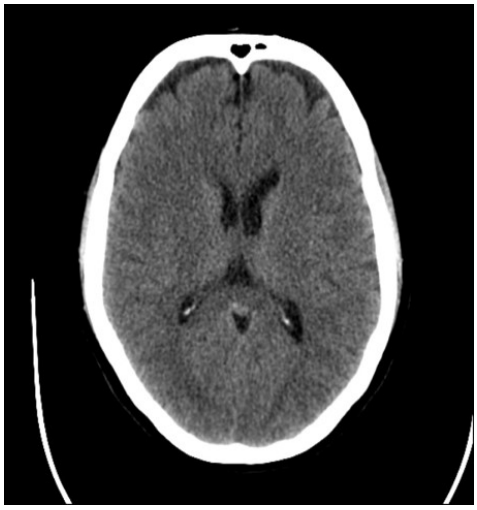


Image 1: CT Head

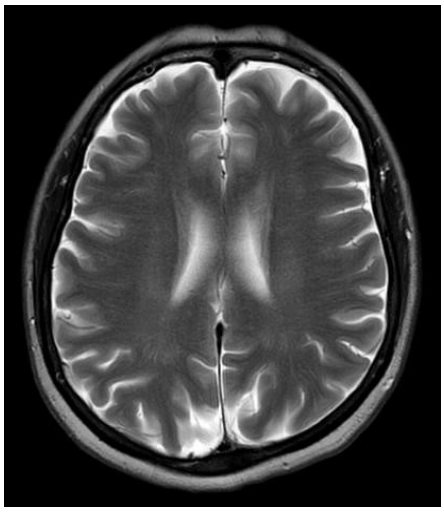


Image 2: MRI head

Question 1: What are the findings demonstrated by the CT and MRI head scans?

Question 2: What is the likely diagnosis?

Question 3: What is the significance of variable power and "give-way" weakness on examination?

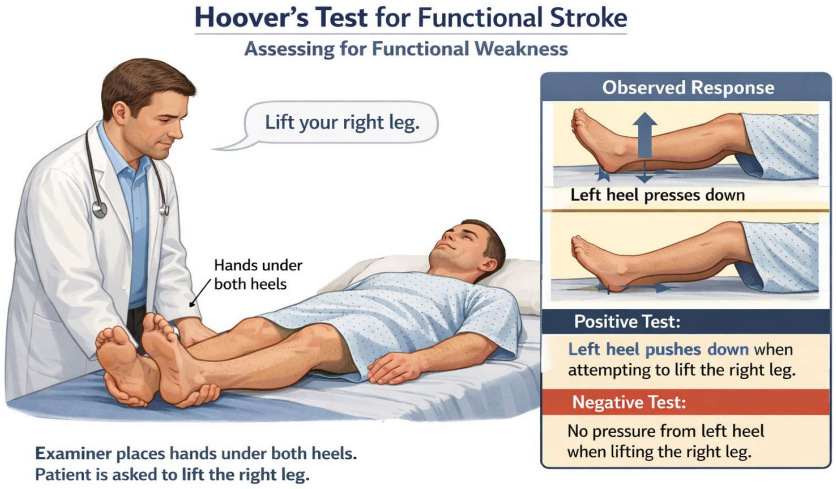
Question 4: How can organic weakness be differentiated from functional weakness at the bedside?

Answer 1: The images for both scans do not demonstrate any obvious abnormalities or signs of infarction or haemorrhage.

Answer 2: The diagnosis in this case was functional stroke. A functional stroke is considered where a patient presents with stroke-like symptoms, such as sudden weakness, numbness, or speech disturbance, but without evidence of structural brain damage.

Answer 3: It is a positive sign of functional weakness, as organic weakness is typically consistent and effort-independent.

Answer 4: Hoover's sign (as shown below), collapsing weakness ("give-way" weakness) and arm drop test (non-physiological movement patterns).



<u>Diagnosis</u>: Functional Stroke

Case 31

A 24-year-old Asian woman was admitted to the emergency department with a one-week history of severe right occipital headache. The headache was described as constant, progressively worsening, and pressure-like in nature. Over the preceding two days, she had noticed mild weakness affecting her left arm and leg. She also reported associated nausea, one episode of vomiting, and mild phonophobia. She denied photophobia, visual disturbances, seizures, loss of consciousness, or fever. There was no recent head trauma. The headache was partially relieved by simple analgesia.

She had no significant past medical history and was not taking any regular medications. There was no personal or family history of migraine, epilepsy, or cerebrovascular disease. She had immigrated to the UK five years earlier and reported having travelled to rural areas of South Asia within the past year. She was independent in activities of daily living, was a non-smoker and did not consume alcohol.

On examination, she was alert and oriented, with normal vital signs. Cranial nerve examination was unremarkable, with no papilloedema, visual field defects, or facial asymmetry. Motor examination demonstrated mild weakness in the left upper and lower limbs (MRC 4+/5), with normal tone. Sensory examination was intact, and coordination was normal. Reflexes were symmetrical, and plantar responses were flexor bilaterally. Gait assessment was limited by headache but did not reveal ataxia.

Initial blood investigations were within normal limits, including inflammatory markers and electrolytes (Na 140, eGFR 92, CRP <1, Hb 134, WCC 5.8).

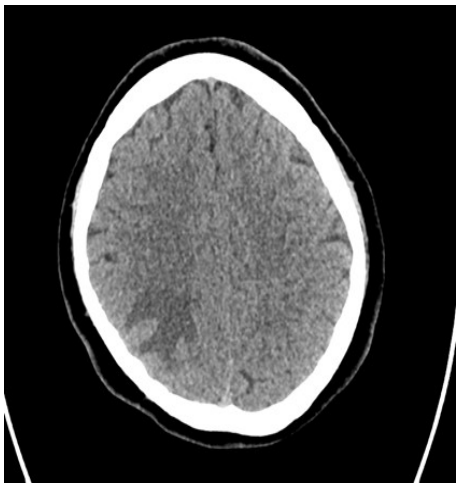


Image 1: CT Head

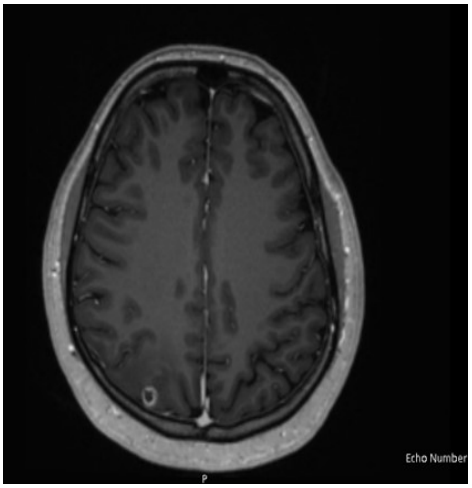


Image 2: MRI Head

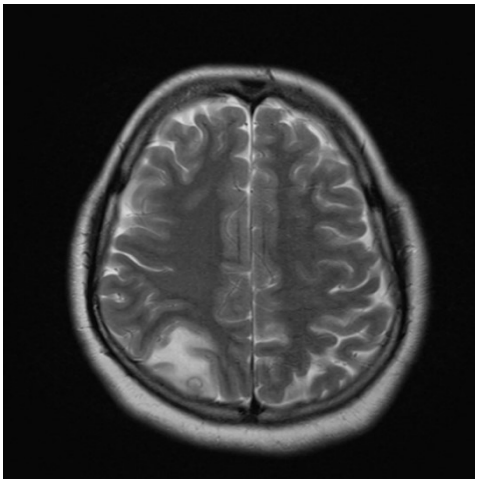


Image 3: MRI Head

Question 1: What abnormality is seen on the CT scan? What other investigations would you like and are the possible differentials?

Question 2: What abnormality do you see on the MRI head with contrast?

Question 3: What are the other investigations that can be done to confirm the likely diagnosis?

Question 4: What complications can occur with this condition?

Question 5: What is the appropriate management?

Question 6: What complications should be monitored during treatment and follow-up?

Answer 1: There is a right parietal lobe lesion with surrounding oedema with differentials include primary or secondary brain tumour, abscess and cavernoma.
Other investigations include baseline bloods including CRP, ESR, Blood cultures, tumour markers (CEA, Ca 19-9, Ca 125), further imaging CT TAP and MRI head with contrast.

Answer 2: MRI Head with contrast showed solitary rim enhancing lesion with hypointense rim. Possible differentials include cavernoma, neurocysticercosis, tuberculoma, metastases and brain abscess.

Answer 3: Other investigations include Aquaporin 4 and Anti MOG antibodies, TB blood & urine culture, stool for Cryptosporidium sporidia cyst and ova, Toxoplasma serology, Cryptococcal antigen test, Cysticercosis antigen test. Lumbar puncture after ophthalmology input.

Answer 4: It can lead to seizures, raised intracranial pressure, focal neurological deficits and chronic epilepsy. Ocular cysticercosis can cause visual loss .

Answer 5: Management of cysticercosis includes corticosteroids, anti-epileptics, anti-parasitic medications (albendazole and praziquantel).

Answer 6: Patients with neurocysticercosis should be monitored for treatment related worsening inflammation (around cysts) which could present as worsening seizures or worsening focal neurology. Patients may

have side effects from systemic steroids, hepatotoxicity or low white blood cell count from prolonged antiparasitic medication use, and long-term residual calcified lesions can cause epilepsy.

References:

P. Poeschl, A. Janzen, G. Schuierer, J. Winkler, U. Bogdahn and A. Steinbrecher Calcified Neurocysticercosis Lesions Trigger Symptomatic Inflammation During Antiparasitic Therapy
American Journal of Neuroradiology March 2006, 27 (3) 653-655

Diagnosis: Neurocysticercosis

Case 32

A 67-year-old man was admitted to the emergency department with sudden-onset weakness affecting his left lower limb. He reported an abrupt, painless loss of power in the left leg while walking, which resulted in a fall. This was followed by several more falls over the same day due to persistent leg weakness. He denied any involvement of the upper limbs and reported no bulbar symptoms, respiratory difficulties, sensory disturbance, or sphincter dysfunction. There was no history of recent infection or systemic illness.

His past medical history was significant for previous stroke, transient ischaemic attack, and atrial fibrillation. His regular medications included anticoagulation and secondary stroke prevention therapy. He had been independently mobile prior to this episode. There was no recent change in medication, and no history of similar symptoms in the past.

On neurological examination, upper limb power, tone, reflexes, and sensation were normal. Examination of the lower limbs demonstrated marked proximal weakness, with relatively preserved distal strength. Reflexes were absent bilaterally at the knees and ankles. Sensory examination did not reveal a clear sensory level, and coordination was intact. Cranial nerve examination was unremarkable. Notably, there was marked bilateral lower limb oedema with associated chronic skin changes.

Over the following weeks, the weakness failed to improve and progressively worsened, evolving into bilateral lower limb weakness predominantly affecting the proximal muscles. The patient developed

increasing difficulty climbing stairs and rising from a seated position, preceded by discomfort in the thighs. He remained free of sensory, bulbar, or sphincter symptoms throughout this period.

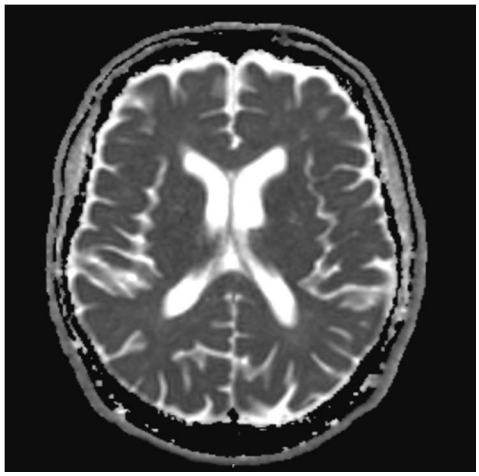


Image 1: MRI Head

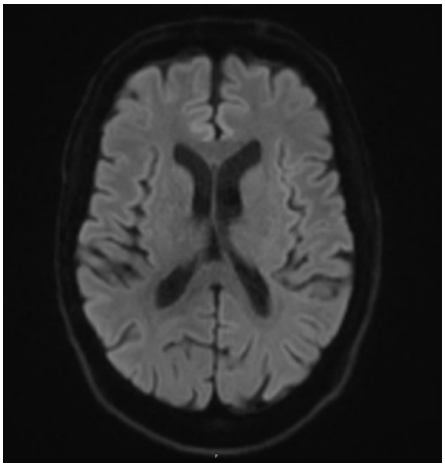


Image 2: MRI Head

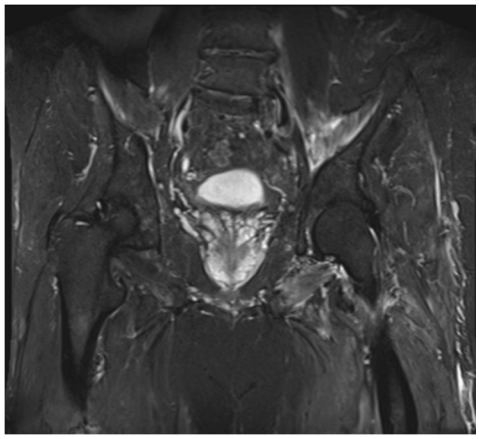


Image 3: MRI pelvis & thigh

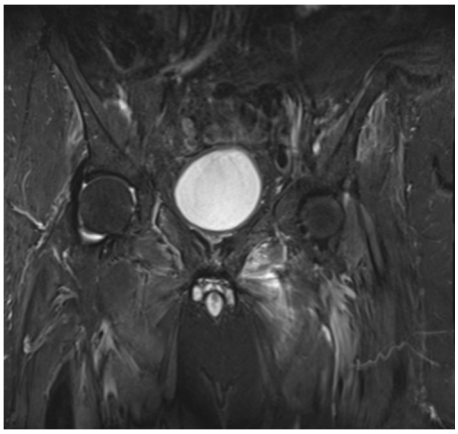


Image 4: MRI pelvis and thigh

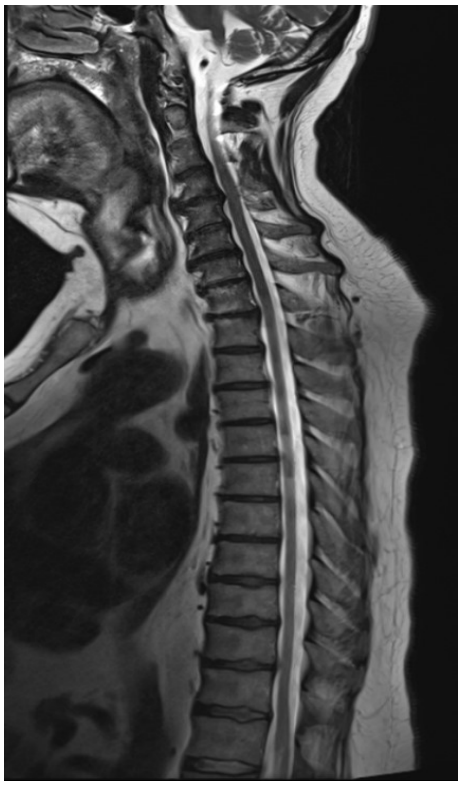


Image 5: MRI cervicothoracic spine

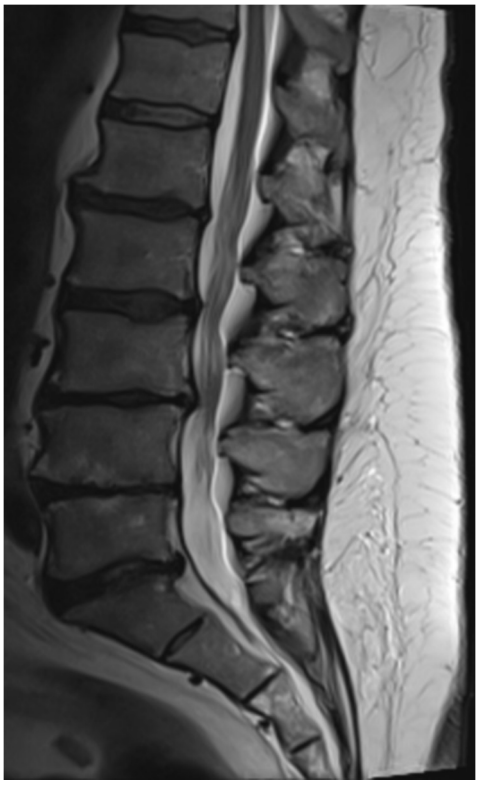


Image 6: MRI lumbosacral spine

Question 1: What initial investigations would you request?

Question 2: What are the findings in the MRI head images?

Question 3: What further investigations would you like to consider?

Question 4: What other radiological investigations would you like to consider?

Question 5: What is your differential diagnosis?

Question 6: How would you like to confirm your diagnosis?

Question 7: What are the treatment options and prognosis?

Answer 1: The initial investigations in this case would include baseline blood tests, CT head, and MRI head scans.

Answer 2: MRI head images show a normal MRI head scan with no restricted diffusion.

Answer 3: Other investigations to consider in this case include Creatine Kinase (CK), ESR, autoantibody screen, serum ACE levels, HIV and syphilis serology, neuroimmunology panel, myositis profile, paraneoplastic antibody screen, HMGCR antibody, EMG and nerve conduction studies (NCS).
We also performed an MRI cervico-thoracic and lumbosacral spine. This revealed non-specific degenerative changes noted with no cord signal changes to suggest cord infarct and no defined areas of any abnormal contrast enhancement. The images are as follows.

Answer 4: In terms of further investigations, a MRI of the lower limbs (thighs) was performed as shown below.
MRI Lower limbs
The MRI scan showed non-specific oedema of the proximal muscle groups suggestive of possible myositis. There was no evidence of fatty degeneration or muscle atrophy.

Answer 5: The differential diagnosis for this case includes myositis, polymyositis, Immune-mediated necrotising myopathy (IMNM) and other myopathies.

Answer 6: The definitive test to confirm the diagnosis would be a muscle biopsy, possibly guided by MRI findings. The image from the muscle biopsy below shows scattered necrotic patches replaced by myophages.

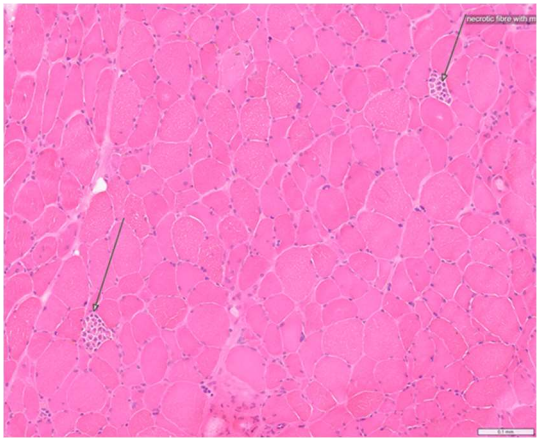


Image 7: Muscle biopsy

Answer 7: There are two main treatment options:

- Intravenous methylprednisolone 500 mg per day for 5 days, followed by a tapering dose of oral prednisolone.
- Intravenous immunoglobulin (IVIG) or targeted immunotherapy such as rituximab.

Prognosis:

- **Anti-SRP IMNM:** Typically, more severe with worse functional outcomes.
- **Anti-HMGCR IMNM:** Generally, responds better to treatment but often requires long-term immunosuppression.

- **Seronegative IMNM:** Response to treatment is less predictable

Diagnosis: Necrotising myopathy mimicking stroke

Case 33

A 92-year-old man was admitted to the emergency department with sudden-onset right-sided weakness. The symptoms developed abruptly and involved both the right arm and leg. He denied headache, loss of consciousness, seizure activity, visual disturbance, or sensory symptoms. There was no preceding trauma or recent illness.

His past medical history was significant for hypertension and hyperlipidaemia. He lived independently with minimal assistance prior to presentation and was able to mobilise without aids. His regular medications included antihypertensive therapy and a statin. He was a non-smoker and did not consume alcohol.

On neurological examination, he was alert and orientated. Cranial nerve examination revealed mild dysarthria without facial asymmetry. Motor examination demonstrated reduced power in the right upper and lower limbs (MRC 4/5), with normal tone. Sensory examination was intact, coordination was preserved, and reflexes were symmetrical. Plantar responses were flexor bilaterally. His National Institutes of Health Stroke Scale (NIHSS) score was calculated as 6.

Baseline blood investigations were unremarkable, including electrolytes, renal function, inflammatory markers, and full blood count (Na 139, eGFR 78, CRP 2, Hb 131, WCC 6.4). An electrocardiogram demonstrated sinus rhythm with no acute ischaemic changes.

A CT and MRI head scan were performed as shown below.

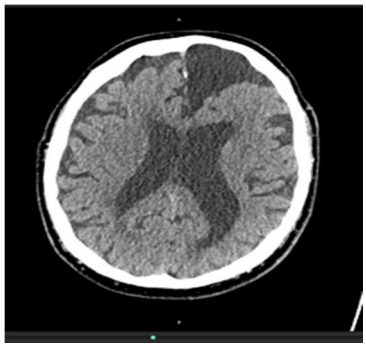


Image 1: CT Head

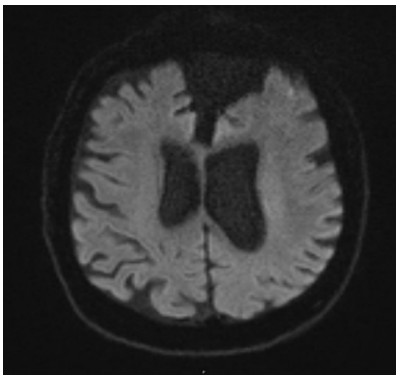


Image 2: MRI Head

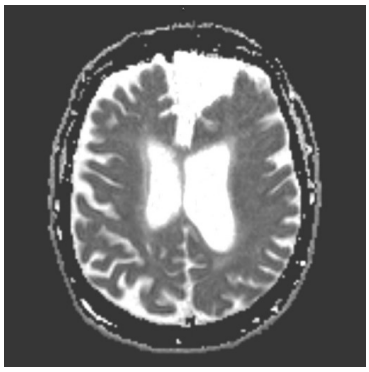


Image 3: MRI Head

Question 1: What abnormality is seen on the CT scan?

Question 2: What is the likely nature of this lesion based on its radiological appearance?

Question 3: What other investigations would you request? Please interpret the MRI findings.

Question 4: What is the appropriate management for this lesion in an elderly patient?

Question 5: What complications can this type of lesion potentially cause?

Answer 1: A large, well-defined hypodense lesion in the left frontal lobe causing mild mass effect, without midline shift.

Answer 2: A benign, CSF-filled cystic lesion, most consistent with an arachnoid cyst.

Answer 3: MRI brain with diffusion-weighted imaging (DWI) shows no restricted diffusion to suggest acute infarction. The arachnoid cyst is well defined, with no evidence of acute infarct on DWI. Other relevant investigations include EEG and fundoscopy. The patient's acute symptoms are due to MRI-confirmed infarcts; the frontal lesion shows no acute changes. This patient's presentation represents a stroke mimic due to mass effect from an arachnoid cyst, leading to transient focal neurological deficits.

Answer 4: Conservative management with close neurological observation and neurosurgical input is appropriate.

Answer 5: Potential complications include progressive mass effect, seizures, headaches, or the development of a subdural hygroma following cyst rupture.

Diagnosis: Arachnoid Cyst

Case 34

A 79-year-old man was admitted to the emergency department with acute-onset confusion and behavioural disturbance that evolved over several minutes. This was accompanied by left-sided weakness and generalized rigidity. Shortly after presentation, his condition deteriorated rapidly with the development of generalized tonic–clonic seizures and a reduced level of consciousness. There was no preceding headache, head trauma, or febrile illness reported.

His past medical history was significant for hypertension, epilepsy, and hypercholesterolaemia. He had been living independently prior to presentation and was cognitively intact at baseline. His regular medications included antihypertensive therapy, antiepileptic medication, and a statin. There was no history of anticoagulant use.

On initial examination, he was confused, agitated, and had reduced responsiveness. Neurological assessment demonstrated left-sided weakness affecting both the upper and lower limbs, with increased tone and generalized rigidity. Cranial nerve examination was limited by poor cooperation but did not reveal clear asymmetry. Reflexes were brisk, and plantar responses were equivocal.

Despite seizure control and supportive management, his neurological status failed to improve significantly. He remained persistently encephalopathic with fluctuating responsiveness and a Glasgow Coma Scale score of 9/15 over the ensuing weeks. Recurrent focal and generalized seizures were noted during his admission, requiring escalation of antiepileptic therapy.

The overall clinical picture was characterised by a rapidly progressive encephalopathy with focal neurological deficits and refractory seizures, in the absence of an alternative systemic or metabolic cause.

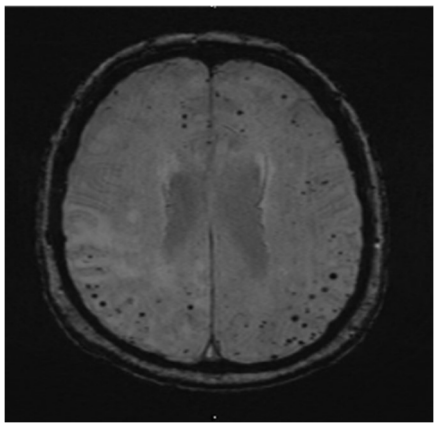


Image 1: MRI Head Pre-Treatment

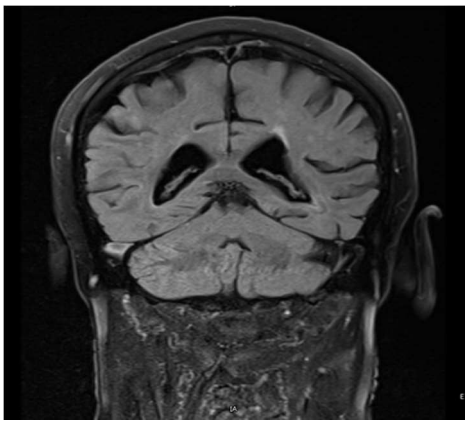


Image 2: MRI Head Pre-Treatment

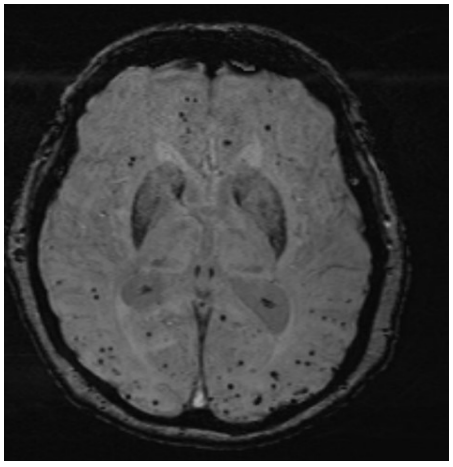


Image 3: MRI Head Post-Treatment

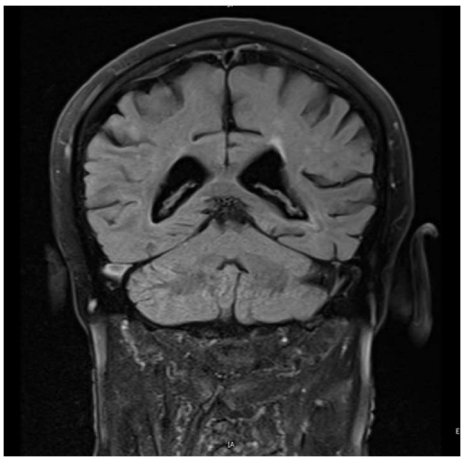


Image 4: MRI Head Post-Treatment

Question 1: What investigations would you consider, and what would be your initial management plan?

Question 2: The patient's initial CT head was unremarkable, and the initial MRI was inconclusive due to motion artefacts. A subsequent MRI was performed a couple of weeks later under sedation. What are the findings seen in the MRI head images?

Question 3: What is the likely diagnosis in this case?

Question 4: What other investigation would you recommend confirming the diagnosis?

Question 5: What is the best management for this patient?

Answer 1: Investigations should include CT head, MRI head, baseline blood tests, lumbar puncture, EEG, neuroimmunology, vasculitis and autoimmune encephalitis screens, as well as HIV and syphilis serology. Empirical treatment for meningoencephalitis should be initiated.

Answer 2: MRI demonstrates subcortical white matter signal abnormality in the right parietal lobe, along with extensive bilateral microhemorrhages, more pronounced in the parietal and occipital lobes. Improvement in the MRI head showed subcortical white matter changes, post steroid therapy.
Answer 3: The diagnosis is inflammatory cerebral amyloid angiopathy. This can be divided into two types: cerebral amyloid angiopathy–related inflammation (CAA-RI) and amyloid β–related angiitis (ABRA).

Answer 4: The gold standard investigation is a brain biopsy, which would demonstrate amyloid deposits (positive on Congo red and anti–beta-amyloid staining) in small blood vessels, surrounded by significant perivascular inflammation (T lymphocytes and macrophages). In this patient, the diagnosis was made based on clinical and radiological features.

Answer 5: Management involves high-dose intravenous corticosteroids followed by a prolonged oral steroid taper (for at least six months). Other immunosuppressive agents, such as cyclophosphamide, may also be considered.

Diagnosis: Inflammatory cerebral amyloid angiopathy

Case 35

An 82-year-old gentleman was admitted to the emergency department with acute-onset confusion and behavioural change, which developed abruptly over the course of an hour. He was noted by a neighbours to be disoriented, repeatedly asking the same questions, and appearing unable to recall recent events. There was no reported headache, loss of consciousness, seizure activity, or preceding trauma. He did not complain of focal limb weakness, visual disturbance, or speech difficulty at the time of presentation.

His past medical history included vitamin B12 deficiency, gastro-esophageal reflux disease, benign prostatic enlargement, previous skin cancer, and possible right knee osteoarthritis. He lived alone and was independent in his activities of daily living. He mobilized independently with the aid of a stick and had been driving prior to admission. He was a non-smoker and did not drink alcohol. His regular medications included vitamin B12 supplementation, a proton pump inhibitor, and treatment for urinary symptoms.

On examination, he was alert but markedly confused, with impaired short-term memory and reduced attention. He was oriented to person but not to time or place. His speech was fluent, with no dysarthria or aphasia. The examination of the Cranial nerves was unremarkable. Motor examination revealed normal tone and strength in all four limbs, with no focal weakness. Sensory examination was intact, coordination was preserved, and reflexes were symmetrical with flexor plantar responses bilaterally. Gait was not assessed initially due to cognitive disturbance.

Baseline blood investigations did not reveal a metabolic or infective cause for his presentation (Na 137, eGFR 81, CRP 1, Hb 129, WCC 6.0). Vitamin B12 levels were within the therapeutic range.

Question 1: What are the findings in the CT head scan?

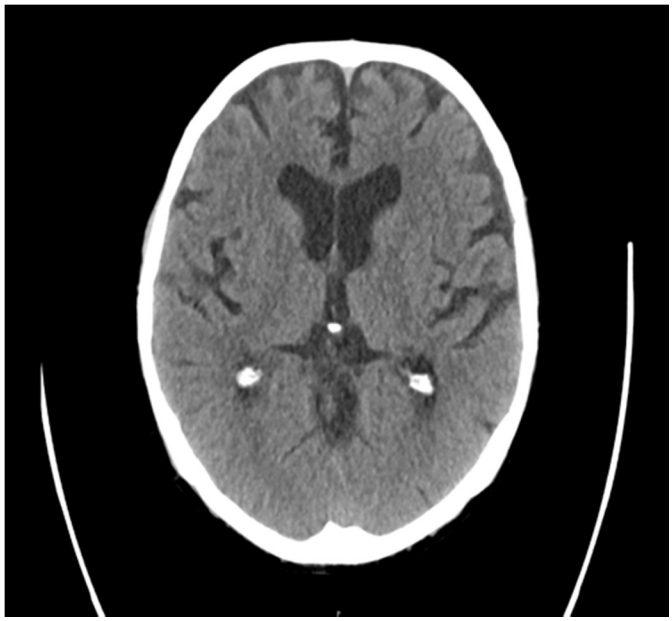


Image 1: CT Head

Question 2: What is the most likely diagnosis, and which further investigations and treatments would you consider?

Question 3: This patient's lumbar puncture results were negative for viral PCR and MC&S, and blood tests including inflammatory markers were normal. The MRI images are as shown below. What abnormality does the MRI demonstrate.

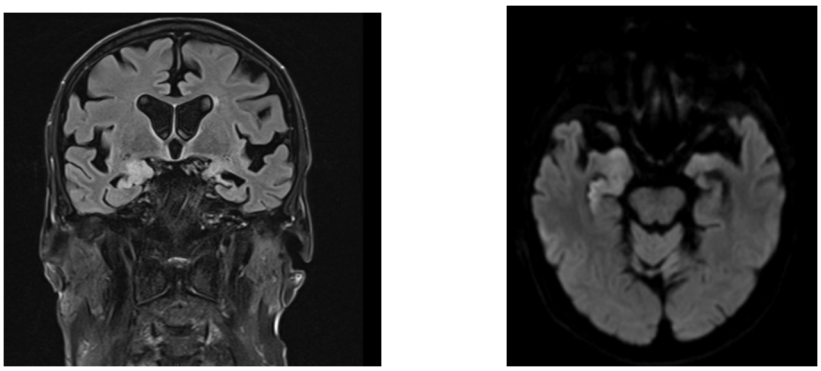


Image 2: MRI Head

Question 4: If this patient's neuroimmunology screen - including CSF NMDA receptor antibodies, LGI1 antibodies, autoimmune encephalitis, and paraneoplastic encephalitis screen – were negative with normal EEG, what are the two most likely diagnoses?

Question 5: List four important differential diagnoses for acute-onset confusion in an elderly patient.

Answer 1: The CT head scan shows age-related changes but no acute or significant abnormalities.

Answer 2: Meningoencephalitis is the most likely differential diagnosis. Recommended management includes empirical treatment with intravenous antibiotics and acyclovir. Further investigations should include lumbar puncture for viral PCR, gram staining, and biochemical analysis, CSF neuroimmunology and serum autoantibody panel screen for autoimmune encephalitis, paraneoplastic antibody screen & EEG.

Answer 3: The MRI head scan shows bilateral hippocampal signal abnormalities (right greater than left), with asymmetrical enlargement of the right medial temporal lobe. Restricted diffusion also seen faintly in the right medial temporal lobe.

Answer 4: Given the normal results of all investigations, a limbic infarct should be considered. Other possible diagnoses include HSV-negative autoimmune encephalitis, such as anti-LGI1 or anti-NMDA receptor encephalitis.

Answer 5: Important differentials for acute confusion in an elderly patient include:

1. Delirium (e.g. infection, dehydration, metabolic disturbance)
2. Transient global amnesia
3. Seizure or post-ictal state
4. Encephalitis

Diagnosis: Limbic infarct mimicking as meningitis

Case 36

An 82-year-old man was admitted to the emergency department with a two-week history of intermittent slurred speech and difficulty swallowing. He reported that the symptoms began following a recent influenza-like illness and sore throat, for which he had taken a short course of oral medication. On the morning of admission, he noticed new left-sided facial droop and difficulty opening his left eye, prompting urgent hospital attendance.

He described episodic deterioration in speech, particularly with prolonged conversation, during which articulation became effortful, and words were difficult to pronounce. Swallowing was described as increasingly difficult rather than painful, with a sensation of food sticking, predominantly on the left side. He denied limb weakness, numbness, visual loss, diplopia, headache, sphincter disturbance, or breathlessness.

His past medical history included ischaemic heart disease and prior antiplatelet-associated bleeding, and he was on clopidogrel. He had been fully independent prior to this illness, with no previous neurological symptoms.

On examination, he was alert and cognitively intact. Speech was mildly dysarthric but fluent. Cranial nerve examination demonstrated left-sided ptosis with impaired eyelid elevation. Facial movements were otherwise symmetrical. Extraocular movements were full. Visual acuity improved when the eyelid was manually elevated. There was no facial sensory deficit. Bulbar examination revealed mild dysarthria without tongue atrophy or fasciculations. Upper and lower limb examination showed normal tone,

bulk, and power throughout, with intact reflexes and sensation. Coordination and gait were normal.

Over the first several days of admission, a consistent temporal pattern emerged. In the mornings, the ptosis was minimal and speech relatively clear. As the day progressed, the left eyelid gradually drooped, speech became increasingly slurred, and swallowing required greater effort. These changes were particularly evident after prolonged talking or eating and during periods of emotional stress. With periods of rest, partial improvement was observed. Limb strength remained normal throughout, and there was no sensory or sphincter involvement.

Speech and language therapy assessment identified dysarthria with variability in clarity and effort, and swallowing was safe but required pacing and compensatory strategies. Ear, nose and throat assessment did not demonstrate any structural abnormality of the upper airway or vocal cords.

Question 1: What do the CT and MRI head images show?

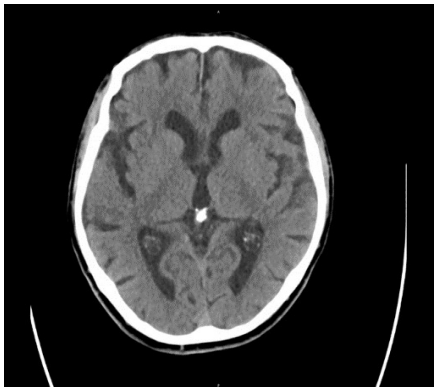


Image 1: CT Head

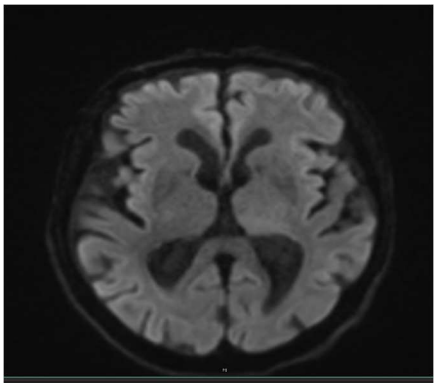


Image 2: MRI Head with DWI sequence

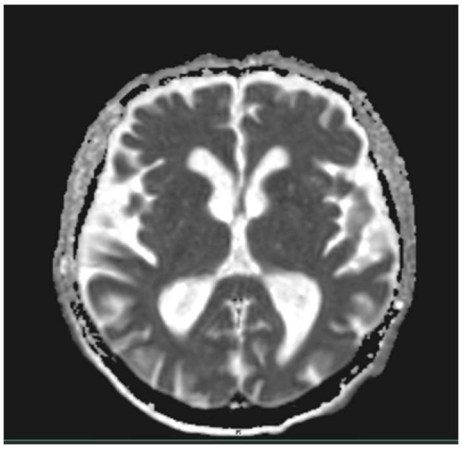
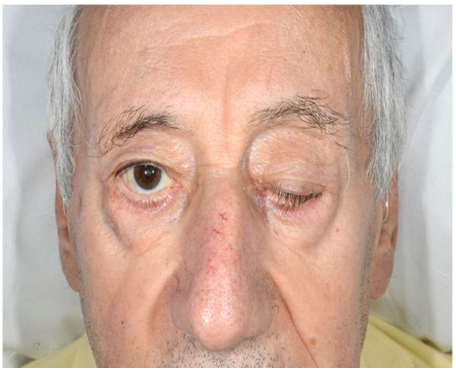


Image 3: MRI Head with ADC sequence



Question 2: What are the differential diagnoses, and how would you investigate further?

Question 3: How is this condition treated?

Question 4: What complications should this patient be monitored for?

Answer 1:

CT head shows chronic small vessel ischemic changes without any major acute territorial infarct, and MRI head shows no foci of restricted diffusion to indicate any acute infarct. The patient was initially treated for suspected ischemic stroke but had persistent dysarthria and swallowing difficulty. Speech and clarity worsened over the days.

Answer 2: Possible differential diagnoses include:

- Myasthenia gravis (ocular or bulbar predominant)
- Brainstem pathology (structural or demyelinating)
- Motor neuron disease
- Neuromuscular junction disorders (e.g., Lambert-Eaton syndrome)

Further investigations may include:

- Tensilon (edrophonium) test, if available
- Trial of pyridostigmine
- Repetitive nerve stimulation
- Single fibre EMG
- Serum acetylcholine receptor (AChR) and MuSK antibodies
- Thyroid function tests and autoimmune antibody screen

Answer 3:

Initial treatment includes pyridostigmine as symptomatic therapy and prednisolone (or other immunosuppressive therapy depending on response and specialist guidance).

Answer 4:

Key complications to monitor include respiratory failure due to bulbar or respiratory muscle involvement. The patient requires close monitoring with serial forced vital capacity (FVC) assessments and consideration for early escalation to high-dependency care if deterioration occurs.

Diagnosis: Ocular Myasthenia Gravis

Index

Alistair J Lewthwaite, Consultant Neurologist, Dudley Group NHS Foundation Trust, March 2026

I would recommend this book of a case series of 36 stroke mimics to both medical and non-medical practitioners of varying grades and experience who are likely to come across strokes and stroke mimics in their daily practice. Indeed the interactive nature of this book in particular will I believe be very helpful in allowing the reader to test and develop their knowledge in the diagnosis and management of stroke mimics. This includes conditions which require very prompt recognition and management in clinical practice and the book also covers a number of rarer stroke mimics which are also important conditions to consider in the correct clinical context as potential differential diagnoses of stroke.

Donna Beckley

Clinical Nurse Specialist: Stroke Medicine, Russells Hall Hospital

The book presents a collection of case studies which enhance critical thinking alongside practical understanding. The book gives a wide selection of patients presenting to an acute hospital with neurological symptoms, with differential diagnosis. Each case is outlined in brief, with excellent use of different imaging modalities. The question-and-answer format enables the reader to focus on key issues in a structured way. This case study book is a valuable and practical resource for learners and healthcare professionals.

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