Neurosurgery

A Case-Based Approach

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The first edition of the Neurosurgery Handover Book is dedicated to our families, who's continuous and loving support has made this book worthwhile.

Handover Framework

Effective handover lies at the very heart of good patient care. (Prof Peter Rubin. GMC)

Good medical handover has gained increasing notoriety as one of the cornerstones of efficient, safe and sage clinical care. With increasing throughput of junior doctors and the evolution of a shift-based hospital staffing system, established techniques for patient handover are mandatory.

Neurosurgical handover is particularly unique as clinical stories evolve rapidly and patients are often unable to communicate for themselves. By the time the patient is referred to the neurosurgeon, the story has often been retold many times and the patient's neurology may have changed. Having a structured approach to telling the patient's story is key. The developing neurosurgeon should be able to quickly decide from the history what the likely diagnosis is/what tests to organise/what treatment the patient may need.

At King's, we are encouraged to use the following headings to structure our handover as will this book:

- Clinical presentation
- Differential diagnosis
- Investigations
- Management
- Evidence
- Outcome

Clinical Presentation

Conveying a clinical presentation can take years of practice. It is an amalgamation of the patient's history, clinical examination, management and current clinical status. It should be delivered in a succinct and confident manner.

There is no 'correct way' to do this, and it will vary between clinical specialties, but from our neurosurgical experience, we recommend the following structure:

Introduce the Patient

When making decisions in neurosurgery, having a good understanding of the premorbid state of the patient is crucial. When presenting a case, starting with a description of the patient hooks your listener in and allows them to create a visual image of the person you are describing. Whilst we do not encourage prejudice and type casting, medicine is often about pattern recognition, so putting patients into groups dependent on their background can be very useful.

Things to include are:

(a) **Age**

Neurosurgical problems affect patients of all age groups. Knowing a patients age is diagnostically relevant as certain problems affect different stages of life (Table 1). Age also in part determines prognosis.

For example a primary lobar brain haemorrhage in a 27-year-old is more likely to be caused by an underlying arteriovenous malformation than in an 87-year-old where it is more likely secondary to cerebral amyloid angiopathy. And a posterior fossa mass in a 4-year-old is more likely a primary brain tumour, whereas in a 64-year-old, it is more likely a secondary metastatic brain deposit.

(b) **Sex**

Most neurosurgical problems are gender neutral. Certain problems, however, can show a sex preponderance (i.e. cauda equina syndrome from lumbar disc prolapse is more common in young women). Knowing the sex of your patient will also help your bed manager assign the appropriate ward for your patient.

(c) **Hand dominance**

Establishing hand dominance is tradition in neurology. It aims to assert which hemisphere is 'dominant' for speech. In reality, most people display left hemisphere

Table 1 Age related neurosurgical problems

Age	Problems
0–1	Spinal dysraphism, hydrocephalus, prematurity related intraventricular haemorrhage
1–18	Primary posterior fossa brain tumours, intra-cranial infection, shunt problems
18–50	Cauda Equina Syndrome, traumatic brain injury, spinal trauma, primary brain tumours, intra-cranial infections, aneurysmal sub-arachnoid haemorrhage
50–70	Haemorrhagic hypertensive stroke, aneurysmal sub-arachnoid haemorrhage, degenerative radioculopahthies/myelopathy, primary brain tumours, secondary brain and spinal tumors
>70	Chronic Sub-dural haematoma, Cerebral Amyloid angiopathy, Hypertensive haemorrgahic stroke, osteoporotic spinal compression fractures, degenerative radioculopahthies / myelopathy, primary brain tumours, secondary brain and spinal tumours, normal pressure hydrocephalus

dominance for speech (88% of right handers and 78% of left handers). Approximately 12% of right handers and 15% of left handers are co-dominant for speech, and only 7% of left handers are truly right hemisphere dominant. Why mention it then? Well it shows you have thought about the patient's individual wiring, and on rare occasion, when you find a right dominant left handed with right hemispheric pathology and speech dysfunction, you will be pleased you asked.

(d) **Profession**

This is an often overlooked part of the medical history and patient description. Knowing your patient's profession gives you an idea about their daily life, cognitive abilities and how the problem they have may affect them. Probe deeply to get an exact picture of what their job entails. If a patient tells you they are an engineer, explore what that means to them—in some cases, they will be a car mechanic, in others they will work for the design team at Tesla. If your patient is unemployed, find out what they do during the day. If they use illicit drugs, this should be mentioned due to the comorbidities associated with this. Without asserting prejudice, categorising patients into groups assists in patient management.

(e) **Independence**

The functional baseline of your patient is an extremely relevant part of the neurosurgical history. It paints an immediate description of a patient's rehabilitation potential which is vital in neurosurgical illness which is often associated with significant disability.

(f) **Significant comorbidities/relevant drug history**

Comorbidities and current drug history affect prognosis and also influence surgical decision-making. For example, if a patient has a chronic subdural haematoma which needs surgical management and is on Warfarin, this will need to be reversed prior to surgery and for a period after. The reasons for the anticoagulation should be highlighted—if it is prophylactic for a history of atrial fibrillation, less consideration for stopping/restarting it will be needed than if they have an active pulmonary embolism. In a presenting summary, not all drugs need to be listed just the most neurosurgically relevant ones e.g. anticoagulants.

In order to save time (and words on the handover sheet), you can abbreviate, and in one succinct line, set the scene and allow your audience to visualise the patient such as the following:

Example 1, '77M RH Retired Postman, Independent, PMhx Htn, CKD, On aspirin…'

Example 2, '44F LH IVDU, NFA, unemployed. Pmhx Hep C, IE, on methadone…'

Example 3, '5M RH, NVD at term, development normal, lives with cohabiting parents, no pmhx…'

Example 4, '27F RH lawyer, obese, Pmhx LBP…'

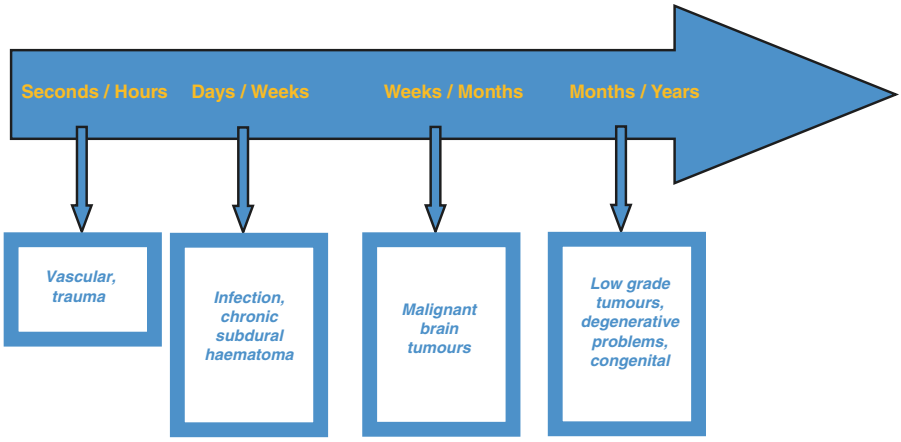


Fig. 1 Temporal association of neurosurgical conditions

Abbreviations are an important part of written handover. Common neurosurgical abbreviations can be found at the start of this book.

Describe the Problem

This comes with practice and knowledge about what to look for. Broadly speaking, neurosurgical problems either occur in the brain or the spine, and they are caused by infection, tumours, degenerative changes, vascular problems, congenital problems and trauma. Some basic knowledge of brain/spine functional anatomy can be key to localising the lesion. The time scale of symptoms/presence of other constitutional symptoms/comorbidities can be helpful to decipher what the lesion is (Fig. 1).

The description of the problem at handover should paint a picture of **where the lesion is**, **what the lesion is** and **how unwell the patient is.** You need to have taken the history and examined the patient yourself in order to do this correctly.

Where Is the Lesion?

A good neurosurgical history/examination should give the clinician a very good idea about where the problem is before any tests have been reviewed. Neurosurgical symptoms can also be broadly grouped into pain and neurological deficit—detail about both gives clues to the diagnosis.

Pain: in the brain, this largely presents as headache. There are many different features of headache, and any red flag symptoms should be highlighted in the case presentation (Fig. 2). In the spine, pain can present as back pain or radicular pain. Similarly red flag symptoms should be mentioned (Fig. 3).

Neurological deficit: neurological deficit can help locate the area affected by the neurosurgical problem. The experienced neurosurgeon should be able to locate very accurately the location of a lesion based on the history and examination before looking at any neuroimaging. Neurological deficit should be described in terms of 'global deficit' and 'focal deficit'.

Fig. 2 Red flag symptoms of headache

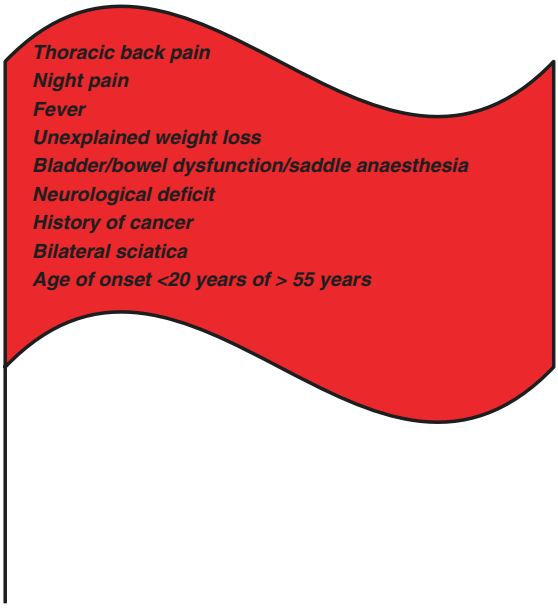


Fig. 3 Red flag symptoms of back pain

Global Neurological Deficit

In the brain, one should first consider global brain function which is most easily assessed by the patient's conscious level. Many scoring systems are described, but the most common, universally adopted, is the Glasgow Coma Scale (Fig. 4). This should be communicated according to individual component score in words as

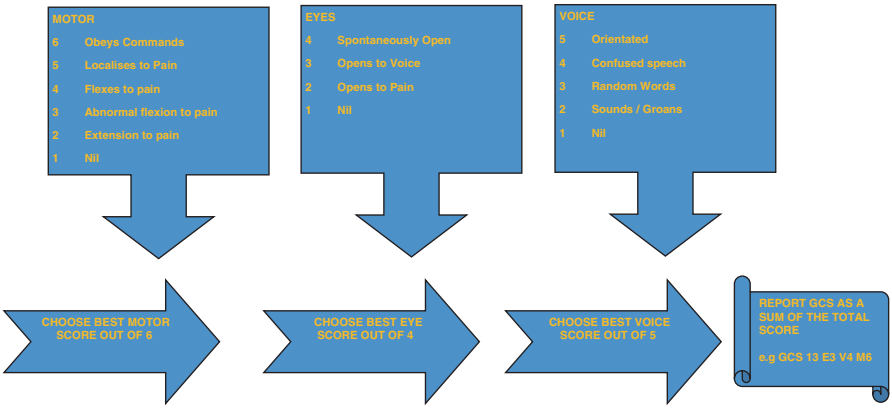


Fig. 4 Glasgow coma scale

opposed to total score. We encourage the GCS to be reported as words rather than numbers as they are a far better descriptive tool of a patient's conscious level, e.g. the patient was localising to pain (M5), eyes open to speech (E3) and groaning (V2) with a total GCS of 10.

One of the main reasons to break the GCS down into component parts is that neurosurgeons tend to place the most weight on the motor score. A patient with a GCS of 10 who is localising to pain (M5), groaning (V2) and eyes opening to voice E3 could score the same as a patient who was obeying commands (M6), aphasic (V1) and eyes opening to voice (E3). The first patient would elicit more concern from the experienced neurosurgeon due to the diminished motor score which implies a severe problem with conscious level. The second patient is more likely to be fully conscious with a focal neurological deficit (i.e. left frontal stroke affecting speech production) which is a different concern. Learning the GCS as a descriptive tool as opposed to numerical scoring system makes it more user-friendly, a better descriptive tool and will please the older generation of neurosurgeon.

If a patient has a reduced conscious level, pupillary response should be reported. Pupils should be assessed for size, response to light and symmetry. They should be assessed regularly and any changes noted alongside the temporal sequence of this (Fig. 5).

Focal Neurological Deficit

Brain

A focal neurological deficit can be very helpful in localising a brain lesion. During a case presentation positive neurology should be described, repeating the entire findings of your neurological examination will send your listener to sleep! Understanding the functionality of the different areas of the brain is useful in interpreting the pattern of symptoms.

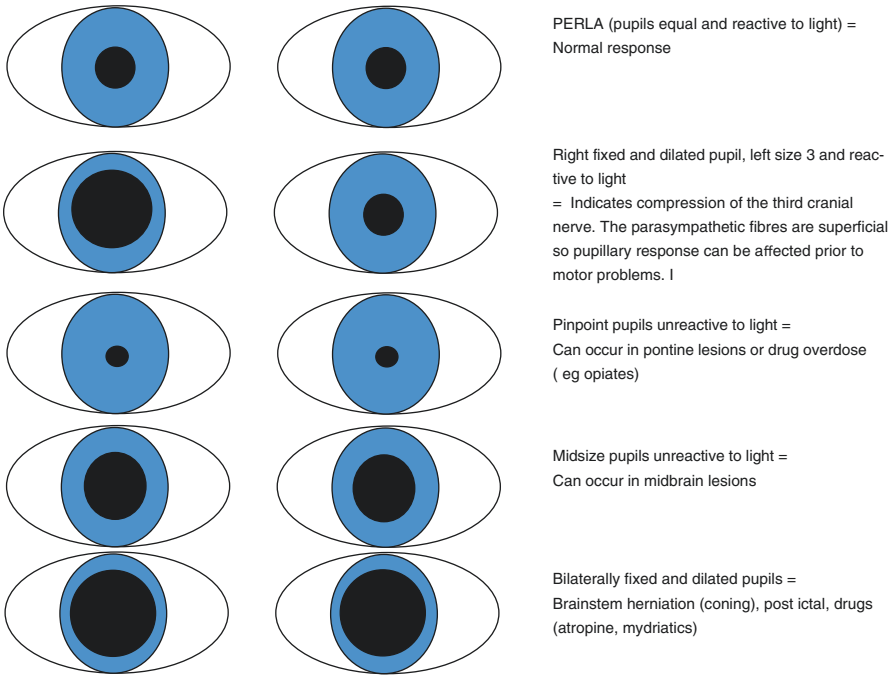


Fig. 5 Pupillary response

Lateralising a lesion in the brain follows these basic principles. The right brain serves the left side of the body and vice versa with the exception of the cerebellum and cranial nerves which have ipsilateral control.

Broadly speaking, there are five lobes of the cerebrum: frontal, temporal, parietal, occipital and cerebellar. Laterality considered, symptoms should be localizable if there is lobar pathology particularly if 'eloquent cortex is affected' (Figs. 6 and 7). Eloquent cortex describes areas of the brain that if lesioned/removed will produce a distinct deficit e.g. left frontal lobe pathology can cause an expressive dysphasia and right occipital lobe lesion will produce a left homonymous hemianopia. Certain areas of the brain (right frontal lobe, right anterior temporal lobe) are described as 'non-eloquent' or 'silent'—this does not mean that they do not serve a function, rather that if they are lesioned, neurology may be too subtle to detect.

Other areas of the brain which can produce classic focal neurological symptoms include the pineal region, suprasellar region, cerebellar-pontine angle and brainstem.

Pineal region lesions are associated with Parinaud's syndrome due to compression on the midbrain/tectal plate. Symptoms include vertical gaze palsy with loss of upgaze (sunsetting eyes), nystagmus retractoris, eyelid retraction and pseudo-Argyll Robertson pupillary response (accommodates but does not react to light).

Suprasellar region lesions are associated with deficits to the optic nerve and cranial nerves that run in the cavernous sinus (III, IV, V, VI) causing visual field loss

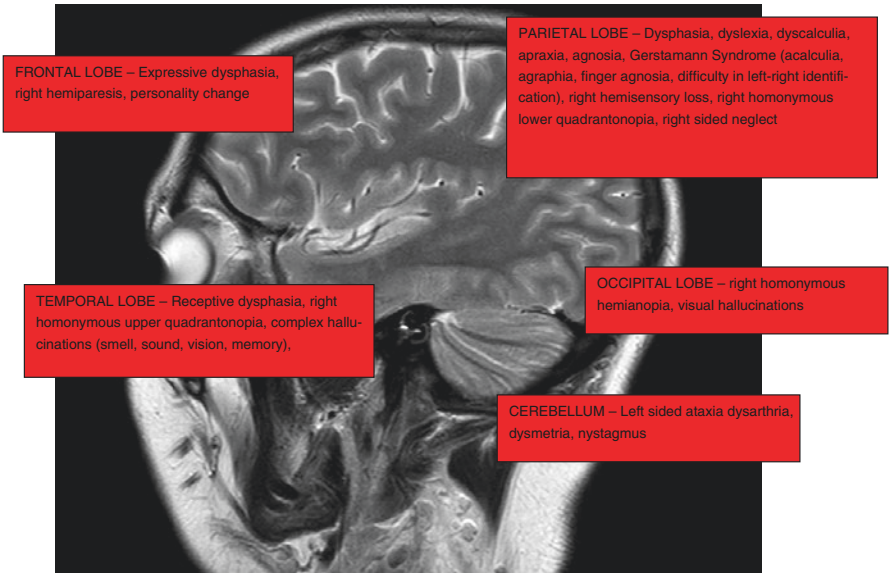


Fig. 6 Symptoms of Left lobar hemisphere dysfunction

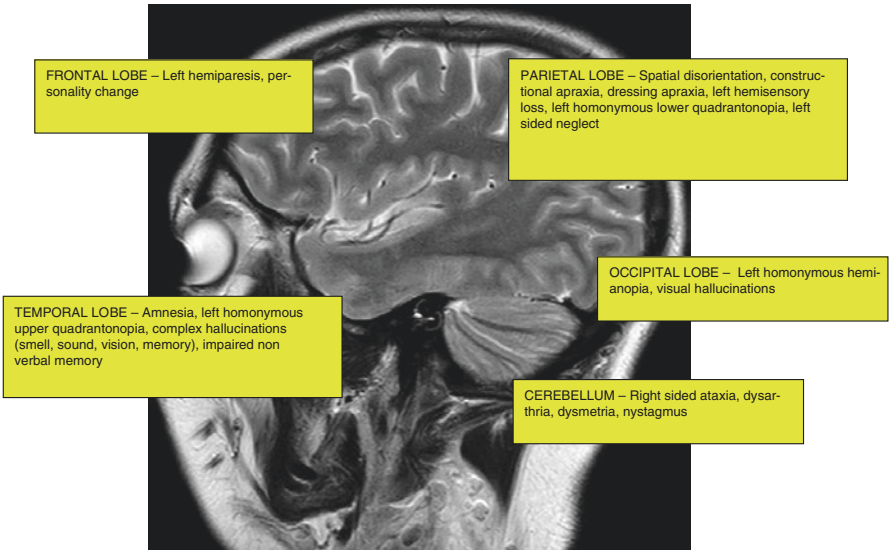


Fig. 7 Symptoms of right lobar hemisphere dysfunction

(typically tunnel vision or bitemporal hemianopia) and diplopia (ophthalmoplegia) with or without endocrinological dysfunction.

Cerebellopontine angle lesions may result in lower cranial neuropathies causing disordered swallowing, facial weakness/hemifacial spasm and hearing loss.

Table 2 Cranial nerve dysfunction

	Nerve	Nucleus	Location of CN nucleus	Symptoms of cranial nerve dysfunction	Neurosurgical conditions which may cause cranial nerve dysfunction
I	Olfactory	Olfactory Bulb	Anterior skull base	Loss of smell	Olfactory groove meningioma, traumatic brain injuries
II	Optic	Lateral geniculate nucleus	Thalamus	Visual changes	Pituitary tumours/Supra-sellar lesions,
III	Oculomotor	Oculomotor nucleus	Midbrain	Ipsilateral eye looks down and out.	Posterior Communicating Artery Aneurysm
		Edinger-Westphal nucleus		Ptosis (drooping eye lid)	Cavernous sinus lesions
				Pupil dilated and unresponsive to light	
IV	Trochlear	Trochlear nucleus	Midbrain	Diplopia, noticeable when walking downstairs	Traumatic brain injuries
V	Trigeminal	Trigeminal nerve nuclei:	Midbrain, pons, medulla, high cervical cord	Ipsilateral change in sensation in face.	Lesions in the cavernous sinus
		Mesencepahlic nucleus		Loss of corneal reflex	
		Principal sensory nucleus			
		Spinal trigeminal nucleus			
		Trigeminal motor nucleus			
VI	Abducens	Abducens nucleus	Pons (floor of 4th ventricle)	Ipsilateral lateral rectus palsy, eye looks medially	Raised intra-cranial pressure (false localising sign)
VII	Facial	Facial motor nucleus	Pons, medulla	Ipsilateral facial weakness (forehead sparing)	Lesions in the Cerebelllo-pontine angle (e.g., acoustic schwannoma)
		Superior salivatory nucleus		Ipsilateral loss of taste on anterior 2/3 of tongue	
		Solitary nucleus		Changes in hearing	
VIII	Vestibulo-cochlear	Vestibular nuclei	Pons, medulla	Ipsilateral sensorineural deafness	
		Cochlear nucleus			

(continued)

Table 2 (continued)

	Nerve	Nucleus	Location of CN nucleus	Symptoms of cranial nerve dysfunction	Neurosurgical conditions which may cause cranial nerve dysfunction
IX	Glossopharyngeal	Solitary nucleus	Pons, medulla, high cervical cord	Ipsilateral reduced gag reflex	Foramen magnum lesions
		Spinal nucleus of trigeminal nerve		Ipsilateral loss of taste on posterior 1/3 of tongue	
		Lateral nucleus of vagal trigone			
		Nucleus ambiguus			
		Inferior salivatory nucleus			
X	Vagus	Dorsal nucleus of vagus nerve	Medulla, high cervical cord	Ipsilateral paralysis of larynx and soft palate	
		Nucleus ambiguus			
		Solitary nucleus			
		Spinal trigeminal nucleus			
XI	Accessory	Spinal accessory nucleus	Medulla, high cervical cord	Ipsilateral weakness of neck turning and shoulder shrugging	
		Nucleus ambiguous			
XII		Hypoglossal nucleus	Medulla	Ipsilateral flaccid tongue weakness	

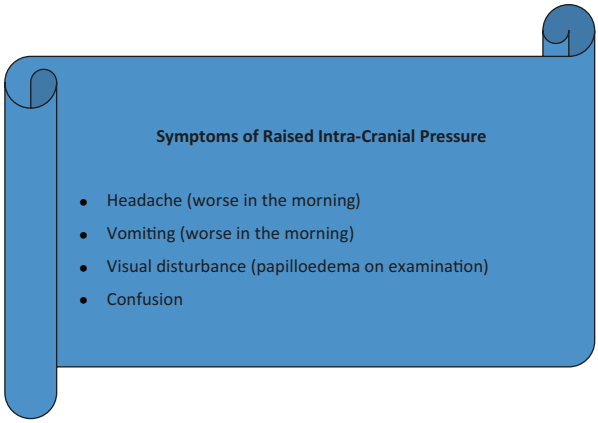


Fig. 8 Symptoms of raised intracranial pressure

Brainstem lesions can cause a myriad of focal and global deficits dependent on the tracts/nuclei affected. When a patient presents with a combination of peripheral weakness and cranial neuropathies, a brainstem lesion should be considered.

Clinical history should indicate if there is a potential cranial neuropathy, e.g. diplopia, and examination be used to confirm which nerve is affected (Table 2). More than one nerve may be affected. If cranial neuropathies are present, one should consider pathology in the cranial nerve nuclei which are located in the thalamus, midbrain, pons, medulla and high cervical cord (Table 2).

As described previously, there are certain areas in the brain which are 'silent' meaning lesions to them will not produce a focal neurological deficit. The ventricular system is one such 'silent' area, so pathology affecting this area can be subtle and difficult to localise. Patients with ventricular disease often present with symptoms of raised intracranial pressure (Fig. 8) secondary to hydrocephalus.

A false localising sign occurs when pathology discrete from the area affected produces a neurological sign which may be wrongly located. The most commonly appreciated 'false localising sign' is the CN VI palsy in the context of raised intracranial pressure. The VI nerve takes a steep perpendicular trajectory when it leaves the pons on its course to the cavernous sinus. By doing so, it is left vulnerable to pressure changes in the brain which can cause traction on the nerve and thus dysfunction. It is not uncommon for shunt-dependent child to present with a CN VI palsy in the context of a blocked shunt and raised ICP.

Spine

Spinal lesions should be considered/described under the following determinants:

1. Is it an upper/lower motor neuron problem? (Fig. 9) This will help you decide whether the problem is in the spinal cord or cauda equina/peripheral nerves.
2. What level is the injury? Knowledge of whether it is UMN or LMN should help you determine this. Try and give a motor and sensory level if present. Knowledge of motor supply to key motor groups (Table 3) is important, and muscle strength should be graded using the MRC grading system (Table 5). Sensory level should be given

Upper Motor Neurone = Lesions are above the anterior horn cell (spinal cord, brainstem, motor cortex)

- **Increased tone / spasticity / sustained clonus**
- **Pyramidal pattern of weakness (extensors weaker than flexors in the arms and in reverse in the legs)**
- **brisk reflexes, up going plantars**

Lower Motor Neurone = lesion is in the anterior horn cell or distal to it (root, nerve, neuro-muscular junction)

- **Reduced tone / flaccidity**
- **Muscle wasting / fasiculations**
- **Loss of reflexes**
- **Muscle**

Fig. 9 Upper vs Lower Motor Neurone Pathology

Table 3 Myotomes to determine motor level

Root	Muscle group
C5	Elbow flexion
C6	Wrist extension
C7	Elbow extension
C8	Finger flexion
T1	Little finger abduction
L2	Hip flexion
L3	Knee extension
	Ankle dorsiflexion
L5	Long toe extensors
S1	Ankle plantar-flexion
S4-5	Voluntary anal contraction

Table 4 Roots of spinal reflexes

Reflex	Root
Biceps	C5,6
Triceps	C6,7
Supinators	C5, 6, 7
Knee	L3
Ankle	S1

by dermatomal level of lowest normal sensation (Fig. 10), and proprioception should be reported if abnormal. In the case of spinal cord injuries, an ASIA (American Spinal Injury Association) grade should be assigned (Table 6 and Fig. 10).

3. Sacral segments—bladder and bowel dysfunction can occur in both spinal cord and cauda equina pathology. Good examination and history taking will help to

Table 5 MRC scale for muscle power

5	Active movement, full range of motion against
4	Active movement, full range of motion against
3	Active movement, full range of motion against
2	Active movement, full range of movement with
1	Visible or palpable contraction
0	Total paralysis

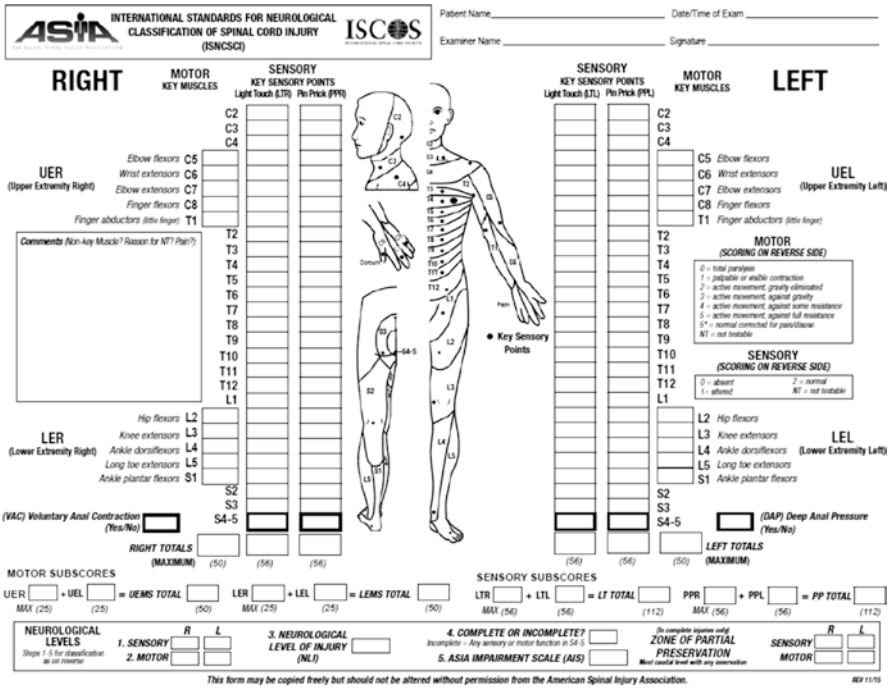


Fig. 10 ASIA Scoring for spinal cord injury

Table 6 ASIA grade of spinal cord injury

ASIA grade	Type of injury	Description
A	Complete	No motor function below the level of the injury. No sensory function is pre-served in the sacral segments S4-5
B	Incomplete	Sensory but no motor function is pre-served below the neurological injury and includes sacral segments S4-5
C	Incomplete	Motor function is preserved below the injury and more than half of the key muscles have an MRC grade of less than 3 OR voluntary anal contraction is present
D	Incomplete	Motor function is preserved below the neurological level and at least half of the key muscles have and MRC grade of 3 or more
E	Incomplete	Motor and sensory function are normal

distinguish. Cauda equina syndrome (CES) is a medical emergency and when complete involves insensate and painless urinary retention, perineal sensory loss in a saddle distribution, altered anal tone with faecal incontinence and loss of sexual function. It often occurs alongside bilateral sciatica and lower back pain and can be associated with lower limb paraesthesia/motor weakness. Most patients do not present with the 'full house' of symptoms and can be referred to as having an 'incomplete' syndrome. Both complete and incomplete CES should be managed with the same degree of clinical urgency with appropriate investigations/treatment. When presenting cases of CES, it is crucial to have good grasp on the chronology of symptoms/deficits including when and how the patient sought medical attention. Given the devastating consequences of untreated CES and how highly charged it is in a medico-legal context, excellent documentation and handover is essential.

Conus medullaris syndrome is associated with pathology to the caudal cord and lumbar nerve roots at the L1/L2 level leading to a combination of upper and lower motor neuron signs/symptoms. This includes insensate painless urinary retention, saddle distribution of perineal numbness, faecal incontinence and sexual dysfunction. It is less often associated with sciatica or motor/sensory dysfunction in the lower limbs, it is more likely to be symmetrical and patients may have upper motor neuron signs (i.e. spasticity, hyperreflexia).

4. Spinal cord syndromes
   Having a basic knowledge of spinal cord anatomy will help ascertain if a spinal cord syndrome is present (Fig. 11).

What Is the Lesion?

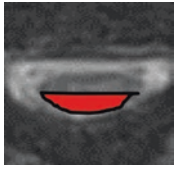
Localising the lesion should give you an idea what the lesion might be by understanding the types of pathology that you might find in that area of the central nervous system. Another crucial consideration is the temporal relationship of symptoms to presentation which is often the key indicator to the differential diagnosis (Fig. 1). Care should be taken to communicate when and how (suddenly or gradually) symptoms began.

Other factors that will help you decide what the lesion is will have been mentioned in your patient introduction (age, profession, comorbidities, drug history).

Care should be taken to mention if the patient has any constitutional symptoms (weight loss, fever, malaise, septic symptoms). If the patient is well aside the neurological presentation, this can be summarised as 'the patient is otherwise fit and well'.

How Unwell Is the Patient?

If you have described the patient as suggested above, you will have painted a picture of what the patient is normally like, what they are currently like and how quickly any symptoms are evolving. This will give your audience a picture of the severity of the neurosurgical problem.

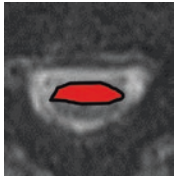


Posterior Cord Syndrome:

Affects the dorsal columns (vibration sense and proprioception),

Symptoms: loss of vibration sense and proprioception

Causes: cervical myelopathy, epidural spinal metastasis, MS

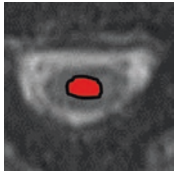


Anterior Cord Syndrome:

Affects bilateral anterior + lateral spinothalamic tract and corticospinal tract

Symptoms: bilateral loss of motor function, pain and temperature sensation below the lesion but preservation of dorsal column function

Causes: flexion injury , injuries to the anterior spinal artery

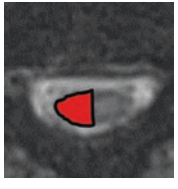


Central Cord Syndrome:

Affects: medial spinothalamic/corticospinal and anterior dorsal columns

Symptoms: motor + sensory dysfunction upper limb > lower limb

Causes: central cord injures result from a vascular watershed area e.g hyperextension injury in the elderly with a background of cervical stenosis—

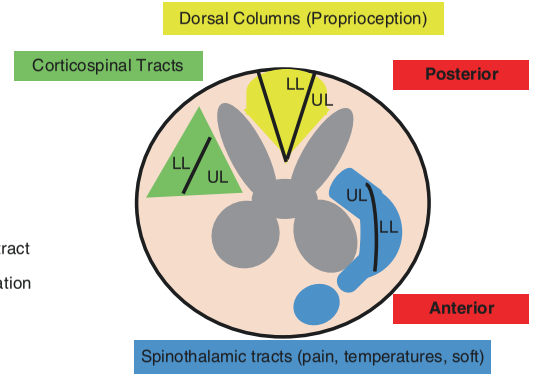


Cord Hemisection / Brown Sequard Syndrome:

Affects: unilateral dorsal column, spinothalamic and corticospinal tracts

Symptoms: ipsilateral loss of vibration/proprioception/motor. Contralateral pain and temperature loss

Causes: stab injuries, thoracic disc herniation



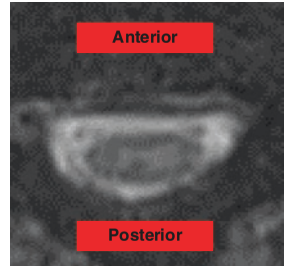


Fig. 11 Spinal anatomy and cord syndromes

Differential Diagnosis

In the King's handover meeting, once the clinical presentation has been given, we consider as a group the possible differential diagnoses for the case and discuss rationale for the hypothesis. Using the example from our introduction,

'51M RH builder, co-habits with his wife and children. Witnessed collapse on pavement, no obvious head injury. GCS 8 when LAS arrived (M5, V2, E1). En route to hospital fixed and dilated R pupil. GCS 6 on arrival (M4, V1, E1). I+V + mannitol in A+E, R pupil remained fixed.'

Differential Diagnosis

- Given the sudden onset nature of his symptoms, this is most likely a vascular event. Possible explanations include subarachnoid haemorrhage, intra-parenchymal haemorrhage and bleed into an underlying tumour.
- The presence of the unilaterally fixed and dilated right pupil would imply predominantly right hemispheric pathology with possible third nerve compression from uncal herniation or compression elsewhere on the nerve. In this case, if the explanation is a subarachnoid haemorrhage, one could expect a posterior communicating artery aneurysm to have ruptured causing a third nerve palsy or causing a subdural haematoma with mass effect and uncal herniation. If the patient has an intra-parenchymal haemorrhage, it would be located in the right hemisphere (likely fronto-temporal) causing uncal herniation.

Investigations

Once the clinical presentation and differential diagnoses have been considered, the appropriate modality for investigation can be considered. In a neurosurgical setting, this usually involves imaging—most often CT and MRI. Trainee neurosurgeons will quickly become adept at reviewing and presenting scans.

Having an idea of the appropriate line of investigation is the starting point to being able to interpret them correctly, and we will use this book to guide you to making the correct choices for your patient.

The following principles should help guide you:

Neuroimaging

Brain Imaging

Computed Tomography (CT) Head

This is the most commonly used investigation in the acute setting. It is quick to acquire, readily available and produces imaging which highlights gross brain

pathology and acute injuries. It is also the investigation of choice for assessing bony structures (i.e. skull base).

Scans are normally performed without contrast, but in the case of certain suspected pathology (brain tumour, intracranial infection), you may also want to request pre- and post-contrast imaging.

Magnetic Resonance Imaging (MRI) Head

MRI is able to produce greater detail and definition of the brain than CT. It takes longer to acquire the imaging and MRI scanners are often not readily available out of hours. This would be the investigation of choice usually after a CT head has shown intracranial pathology which you want more detailed imaging of, e.g. brain tumours, intra-cerebral infection and hydrocephalus. Patients who need repeated imaging are served better with MRI as there is no radiation dose associated unlike CT. This is particularly applicable to children who are more vulnerable from the effects of imaging-associated radiation.

Different MRI sequences are used to highlight different types of pathology. Generally speaking most brain MRIs will include T2WI, T1WI, FLAIR, DWI, ADC, GRE and T1 post-contrast sequencing.

Image Guidance MRI (or CT) can be loaded into an image guidance machine and used intraoperatively as brain navigation. If an Image guidance scan is desired, the radiologists will need to be informed as it has to include more cuts of the patient's face in order to ensure the scan can be accurately registered to the patient.

In infants, MRI poses a particular challenge as the scans are long and require still, compliant patients. In children, they often require a general anaesthetic which can be difficult to organise during a busy on call. If the child is a neonate, a technique known as 'feed and wrap' can be attempted. This involves feeding the baby and then swaddling them to encourage sleep; the baby is then quickly placed in the MRI machine and the imaging attempted. It can be hit and miss and would be suboptimal for acquiring Image guidance scans (where high quality is key to ensure appropriate correlation with real brain anatomy).

Angiography

Angiography is used to look at the cerebral blood vessels. Angiograms can be acquired by CT, MRI and catheter angiogram.

- CT angiography: this is used in the first instance to look for aneurysms in patients with SAH. It is quick to acquire but requires a skilled neuro-radiographer to interpret the imaging.
- MR angiography: this is often the imaging modality used to follow up patients with known neurovascular pathology, e.g. surveillance for coiled aneurysms and surveillance of brain arteriovenous malformations.
- Digital subtraction angiography: this is the gold standard investigation for aneurysmal subarachnoid haemorrhage and a dynamic form of imaging which is very useful in the case of vascular problems of the brain and spinal cord like cerebral aneurysms, fistulas and arteriovenous malformations. It is performed by neuro-radiologists via catheter angiogram and is associated with a small risk of stroke (0.5–1%).

Spine Imaging

Computed Tomography (CT) Spine

This is the investigation of choice in the context of trauma given the excellent visualisation of bony structures and ease of acquisition. Spinal imaging can be divided into the region of interest, e.g. lumbar/thoracic/cervical. In the context of major trauma where mechanism of injury is significant and the patient may have distracting injuries, the whole spine is commonly imaged.

Magnetic Resonance Imaging (MRI) Spine

If spinal cord/cauda equina/nerve root pathology is suspected, MRI is the investigation of choice given its superiority for soft tissue imaging. Urgent MRI spine is more commonly performed than MRI brain in the neurosurgical context. It is the investigation of choice for suspected cauda equina syndrome/metastatic spinal cord compression/traumatic spinal cord injury. Non-neurosurgical units often will not offer an out-of-hours MRI service, so patients who require emergent MRI imaging may have to be transferred to the neurosurgical centre.

The region of the spine that should be scanned again depends on the patient's clinical presentation and the differential diagnosis. In the case of suspected cauda equina syndrome, a lumbar spine MRI will suffice; if you are concerned the patient has metastatic spinal cord compression, a whole spine MRI would be required.

Spinal MRI sequences usually include T2, T1 and STIR. Contrast imaging is usually only indicated in the context of suspected spinal cord tumours.

Plain X-Rays

It is rare that neurosurgeons request spinal X-rays. In the context of investigation for acute bony injury following major trauma, CT is the usual modality of choice.

Dynamic spinal X-rays, e.g. standing lumbar X-rays or cervical spine flexion extension X-rays, may be useful in assessing the stability of a fracture.

Bedside Investigations

There is occasion when neuroimaging will not offer all the answers.

Lumbar Puncture

The lumbar puncture can be used to assist in the diagnosis of a number of neurosurgical problems. It can also be used therapeutically.

1. Aneurysmal subarachnoid haemorrhage

The initial investigation of choice for a patient with suspected SAH is plain CT head. When carried out within 24 h of symptom onset, CT is 95% sensitive for SAH which falls to 80% at D3 and 50% at 1w. If a CT does not show subarachnoid blood, a lumbar puncture should be performed 12 h after the onset of symptoms. The LP

should be performed by an experienced clinician to mitigate against traumatic tap which can confuse results. Opening pressure should be recorded (normal in an adult 10–20 mmHg). Three serially numbered bottles with c 20 drops of CSF in each should be taken and transported to the lab in an opaque container to prevent photodegradation of red blood cells which may lead to a false-positive test. CSF should be inspected and appearance described (bloody, turbid, clear and colourless, xanthochromic (yellow)). In the laboratory, CSF should be tested within an hour of sample collection for:

(a) Cell count—raised red blood cells do not necessarily imply the patient has had a SAH, they may be present following a traumatic lumbar puncture. If the red cell count is raised due to SAH, it will be consistently high in all three CSF samples (in a traumatic tap, RBC count should reduce from bottle 1 to 3). In the context of a SAH, the RBC count is usually $>2000 \times 10^6$ cells/L.
(b) Spectrophotometry—this technique identifies a substance by how much light it absorbs. Red blood cells breakdown into oxyhaemoglobin (4–10 h after a bleed) and bilirubin (10 h after a bleed and remains for up to 2w). A LP is considered diagnostic for SAH if bilirubin is present as it could not be present in the sample if there had been a traumatic tap. In some cases, only oxyhaemoglobin is present which is less sensitive for SAH as it may be present from a traumatic tap and delayed processing in the laboratory. On occasion, the oxyhaemoglobin peak masks the bilirubin peak on spectrophotometry due to high concentrations, in which case the suspicion for SAH should remain high and the patient appropriately investigated.

2. Infection

The lumbar puncture is frequently used by our medical colleagues when assessing patients for intracranial infections, e.g. meningitis and encephalitis. There are occasions when neurosurgeons need to use it for the same problem, but this usually follows previous neurosurgical intervention. When testing for infection, CSF should be sent for a cell count and microscopy and culture; if the concern is of infection, the laboratory should be forewarned and the sample processed for cell count and gram stain immediately. Neurosurgical patients with CSF infections may have concurrent hydrocephalus so an opening pressure should always be taken.

Interpreting CSF cell counts can take some practice and requires knowledge of normal CSF cell ratios as traumatic LP can lead to the presence of red and white blood cells in a CSF sample. The following sequence should be adopted when analysing CSF cell counts:

1. Red blood cell to white blood cell ratio
   1 white cell to every 500 red blood cell is the rule. If this ratio is different (i.e. there are >1 white cell to every 500 red blood cells), the white cell count is raised.

2. White Cell count
   The white cell count should be divided into percentage composition of lymphocytes (60 ± 20%), mononuclear cells (30 ± 15%) and polymorphs (2 ± 4%) (normal adult levels in parentheses). If polymorph count is most raised, this is highly suspicious for bacterial infection. Post surgery, the CSF is usually raised; if the predominant white cell remains, the lymphocyte this can be considered a normal inflammatory post-surgical response.
3. Hydrocephalus
   A lumbar puncture offers a bedside assessment of intracranial pressure. Contraindications to LP should prevail (large intracranial mass lesion, obstructive hydrocephalus, evidence of herniation syndromes, derranged clotting).

CSF Beta Transferrin/Tau Protein

If there is suspicion that a patient is having a CSF leak, the fluid can be tested for beta-2 transferrin or tau protein. If present, this indicates that it is indeed CSF.

Blood Tests

Sodium

Serum sodium (Na) is a very important investigation in patients with neurosurgical illness as imbalance happens frequently, the diagnosis and management is nuanced and consequences if ill-treated fatal. Most neurosurgical patients require daily U+E's to monitor serum Na.

Hyponatraemia is a frequently encountered problem in patients with traumatic brain injury, subarachnoid haemorrhage, brain tumours and pituitary disease. It can occur rapidly and produce cerebral oedema, seizures and death. Treatment is vital but should be done with care to avoid over-rapid correction with the risk of catastrophic central pontine myelinolysis.

The common causes of hyponatraemia in neurosurgical patients include syndrome of inappropriate anti-diuretic hormone (SIADH), cerebral salt wasting syndrome (CSWS), acute glucocorticoid insufficiency and excessive fluid resuscitation. Knowing the cause is essential for appropriate management, and we suggest the following ways to assess a patient with low Na:

1. What is the baseline Na (do they suffer from chronic hyponatraemia?)
2. Are they on any drugs that lower Na (e.g. proton pump inhibitors, furosemide, carbamazepine)?
3. How rapid was the drop in Na?
4. What is the fluid balance (hypervolaemic, euvolaemic or hypovolaemic)?
5. What is the plasma and urine osmolality?
6. What is the urinary Na?

In most neurosurgical cases the cause of hyponatraemia is SIADH in which case the patient will be clinically euvolaemic with a low serum osmolality (<280 mOsm/kg), high plasma osmolality (100 mOsm/kg) and elevated urinary sodium (>40 mmol/L). CSWS is rarer and its existence more controversial. The main difference between SIADH and CSWS is fluid balance—CSWS being characterised by hypovolemia and high urine output. It is important to distinguish between SIADH and CSWS as management is different. SIADH requires fluid restriction whereas CSWS needs fluid resuscitation. SIADH in the context of aneurysmal subarachnoid haemorrhage (where hyponatraemia is reported in c.50% of cases) can be especially challenging as fluid restriction can provoke cerebral vasospasm, so these patients may need to be managed with support from the endocrine team and occasionally hypertonic saline in order to maintain a positive fluid balance.

Hypernatraemia is less common in neurosurgical patients but can occur especially after pituitary surgery secondary to diabetes insipidus (DI) and is also seen in traumatic brain injury (either due to direct pituitary dysfunction or as a consequence of hyperosmolar therapy). Knowledge of the patient's fluid balance, hourly urine output and serum/plasma osmolality is key in guiding patient management. These patients will need a urinary catheter to ensure an accurate fluid balance is collected. If they are in a state of DI, this needs to be managed carefully with fluid therapy and on occasion DDAVP.

Most patients with electrolyte disturbance will be managed in conjunction with the intensivists or endocrine team. The neurosurgical junior should understand the principles of management and tests to assess patients with sodium imbalance and the need to escalate this problem to their seniors.

White Cell Count (WCC) and C-Reactive Peptide (CRP)

Inflammatory markers are important in the assessment of infection. Neurosurgical patients are often immobile and immunocompromised from steroid therapy so are at high risk of common hospital infections (urosepsis/line infections/chest sepsis).
In neurological infections the peripheral markers of sepsis are less useful due to the blood brain barrier and difficulties of CNS infection promoting a systemic response. Nevertheless in most instances the WCC and CRP will rise, but in the event that they are normal and infection is still suspected a circumspect appreciation of the WCC and CRP sensitivity to neurological infection should be taken.

Clotting and Platelet Count

Whilst the brain comprises c.2% of the bodies' weight, it takes between 15% and 20% of the cardiac output. Neurosurgical intervention in the context of deranged clotting is highly risky and every measure should be taken to normalise clotting prior to operating.

Management

There is no 'fits all' mould for managing neurosurgical patients. Whilst presentations remain similar, each case is filled with nuance which is often only visible to the experienced clinician, and as a result, decision-making in neurosurgery is touted by senior neurosurgeons as the steepest learning curve.

Handover offers a unique opportunity for young neurosurgeons to develop their own compass for patient management and a forum in which to discuss options. Having a process for doing so is very useful, and we recommend the following approach for considering the options. Despite its simplicity, it is a useful and easy framework for case discussion.

1. Stabilise
   Using the ABCDE approach to neurosurgical management is very sensible. The patients are often very unwell, and this approach is universally approved by doctors as the best way for managing the acutely unwell.
2. Involve
   Neurosurgical patients often need the attention of other medical teams, e.g. the anaesthetist, ICU team or theatre staff. Informing them early leads to a smoother patient pathway.
3. Manage: conservative/medical/surgical
   Generally speaking all patients whatever the problem can be managed according to these three principles. It may sound obvious, but it is a good structure for the options.

As example, we will discuss the management options for the introductory case:

'51M RH builder, co-habits with his wife and children. Witnessed collapse on pavement, no obvious head injury. GCS 8 when LAS arrived (M5, V2, E1). En route to hospital fixed and dilated R pupil. GCS 6 on arrival (M4, V1, E1). CT head showed Fisher Grade 3+4 SAH, R ASDH with mass effect and CTA a R PComm aneurysm.'

This is a neurosurgical emergency. The patient should be stabilised with an ABCDE approach. The anaesthetist, theatre and ICU team should be informed. Conservative management is not appropriate—the patient is young with surgically remediable pathology and a unilaterally fixed pupil. The anaesthetist should perform medical measures to help reduce ICP (give mannitol or hypertonic saline, hyperventilate to a PCO2 of 4 and give Keppra). Immediate surgery should be organised in which the options are:

1. Craniotomy for evacuation of the ASDH with concurrent clipping of the Pcomm aneurysm
2. Craniotomy for evacuation of the ASDH followed by endovascular coiling of the Pcomm aneurysm

Option one is more favourable when the aneurysm morphology allows its satisfactory occlusion as it involves one procedure addressing both problems of mass effect from the ASDH and its cause (the aneurysm rupture).

Outcome

Discussing the outcomes of our neurosurgical patients is often neglected at handover as it is often too early in the patient's clinical pathway to reflect on this. In this book, we will endeavour to go further and show how patients did long term and the outcome measures that can be used to best assess this.

Evidence

Having an appreciation of the evidence base for which neurosurgical decisions are made is crucial, and handover is a good time to review this. In this book, we will offer explanations of the appropriate papers/studies/scoring systems that underpin the neurosurgical practice of each case.

Acknowledgements

We would like to acknowledge all the sources used for the material in this book. Special appreciation is expressed to our patients who generously allowed us to use their cases for training purposes.

We would also like to thank our colleagues and trainees from the South Thames London Neurosurgical Training Programme and especially the Neurosurgical Consultant Body at King's College Hospital, under whose mentorship we are all receiving a state-of-the-art neurosurgical training.

Many thanks to all the editors at Springer Publishing for their support and guidance.

Contents

Part V Tumor

Abbreviations

#	Fracture
Abx	Antibiotics
ACA	Anterior cerebral artery
ACDF	Anterior cervical discectomy and fusion
ACTH	Adrenocorticotropic hormone
AcommA	Anterior communicating artery aneurysm
ADF	Ankle dorsiflexion
A+E	Accident and emergency department
AEDs	Antiepileptic drugs
AF	Anterior fontanelle
AF	Atrial fibrillation
aFP	Alpha fetoprotein
AqS	Aqueductal stenosis
ASDH	Acute subdural haematoma
ASIA	American Spinal Injury Association
AVF	Arteriovenous fistula
AVM	Arteriovenous malformation
BG	Basal ganglia
b-HCG	Beta human chorionic gonadotrophin
BMI	Body mass index
BRAT	Barrow Ruptured Aneurysm Trial
C spine	Cervical spine
Ca	Cancer
CARAT	Cerebral Aneurysm Rerupture After Treatment study
CC	Colloid cyst
CC fistula	Carotid cavernous fistula
CES	Cauda equina syndrome
c/o	Complaining of
CRP	C-reactive protein
CSDH	Chronic subdural haematoma
CSF	Cerebrospinal fluid
CTA	Cerebral tomography angiogram
CT CAP	Computed tomography chest abdomen and pelvis
CTH	Computed tomography head

CT perfusion	Computed tomography perfusion scan (helps identify areas of ischaemic penumbra from established areas of infarct)
D	Days
DAI	Diffuse axonal injury
Dex	Dexamethasone
DH	Drug history
DSA	Digital subtraction angiogram
DVLA	Driving and Vehicle Licensing Agency
ECHO	Echocardiogram
EDEM	Extradural extramedullary tumour
EDH	Extradural haematoma
EHL	Extensor hallucis longus
ENT	Ear, nose and throat specialists
EtoH	Alcohol
EVD	External ventricular drain
ETV	Endoscopic third ventriculostomy
F	Female
FND	Focal neurological deficit
FSH	Follicle-stimulating hormone
F/U	Follow-up
FP	Fronto-parietal
GH	Growth hormone
Gram +	Gram-positive bacteria
Gram −	Gram-negative bacteria
GTCS	Generalised tonic-clonic seizure
GTR	Gross total resection
Gy	Gray
h/a	Headache
HASU	Hyperacute stroke unit
HCP	Hydrocephalus
HDU	High dependency unit
HEMS	Helicopter Emergency Medical Service
HGG	High-grade glioma
HGV	Heavy goods vehicle
HIV	Human immunodeficiency virus
HLD	Herniated lumbar disc
HTN	Hypertension
Hrly	Hourly
Hx	History
ICA	Internal carotid artery
ICH	Intra-cerebral haematoma
ICP	Intracranial pressure
IDEM	Intra-dural extramedullary tumour
IGF-1	Insulin-like growth factor

IDIM	Intra-dural intramedullary tumour
IHD	Ischaemic heart disease
IIH	Idiopathic intracranial hypertension
INR	International normalised ratio (measurement of extrinsic pathway clotting)
IOM	Intraoperative monitoring
ISAT	International Subarachnoid Aneurysm Trial
ISUIA	International Study of Unruptured Intracranial Aneurysms
IT	Intrathecal
ICU	Intensive care unit
IV	Intravenous
I+V	Intubated and ventilated
IVH	Intraventricular haemorrhage
I+V+S	Intubated and ventilated and sedated
L	Left
LAS	London ambulance service
LBP	Lower back pain
LGG	Low-grade glioma
LH	Left-hand dominant
LL	Lower limb
LP	Lumbar puncture
L-S spine	Lumbo-sacral spine
M	Male
MCA	Middle cerebral artery
MDI	Myelopathy Disability Index
MDT	Multidisciplinary team
MEPs	Motor-evoked potentials
Mets	Metastasis
MI	Myocardial infarction
mJOA	Modified Japanese Orthopaedic Association scale
MLS	Midline shift
MS	Multiple sclerosis
MSCC	Metastatic spinal cord compression
MRA	Magnetic resonance angiogram
MRC grading	Medical Research Council muscle grading
MRI	Magnetic resonance imaging
MRI ADC Map	Apparent diffusion coefficient magnetic resonance imaging sequence (used with DWI to look verify if there is evidence of restricted diffusion, i.e. free movement of hydrogen)
MRI DWI	Diffusion-weighted magnetic resonance imaging (looks at restricted movement of hydrogen molecules and is useful in identifying infarct, intracerebral abscess and tumours with high cellularity)

MRI 'feed and wrap'	MRI in a neonate where the infant is fed and then swaddled to encourage it to sleep for the scan
MR perfusion	Magnetic resonance perfusion imaging (helps identify areas of ischaemic penumbra from established areas of infarct)
MRI (T2 sagittal CISS)	Magnetic resonance T2-weighted imaging with a constructive interference in steady state (type of CSF flow study)
Na	Sodium
Neuro obs	Neurological observations
NPH	Normal pressure hydrocephalus
N/V	Nausea and vomiting
O/E	On examination
OFC	Occipital frontal circumference
OGM	Olfactory groove meningioma
OPG	Orthopantomogram
OT	Occupational therapy
PCA	Posterior cerebral artery
PcommA	Posterior communicating artery
PERLA	Pupils equal and reactive to light and accommodation
PF	Posterior fossa
PICC line	Peripherally inserted central catheter
PLAP	Placental alkaline phosphatase
PMH	Past medical history
Post op	Post operative
PPI	Proton pump inhibitor
PR	Per rectal examination
PROM	Premature rupture of membranes
PRL	Prolactin
PSA	Prostate-specific antigen
PT	Physiotherapy
QDS	Four times daily
R	Right
RESCUE ASDH	Randomised Evaluation of Surgery with Craniectomy for patients Undergoing Evacuation of Acute Subdural Haematoma
RH	Right hand dominant
RF EVD	Right frontal external ventricular drain
r/o	Rule out
ROM	Range of movement
RTA	Road traffic accident
SAH	Subarachnoid haemorrhage
SALT	Speech and language therapy
SCA	Superior cerebellar artery
SCC	Squamous cell carcinoma

SDH	Subdural haematoma
Secs	Seconds
SLIC	Subaxial spine injury classification
SLR	Straight leg raise
SOH	Sudden onset headache
SOL	Space-occupying lesion
SRS	Stereotactic radiosurgery
SSEPS	Somatosensory-evoked potentials
STICH	Surgical treatment for intracerebral haemorrhage trial (number I and number II)
T1WI	T1-weighted MRI imaging sequence
T1WI+c (Gad)	T1-weighted MRI imaging sequence with contrast (gadolinium)
T2WI	T2-weighted MRI imaging sequence
T4	Thyroxine
TB	Tuberculosis
TBI	Traumatic brain injury
TDS	Three times daily
TIIDM	Type 2 diabetes mellitus
TLICS	Thoracolumbar injury classification and severity score
TLSO	Thoracolumbar spinal orthosis
TSH	Thyroid-stimulating hormone
UL	Upper limb
UMN	Upper motor neurone
U/S	Ultrasound
VA	Visual acuity
VA	Vertebral artery
VAS	Ventriculo-arterial shunt
VB	Vertebral body
VF	Visual fields
VPS	Ventriculo-peritoneal shunt
VST	Venous sinus thrombosis
W	Weeks
WBC	White blood cell count
WFNS grade	World Federation of Neurosurgeons grading of subarachnoid haemorrhage
WHO grade	World Health Organisation classification of brain tumours (2016)
WHOL	Worst headache of life
XRT	Radiotherapy

Introduction

'51M RH builder, co-habits with his wife and children. Witnessed collapse on pavement, no obvious head injury. GCS 8 when LAS arrived (M5, V2, E1). En route to hospital fixed and dilated R pupil. GCS 6 on arrival (M4, V1, E1). I+V + mannitol in A+E, R pupil remained fixed. CT head showed Fisher Grade 3+4 SAH, R ASDH with mass effect and CTA a R PComm aneurysm. Taken directly to theatre for craniotomy, evacuation of ASDH and clipping of Pcomm aneurysm. Currently I+V with EVD on NICU, pupil remains dilated but post op CT satisfactory. Plan is to wean, wake and assess neurology.'

The above is example of the familiar repartee of the neurosurgical handover. Direct, succinct and yet full of important diagnostic and prognostic information for any medical professional who may become involved in the management of the patient described and also the culmination of excellent handover from the start of this patient's neurosurgical story.

Brief estimation calculates at least eight different individuals involved in communicating this history in order to hand over his care, and yet this forms only the acute stage of his admission. From this point until discharge, his story will be repeated at every nursing and doctor handover in order to ensure he receives the best and most comprehensive care (Fig. 12).

At King's College Hospital, the neurosurgical handover meetings extend beyond the realm of purely good patient care and provide a unique learning opportunity for all who attend. Starting at 07:50 in the radiology seminar room, the on-call neurosurgical SPR leads the meeting which is chaired by the consultant on call. All acute admissions, operations and patient issues that occurred over the preceding 24 h are discussed and dissected—each case forming a vignette to structure a learning experience preparing SHOs for life as registrars and registrars for consultancy.

We have experienced the King's Handover as an SHOs, Registrars and Senior Clinical Fellows. Its influence on our neurosurgical practice has been unsurpassed and is one of the reasons we (the neurosurgical trainees at King's) decided to write this book. Our aim is to present common neurosurgical conditions in the style of the King's Neurosurgical Handover, sharing the wealth of experience and pathology from this busy unit and offering a framework for budding neurosurgeons to use when assessing/presenting neurosurgical cases.

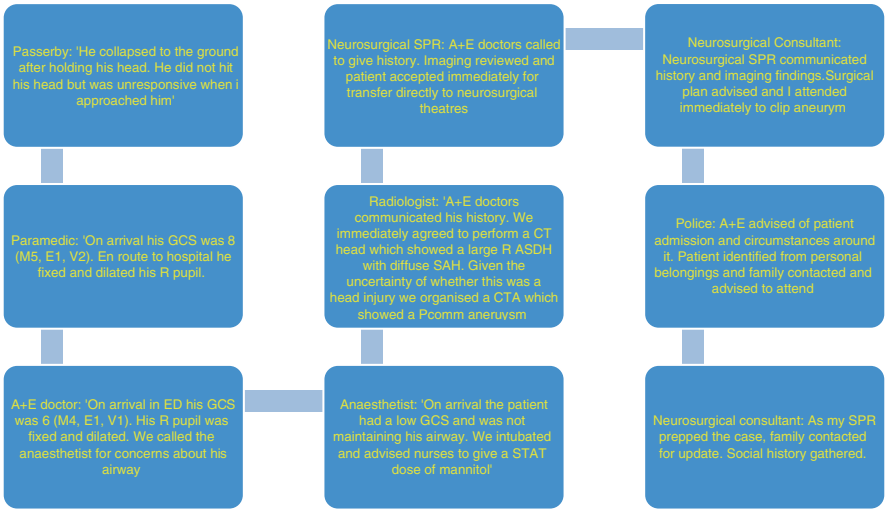


Fig. 12 Summary of all individuals involved in patients handover

Part I

Head Trauma

Contusion

1

Abbreviations

ASDH	Acute sub-dural haematoma
CTH	Computed tomography head
DAI	Diffuse axonal injury
EDH	Extra-dural haematoma
FND	Focal neurological deficit
f/u	Follow up
PT/OT	Physiotherapy and occupational therapy
TBI	Traumatic brain injury

Clinical Presentation

F65, RH, previously healthy, fell backwards and sustained head injury, presents with swelling in left occipital area and drowsiness, GCS E3 V5 M6, no major FND

Differential Diagnosis

1. ASDH
2. EDH
3. DAI
4. Contusion
5. Fracture

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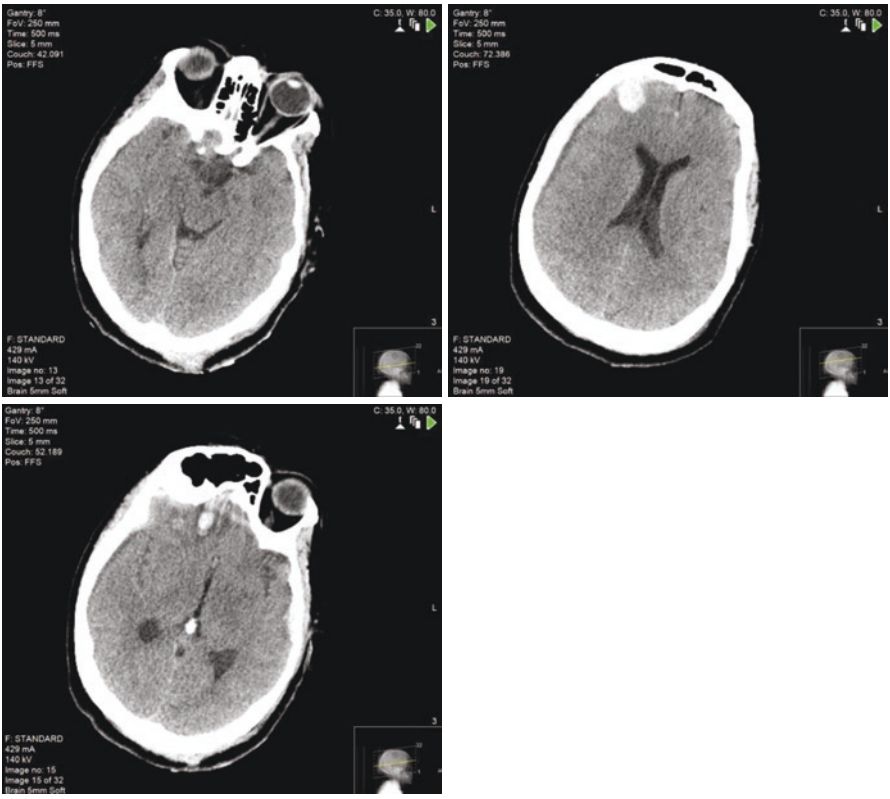
Investigations

CTH revealed left occipital subgaleal haematoma and contra-coup right frontal contusions



Management

Conservative management with neuro obs, analgesia, PT/OT, repeat imaging if neurological deterioration, monitor for hyponatraemia and seizures

Outcome

Recovered gradually over a week to being asymptomatic and intact neurologically with TBI clinic f/u

Depressed Skull Fracture

2

Abbreviations

Abx	Antibiotics
c/o	Complaining of
CTH	Computed tomography head
D2	2 days
LOC	Loss of consciousness
Secs	Seconds

Clinical Presentation

F18, RH, healthy, assaulted with a hammer blow on the head, presented with LOC for 30 secs, o/e intact neurologically, c/o h/a, left parietal laceration

Differential Diagnosis

1. Left parietal compound fracture with or without underlying intracranial injury

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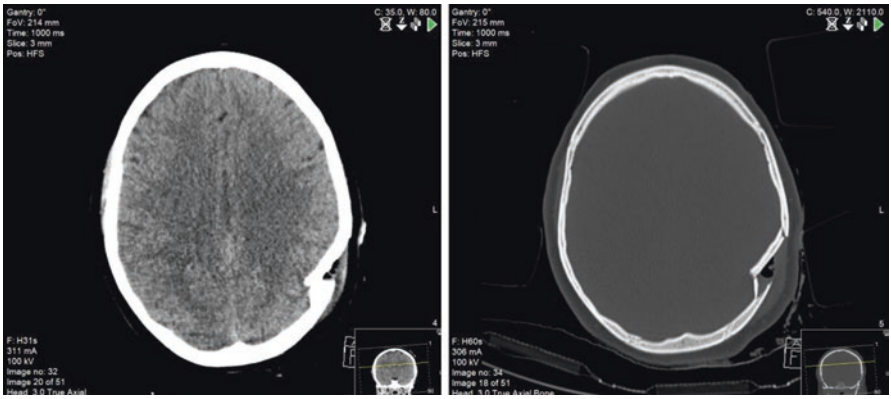
Investigations

CTH revealed left parietal depressed fracture without underlying brain injury

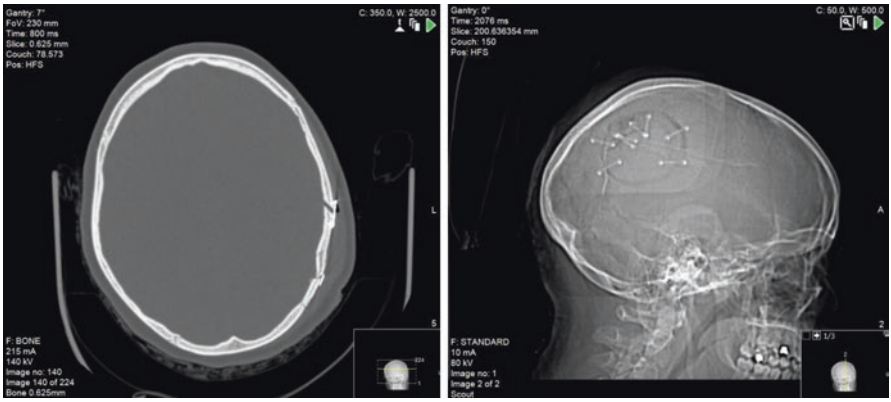


Management

Because the fracture is compound and the depression significant (> the skull thickness) the fracture was elevated and the skull reconstructed. Pneumococcal vaccine was administered and only prophylactic peri operative abx given, in order to avoid bacterial resistance.

Outcome

Discharged home D2 intact neurologically and asymptomatic



Evidence

Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger J, Surgical Management of Traumatic Brain Injury Author Group. Surgical management of depressed cranial fractures. Neurosurgery. 2006;58(3 Suppl):S56–60; discussion Si–iv.

Diffuse Axonal Injury

3

Abbreviations

ASDH	Acute sub-dural haematoma
C spine	Cervical spine
CT CAP	Computed tomography chest abdomen and pelvis
CTH	Computed tomography head
DAI	Diffuse axonal injury
EDH	Extra dural haematoma
F/U	Follow up
ICP	Intracranial pressure
TBI	Traumatic brain injury

Clinical Presentation

M29, RH, plasterer, intoxicated with EtoH and crashed into a tree at 40 mph. At scene GCS E2 V2 M5, I+V+S, upon admission left pupil dilated to 5 mm

Differential Diagnosis

1. ASDH
2. EDH
3. DAI
4. Contusion

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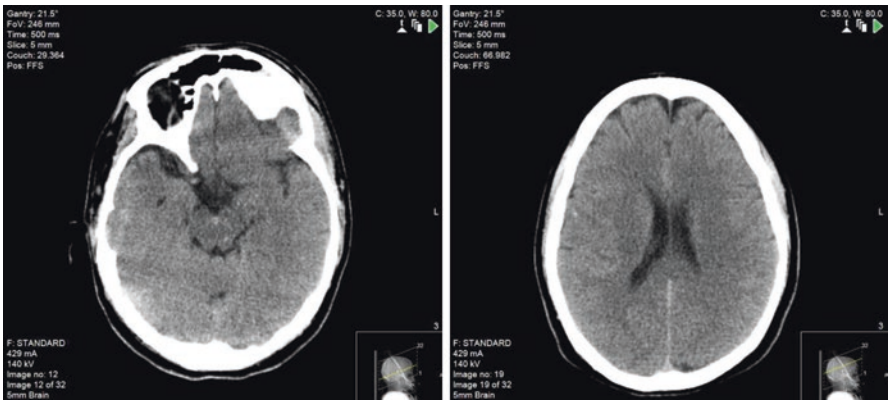
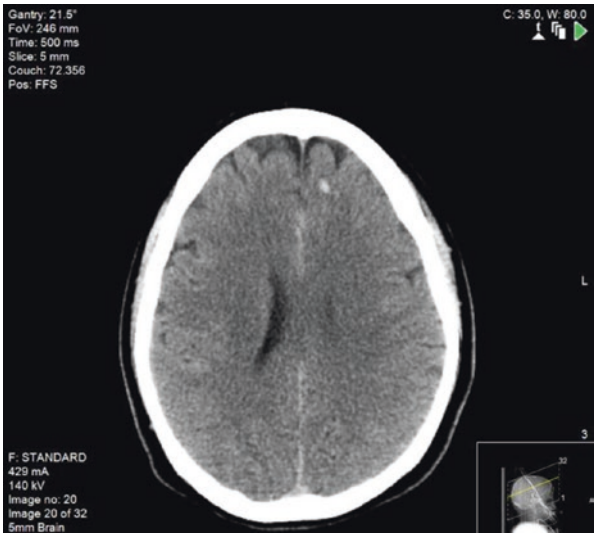
Investigations

CTH revealed DAI with petechial haemorrhages in the midbrain, interpeduncular cistern, anterior corpus callosum, grey-white matter zone

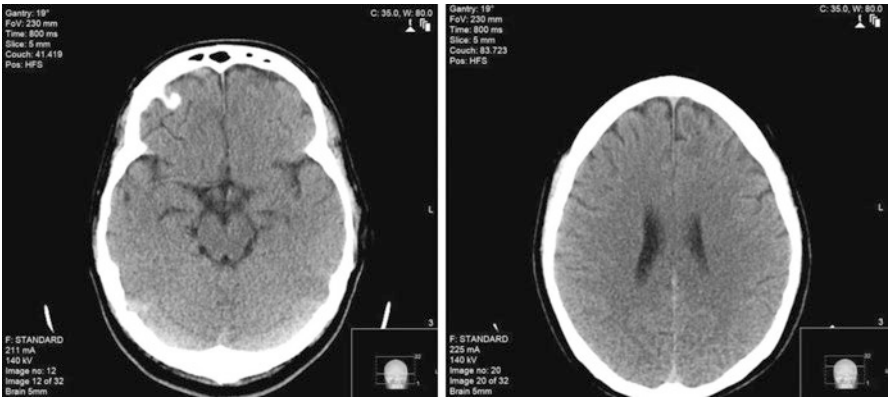
CT C spine ruled out fracture/dislocation, CT CAP revealed pulmonary contusions and rib fractures





Management

Trauma Tertiary Survey
ITU medical management of head injury
Insertion of ICP monitor and neuroprotection for 48 h in ITU
Left pupillary dilation explained from left midbrain contusion and is not secondary to mass effect



Outcome

Prolonged inpatient stay, tracheostomy, neuro rehabilitation
Recovered with cognitive impairment
TBI clinic f/u

Evidence

Abu Hamdeh S, Marklund N, Lannsjö M, Howells T, Raininko R, Wikström J, Enblad P. Extended anatomical grading in diffuse axonal injury using MRI: hemorrhagic lesions in the substantia nigra and mesencephalic tegmentum indicate poor long-term outcome. J Neurotrauma. 2017;34(2):341–52.

Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, Mclellan DR. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology. 1989;15:49–59. https://doi.org/10.1111/j.1365-2559.1989.tb03040.x.

Deepika A, Prabhuraj AR, Saikia A, et al. Comparison of predictability of Marshall and Rotterdam CT scan scoring system in determining early mortality after traumatic brain injury. Acta Neurochir. 2015;157(11):2033–8. https://doi.org/10.1007/s00701-015-2575-5.

Marshall LF, Marshall SB, Klauber MR, et al. The diagnosis of head injury requires a classification based on computed axial tomography. J Neurotrauma. 1992;9(Suppl 1):S287–92.

Vieira RC, Paiva WS, de Oliveira DV, Teixeira MJ, de Andrade AF, de Sousa RMC. Diffuse axonal injury: epidemiology, outcome and associated risk factors. Front Neurol. 2016;7:178. https://doi.org/10.3389/fneur.2016.00178.

Extradural Haematoma

4

Abbreviations

ASDH	Acute sub-dural haematoma
CTH	Computed tomography head
DAI	Diffuse axonal injury
F-P	Fronto-parietal
GTCS	Generalised tonic clonic seizure
ICH	Intra-cerebral haematoma
ICU	Intensive care unit
I+V+S	Intubated + ventilated + sedated

Clinical Presentation

M51, RH, known epileptic, suffered GTCS whilst shopping, post ictal state GCS E1 V1 M4, left pupil fixed and dilated, I+V+S at scene, mannitol given, blue lighted to neuro theatres

Differential Diagnosis

1. EDH
2. ASDH
3. DAI
4. Contusion
5. Post ictal
6. Spontaneous ICH

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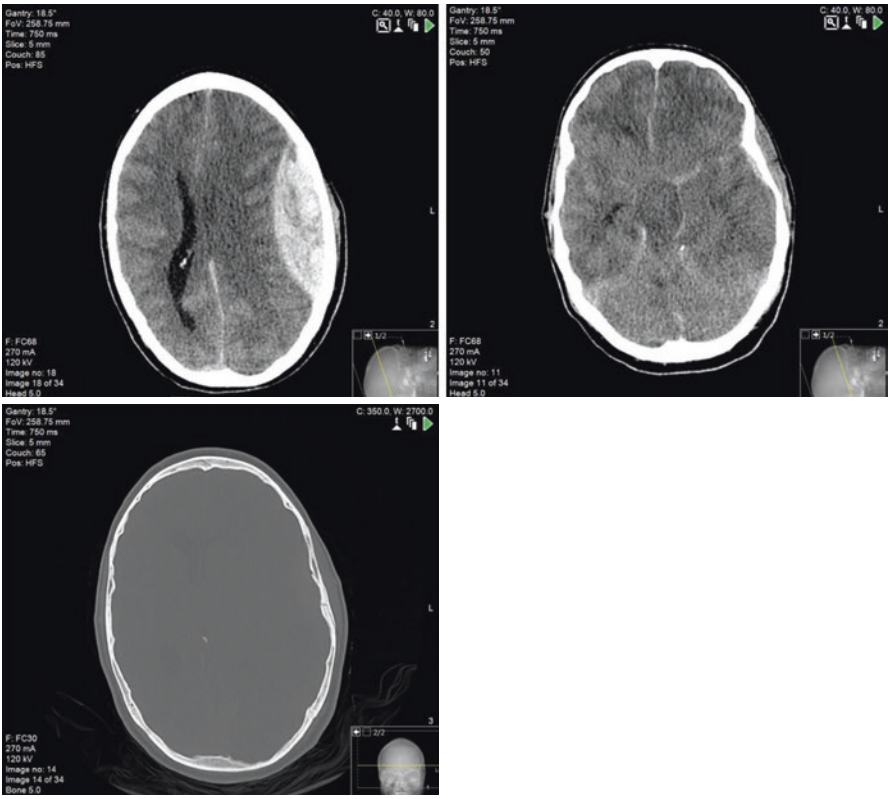
Investigations

CTH revealed large L EDH with significant mass effect and pseudo subarachnoid sign of venous stasis in basal cisterns secondary to raised ICP and un-displaced left F-P fracture



Management

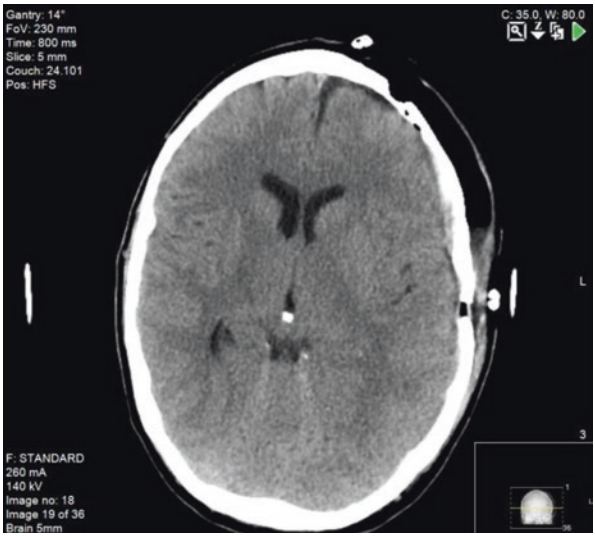
Urgent left sided craniotomy and evacuation of EDH

R posterior shoulder dislocation reduced by Orthopaedic Team

Outcome

Full recovery and back to normal baseline, anti-epileptic therapy tailored

Prompt surgical evacuation of EDH without underlying brain injury facilitates full recovery



Evidence

Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger JE, Surgical Management of Traumatic Brain Injury Author Group. Surgical management of acute epidural hematomas. Neurosurgery. 2006;58(3 Suppl):S7–15; discussion Si–iv.

Maugeri R, Anderson DG, Graziano F, Meccio F, Visocchi M, Iacopino DG. Conservative vs. surgical management of post-traumatic epidural hematoma: a case and review of literature. Am J Case Rep. 2015;16:811–7. https://doi.org/10.12659/AJCR.895231.

Acute Subdural Haematoma

5

Abbreviations

A+E	Accident and emergency department
ASDH	Acute sub-dural haematoma
C spine	Cervical spine
CTH	Computed tomography head
DAI	Diffuse axonal injury
EDH	Extra dural haematoma
ICP	Intracranial pressure
ICU	Intensive care unit
I+V+S	Intubated and ventilated and sedated
MLS	Midline shift
PMH	Past medical history
RESCUE ASDH	Randomised Evaluation of Surgery with Craniectomy for patients Undergoing Evacuation of Acute Subdural Haematoma
SDH	Subdural haematoma

Clinical Presentation

M34, RH, without significant pmh, was assaulted and suffered severe head injury at scene GCS E1 V1 M4, I+V+S, bluelighted in AE where left pupil dilated and fixed but responded to hypertonic saline, no other major injuries

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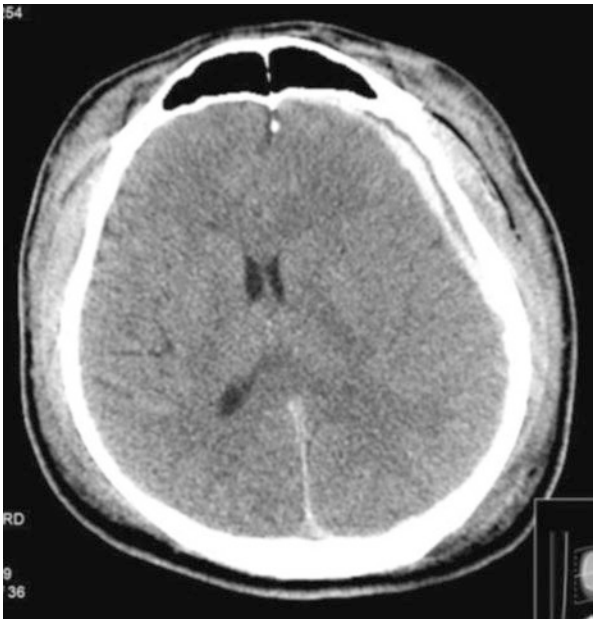
Differential Diagnosis

1. ASDH
2. Acute EDH
3. DAI
4. Contusion

Investigations

CTH revealed Left sided Hyperacute SDH with mixed density and significant mass effect and MLS

CT C spine ruled out fracture/dislocation



Management

Urgent Left sided decompressive craniectomy and evacuation of ASDH and insertion of ICP monitor

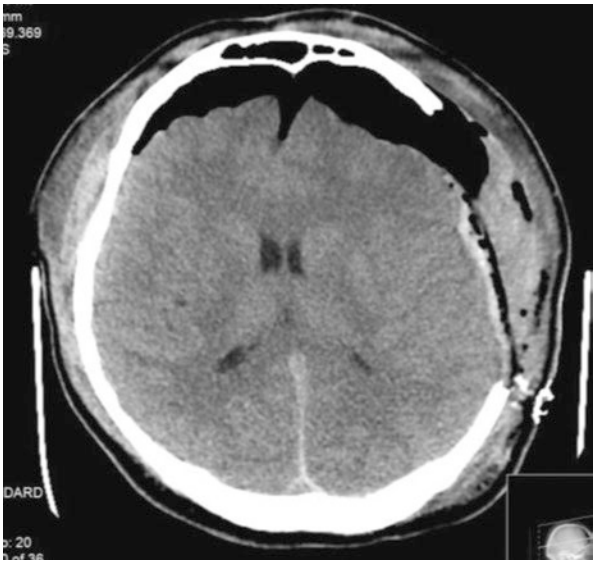
Recruited to RESCUE ASDH

Transfer to ICU

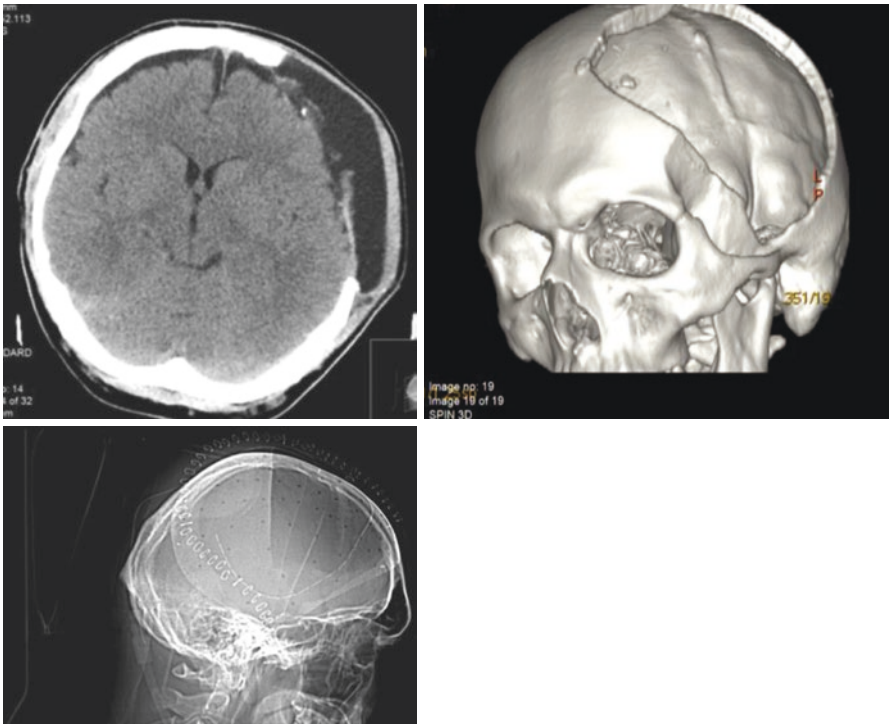
Keep I+V+S, full medical management of raised ICP as per protocol

Head up to 30°

Outcome



Post op CTH revealed adequate evacuation of ASDH and relief of mass effect, bifrontal pneumocephalus managed with high flow oxygen, after 72 h in ICU the ICP monitor was removed, sedation weaned and recovered neurologically to intact level of consciousness without significant motor deficit but cognitive impairment



Discharged eventually home with community support for cognitive deficit and re-admitted 6m later for titanium cranioplasty

Evidence

DECRA

Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, Wolfe R. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med. 2011;364(16):1493–502. https://doi.org/10.1056/NEJMoa1102077.

RESCUE ICP

Danish SF, et al. Quality of life after hemicraniectomy for traumatic brain injury in adults: a review of the literature. Neurosurg Focus. 2009;26(6):E2.

Ho KM, Honeybul S, Litton E. Delayed neurological recovery after decompressive craniectomy for severe nonpenetrating traumatic brain injury. Crit Care Med. 2011;39(11):2495–500.

Hutchinson PJ, Kolias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. N Engl J Med. 2016;375(12):1119–30. https://doi.org/10.1056/NEJMoa1605215.

Pediatric

Taylor A, et al. A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Childs Nerv Syst. 2001;17(3):154–62.

Chronic Subdural Haematoma

6

Abbreviations

AF	Atrial fibrillation
CSDH	Chronic sub-dural haematoma
CTH	Computed tomography head
DVLA	Driving and vehicle licensing agency
HTN	Hypertension
INR	International normalized ratio (measurement of extrinsic pathway clotting)
RTA	Road traffic accident
SAH	Sub arachnoid haemorrhage
SDH	Sub-dural haematoma

Clinical Presentation

M63, RH, taxi ver, pmh of HTN and on warfarin for AF, presents with 1w history of worsening headache and left sided weakness, Involved in RTA 3/52 ago when sustained mild head injury, o/e GCS E3 V4 M6, left sided weakness 4/5, INR 2.6

Differential Diagnosis

1. CSDH
2. ICH secondary to coagulopathy
3. aneurysmal SAH with ICH
4. HTN related ICH

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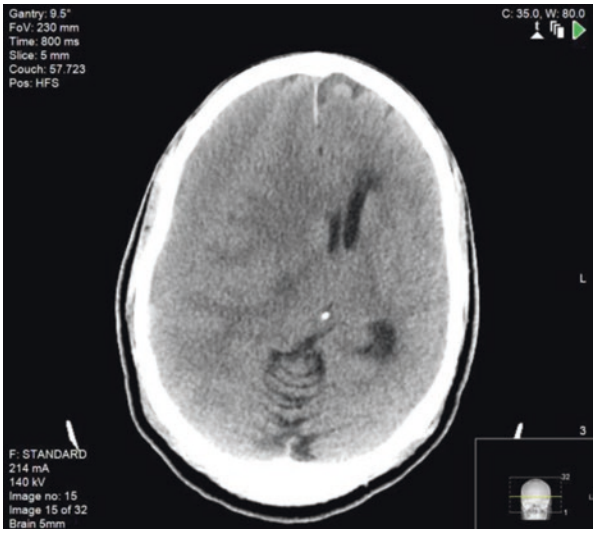
Investigations

CTH revealed large R convexity subacute SDH with MLS



Management

Warfarin reversed with coagulation factors (Octaplex) and Vitamin K as per haematology, INR 1.2

Two burr hole drainage of R CSDH and sub-dural drain
general management;

- conservative
- dexamethasone
- twist drill craniostomy, single burr hole, two burr holes, mini craniotomy (when significant membrane formation) ± subgaleal or subdural drain

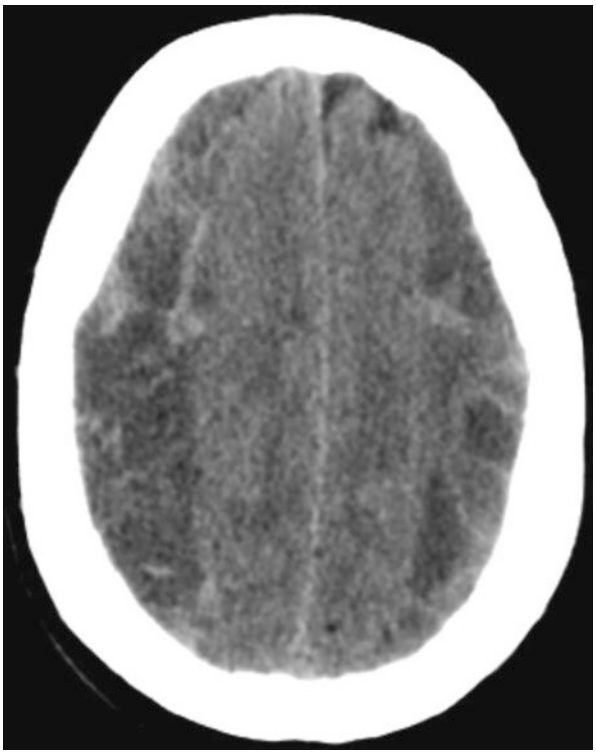


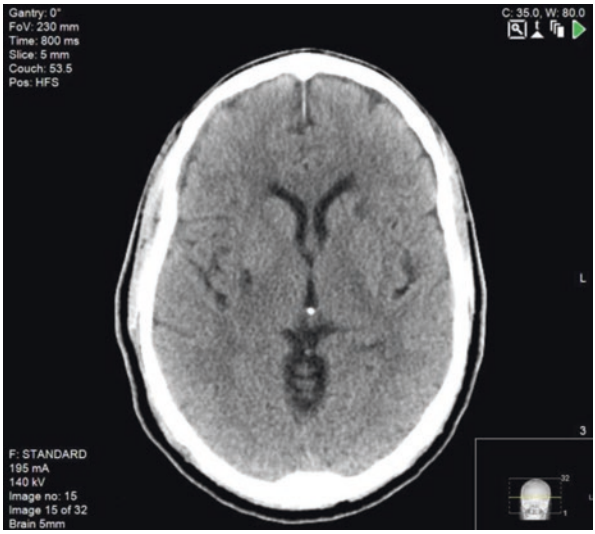
Figure with bilateral CSDH and significant membrane formation

Outcome

Returned back to baseline asymptomatic and intact

DVLA implications

Post op imaging confirmed adequate drainage of the subdural collection and relief of the mass effect



Evidence

Alcalá-Cerra G, Young AM, Moscote-Salazar LR, Paternina-Caicedo A. Efficacy and safety of subdural drains after burr-hole evacuation of chronic subdural hematomas: systematic review and meta-analysis of randomized controlled trials. World Neurosurg. 2014;82(6):1148–57.

Almenawer SA, Farrokhyar F, Hong C, Alhazzani W, Manoranjan B, Yarascavitch B, et al. Chronic subdural hematoma management: a systematic review and meta-analysis of 34,829 patients. Ann Surg. 2014;259(3):449–57.

Brennan PM, Kolias AG, Joannides AJ, Shapey J, Marcus HJ, Gregson BA, Grover PJ, Hutchinson PJ, et al. The management and outcome of patients with chronic subdural haematoma: a prospective, multi-centre, observational cohort study in the United Kingdom. J Neurosurg. 2017; https://doi.org/10.3171/2016.8.JNS16134.test.

Kansal R, Nadkarni T, Goel A. Single versus double burr hole drainage of chronic subdural hematomas. A study of 267 cases. J Clin Neurosci. 2010;17(4):428–9.

Motiei-Langroudi R, Stippler M, Shi S, Adeeb N, Gupta R, Griessenauer CJ, Papavassiliou E, Kasper EM, Arle J, Alterman RL, Ogilvy CS, Thomas AJ. Factors predicting reoperation of chronic subdural hematoma following primary surgical evacuation. J Neurosurg. 2017;15:1–8.

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Santarius T, Kirkpatrick PJ, Ganesan D, Chia HL, Jalloh I, Smielewski P, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet. 2009;374(9695):1067–73.

Severe Traumatic Brain Injury: Brainstem Death Tests

7

Abbreviations

A+E	Accident and emergency department
ASDH	Acute subdural haematoma
DAI	Diffuse axonal injury
EDH	Extradural haematoma
HEMS	Helicopter emergency medical service
I+V	Intubated and ventilated
LAS	London ambulance service
TBI	Traumatic brain injury

Clinical Presentation

M15 previously healthy was hit by a car travelling at 30 mph while crossing the road. He had a cardiac arrest at the scene, with bystander CPR for 4 min. He required chest compressions by LAS with return of systemic circulation after 9 min, not requiring adrenaline. He I+V at scene and had bilateral thoracostomies performed by HEMS who transferred him to A+E. He received hypertonic saline en-route in view of concerns regarding raised intracranial pressures. Pupils had reacted for a short while after but then became persistently fixed and dilated. On arrival to ED, pupils were fixed, dilated 7 cm and non-reactive. Further boluses of hypertonic saline were given to no effect.

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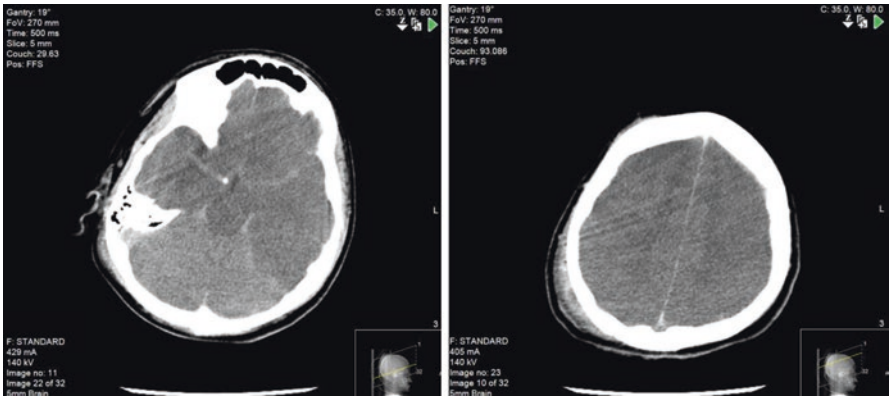
Differential Diagnosis

1. ASDH
2. EDH
3. DAI
4. Contusion
5. Malignant brain oedema secondary to hypoxia/hypovolemia

Investigations

CTH revealed malignant brain oedema and pseudosubarachnoid sign, loss of grey-white matter differentiation, white cerebellar sign



Management

Brainstem death tests

Outcome

Organ donation

Part II

Spine Injuries

Cervical Spine Fracture: Dislocation

8

Abbreviations

ASIA	American Spinal Injury Association
C5–6	Fifth to sixth cervical vertebrae (same applies to C7)
CT C spine	Computed tomography of cervical spine
D5	5 days
SLIC	Sub-axial spine injury classification
VA	Vertebral artery

Clinical Presentation

M35 with background of schizophrenia attempted to suicide hanging himself, found collapsed on the floor with inability to move his legs and arms, o/e intact level of consciousness, complete sensory level at C7, MRC 0/5 power below deltoid muscles, ASIA A

Differential Diagnosis

1. Cervical spine fracture/dislocation
2. Haematoma
3. Traumatic disc prolapse

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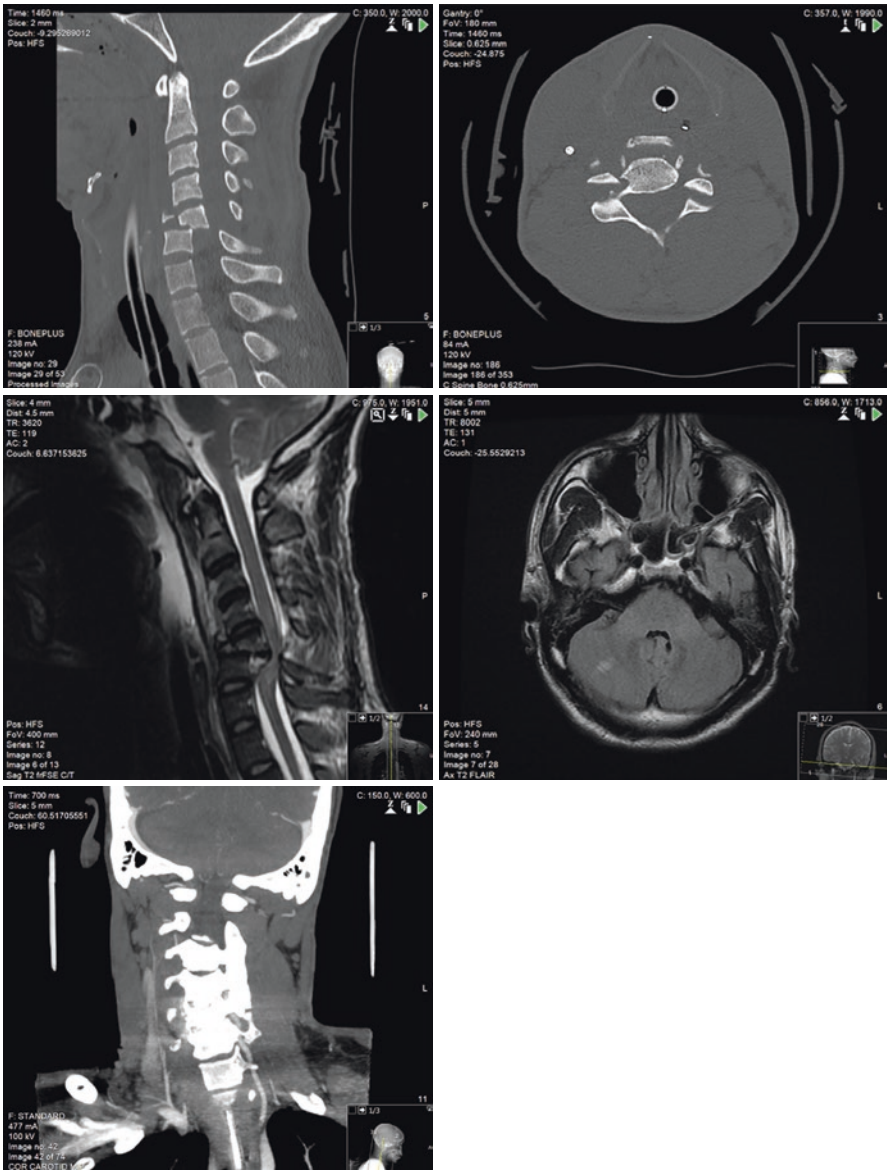
Investigations

CT C spine showed C5–6 fracture/dislocation with canal compromise and bilateral facet dislocation

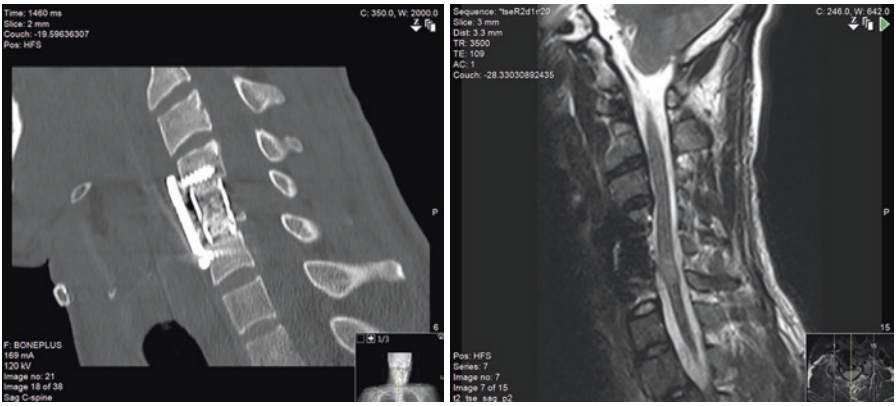
MRI C spine and Brain revealed significant cord compression and contusion and R inferior cerebellum ischaemic area

CTA confirmed occlusion of R VA shortly after it's origin at the R C6 foramen transversarium



Management

ACDF; C5 corpectomy, cage and C4–6 plate



D5 post op commenced on therapeutic anticoagulation for VA dissection

Transferred to Spinal Rehabilitation Unit, no neurological recovery

Spinal stabilisation performed to facilitate rehabilitation, improve pain long term, prevent secondary traumatic syringomyelia

Outcome

ASIA scoring system

Evidence

SLIC

Odontoid Peg Fracture

9

Abbreviations

C spine	Cervical spine
Ca	Cancer
MSCC	Metastatic spinal cord compression
T2WI	T2 weighted MRI imaging sequence
VA	Vertebral artery
VB	Vertebral body

Clinical Presentation

M67, RH, lives on his own, with known spinal and pelvic metastatic prostate Ca received in the past XRT and hormonal therapy presented with fall from stairs 3D ago causing significant neck pain and right shoulder soft tissue injury, o/e neurologically intact except mild limitation of right shoulder ROM due to localised injury

Differential Diagnosis

1. Peg fracture
2. Subaxial C spine fracture/dislocation
3. MSCC

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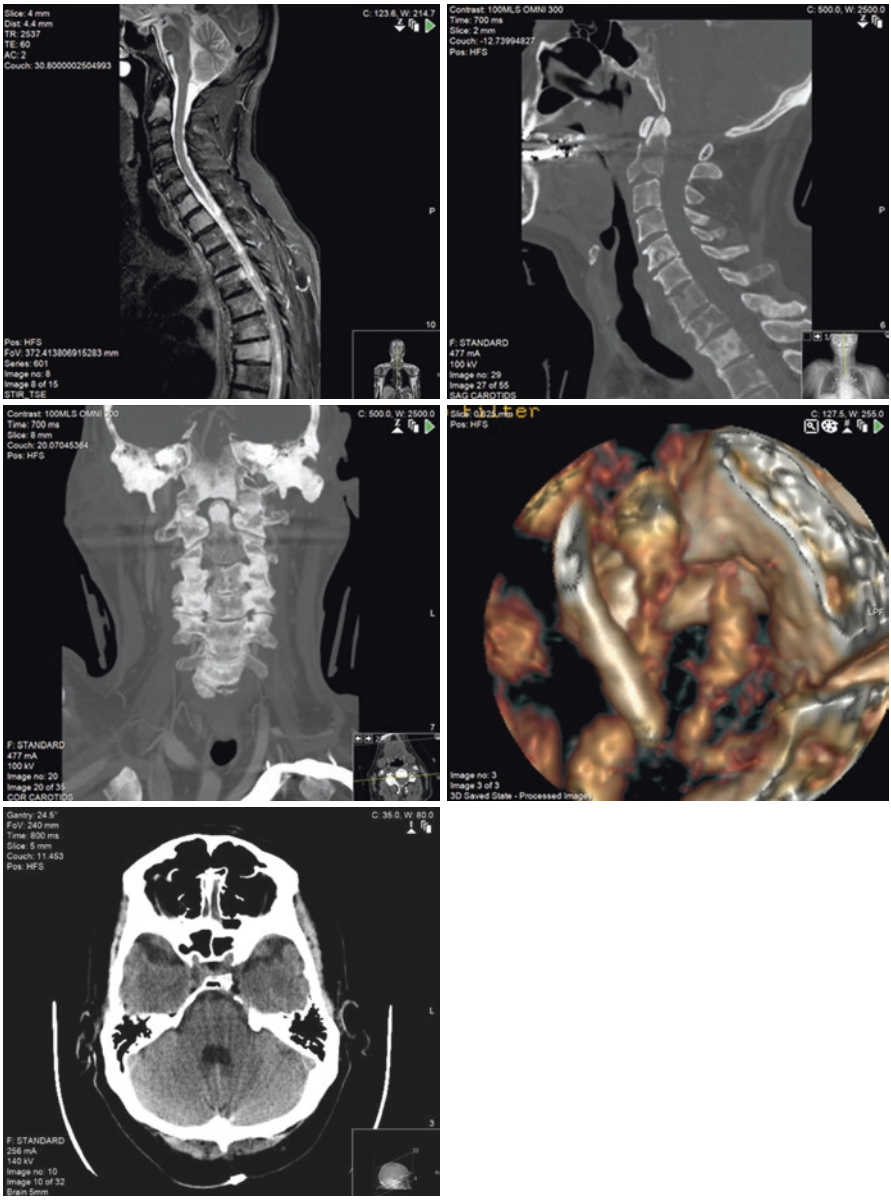
Investigations

CT revealed peg fracture type II and MRI C spine post radiotherapy VB high signal in T2WI

CTA demonstrated R VA traumatic dissection and dominant L VA and CTH ruled out subsequent stroke

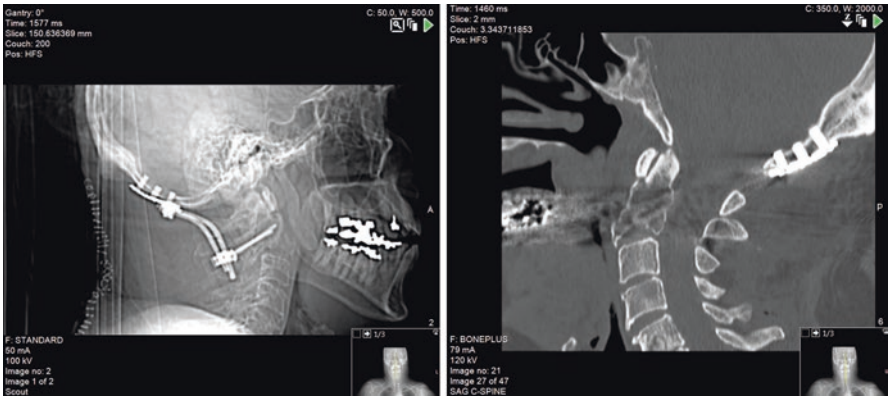


Management

C2-Occiput fusion in this case (reduces 50% rotation and flexion/extension)
If not fit for surgery manage with Miami J Collar or HALO
Odontoid screw is indicated in reducible type II fracture with intact TL
C1–2 fixation (limits 50% rotation)

Outcome

Returned back to baseline, post op imaging demonstrated appropriate metal work placement, D3 post op commenced on therapeutic anticoagulation for R VA dissection



Evidence

Anderson and D'Alonzo Classification

Type I	tip
Type II	base of neck
Type IIA	II with large bone chip
Type III	body

Surgery for type II, IIA or II and III if displacement >5 mm or alignment not maintained with collar/halo

Cervical Traumatic Disc Prolapse

10

Abbreviations

ACDF	Anterior cervical discectomy and fusion
C spine	Cervical spine
C6/7	Sixth and seventh cervical vertebrae
EtoH	Alcohol
r/o	Rule out
SLIC	Sub-axial spine injury classification

Clinical Presentation

M38, RH, with background of Depression, hypertension, EtoH excess, fell from a flight of stairs whilst intoxicated and developed neck pain with numbness and flaccid weakness of his limbs, o/e sensory level at C6, LL power 0/5, elbow flexion / extension 3/5 and O/5 distal UL power, retained bladder catheter sensation

Differential Diagnosis

1. Cervical spine fracture/dislocation with cord compression
2. Acute traumatic disc prolapse
3. Spinal haematoma

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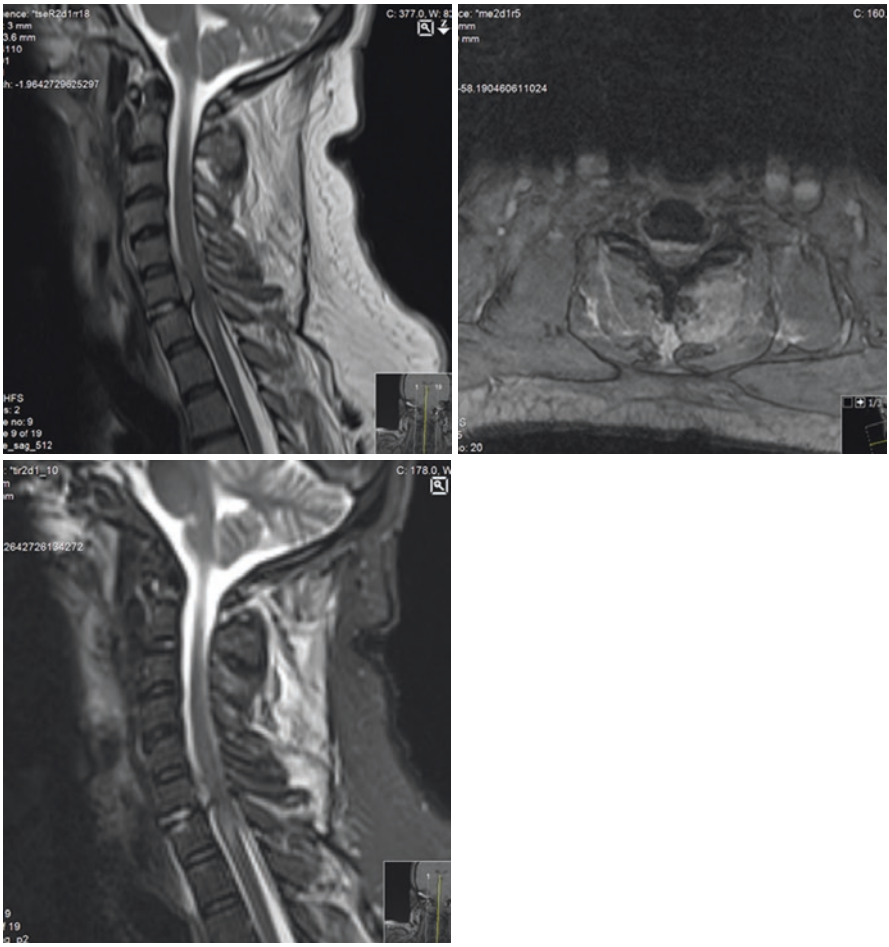
Investigations

Whole body trauma CT since patient is still intoxicated in order to r/o other injuries

C spine CT/MRI revealed C6–7 Acute traumatic disc prolapse causing cord compression

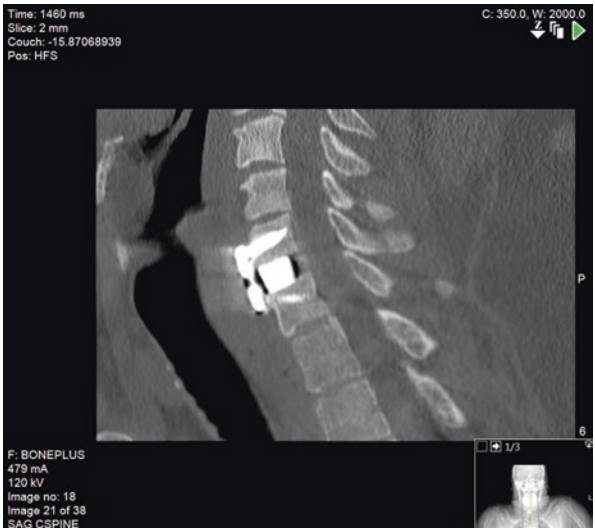


Management

Placed in a Miami J collar and underwent urgent C6–7 ACDF and plate.

Outcome

Post operatively he regained partial sensorimotor function and transferred to a dedicated Spinal Neuro Rehabilitation Unit.



Evidence

SLIC

Dvorak MF, Fisher CG, Fehlings MG, Rampersaud YR, Oner FC, Aarabi B, Vaccaro AR. The surgical approach to subaxial cervical spine injuries: an evidence-based algorithm based on the SLIC classification system. Spine (Phila Pa 1976). 2007;32(23):2620–9.

Moore TA, Vaccaro AR, Anderson PA. Classification of lower cervical spine injuries. Spine (Phila Pa 1976). 2006;31(11 Suppl):S37–43; discussion S61.

Vaccaro AR, Hulbert RJ, Patel AA, Fisher C, Dvorak M, Lehman RA Jr, Anderson P, Harrop J, Oner FC, Arnold P, Fehlings M, Hedlund R, Madrazo I, Rechtine G, Aarabi B, Shainline M, Spine Trauma Study Group. The subaxial cervical spine injury classification system: a novel approach to recognize the importance of morphology, neurology, and integrity of the discoligamentous complex. Spine (Phila Pa 1976). 2007;32(21):2365–74.

Thoracic Wedge Compression Fracture

11

Abbreviations

T9/10	Ninth and tenth thoracic vertebrae
TLSO	Thoracolumbar spinal orthosis
VB	Vertebral body

Clinical Presentation

M30, RH, healthy, motorbike racing driver, sustained thoracic spine injury whilst racing expressed with back pain and no neurological compromise

Differential Diagnosis

1. Soft tissue injury
2. Thoracic spine fracture

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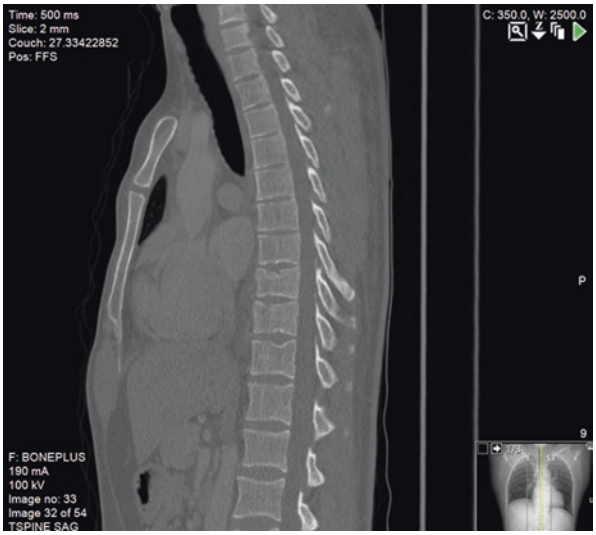
Investigations

CT spine revealed T9,10 wedge compression fractures without significant loss of VB height, angulation or retropulsion into the spinal canal



Management

Managed in a TLSO brace for 3m

Outcome

Remained asymptomatic and intact

Evidence

TLICS

Patel AA, Dailey A, Brodke DS, Daubs M, Harrop J, Whang PG, Vaccaro AR, Spine Trauma Study Group. Thoracolumbar spine trauma classification: the Thoracolumbar Injury Classification and Severity Score system and case examples. J Neurosurg Spine. 2009;10(3):201–6.

Vaccaro AR, Zeiller SC, Hulbert RJ, Anderson PA, Harris M, Hedlund R, Harrop J, Dvorak M, Wood K, Fehlings MG, Fisher C, Lehman RA Jr, Anderson DG, Bono CM, Kuklo T, Oner FC. The thoracolumbar injury severity score: a proposed treatment algorithm. J Spinal Disord Tech. 2005;18(3):209–15.

L1 Burst Fracture

12

Abbreviations

L5/S1	Fifth lumbar and first sacral vertebrae
T12–L1	12th Thoracic to 1st lumbar vertebrae
TLICS	Thoracolumbar injury classification and severity score

Clinical Presentation

M30, RH, previously healthy, sustained lower back injury whilst sledging and presented with severe low back pain and urinary retention, o/e normal LL power, bilateral L5/S1 hyperesthesia, altered perineal sensation, reflexes slightly increased

Clinical picture consistent with conus medullaris syndrome

Differential Diagnosis

1. Thoraco-Lumbar fracture/dislocation
2. Traumatic disc prolapse
3. Spinal haematoma

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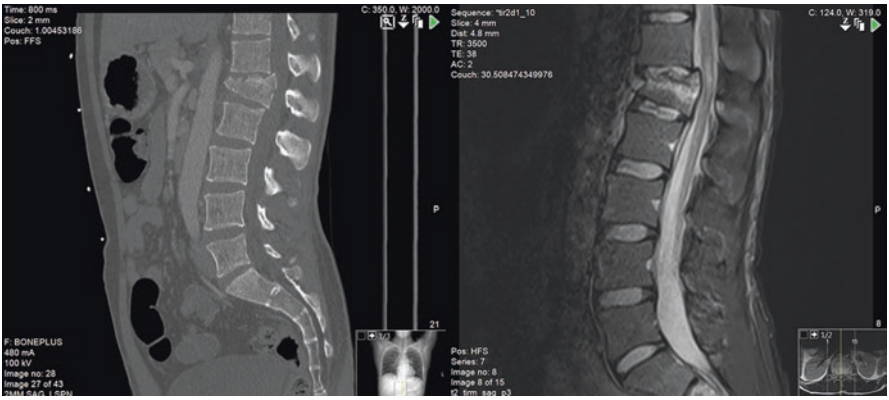
Investigations

CT and MRI Spine revealed L1 burst fracture with retropulsion into the spinal canal and conus medullaris signal change consistent with traumatic contusion



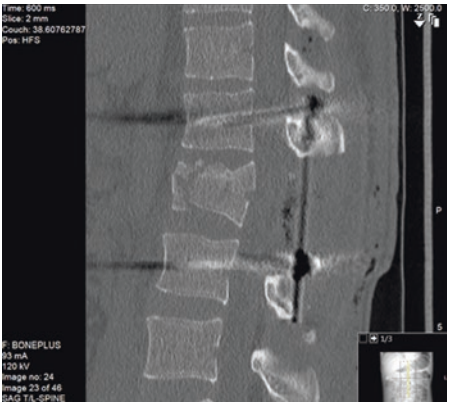
Management

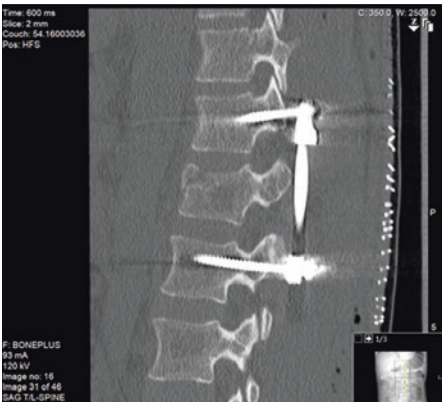
Underwent posterior lumbar decompression and pedicle screw fixation T12–L2

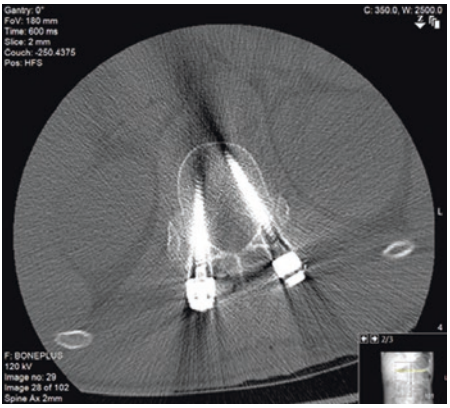
Outcome

Discharged home after 10D with training for self management of residual bladder/bowel dysfunction.

Post op imaging confirmed adequate neuronal decompression and satisfactory metal work placement.







Evidence

TLICS

Lumbo-Sacral Fracture

13

Abbreviations

ADF	Ankle dorsiflexion
EHL	Extensor hallucis longus
L5/S1	Fifth lumbar and first sacral vertebrae
m	Months
TLICS	Thoracolumbar injury classification and severity score
TLSO	Thoraco-lumbar spinal orthosis

Clinical Presentation

F22, RH, previously healthy, sustained polytrauma after jump from three storey building in an attempt to escape from assault, upon admission intact level of consciousness, c/o lumbosacral pain and Right L5/S1 dermatomal distribution loss of pin prick sensation and weakness of EHL/ADF 3/5

Traumatic lumbo-sacral radiculopathy

Differential Diagnosis

1. Lumbo-Sacral fracture/dislocation
2 Traumatic disc prolapse
3. Spinal haematoma

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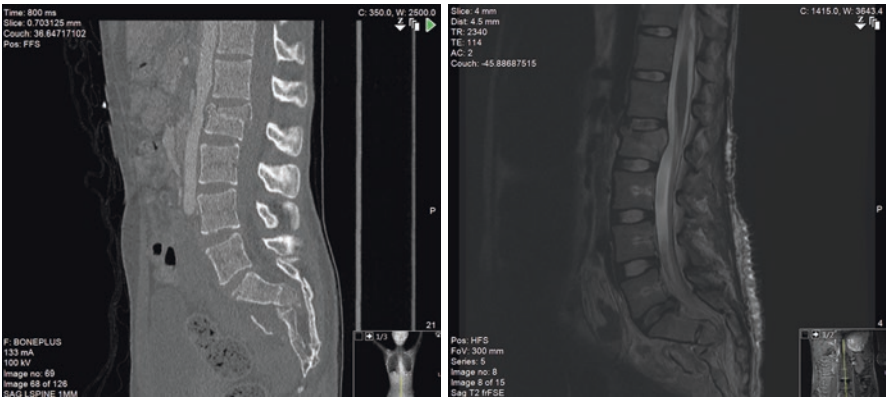
Investigations

Trauma whole body CT identified the following injuries;

- pelvic fracture with large haematoma
- left open comminuted medial calcaneal fracture and talus
- right closed calcaneal fracture
- bilateral mandibular fractures

L2 burst fracture and sacral displaced fracture compromising the sacral canal

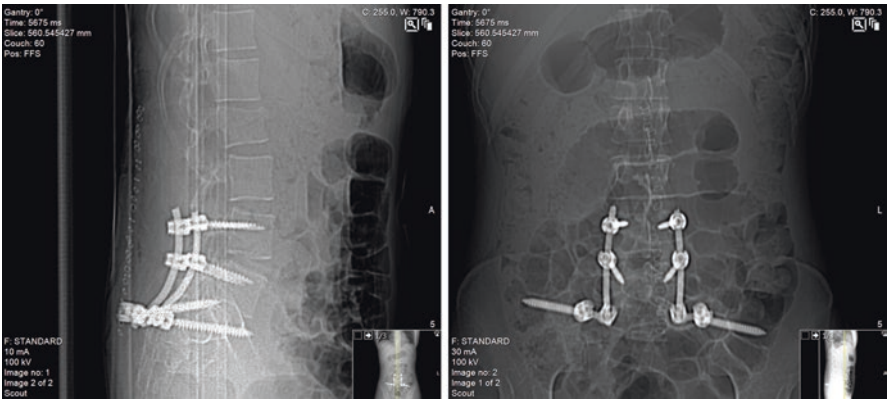


Management

Underwent Iliolumbar fixation (L4-pelvis) for sacral fracture and conservative management with 3m TLSO brace for L2 fracture, also orthopaedic and maxillofacial surgeries for the other injuries

Outcome

After prolonged inpatient stay and neuro rehabilitation she was discharged eventually home minimally symptomatic and neurologically intact and the metalwork was removed electively after 1 year.



Evidence

TLICS

Part III

NeuroVascular

Spontaneous Intracerebral Haematoma

14

Abbreviations

AcommA	Anterior communicating artery aneurysm
ASDH	Acute sub-dural haematoma
AVM	Arteriovenous malformation
dAVF	Dural ateriovenous fistula
CTH	Computed tomography head
CTA	Computed tomography angiogram
h/a	Headache
ICH	Intra-cerebral haematoma
I+V+S	Intubated and ventilated and sedated
MCA	Middle cerebral artery
PERLA 3+	Pupils equal and reactive to light and accommodation and size is 3 mm
PMH	Past medical history
STICH	Surgical treatment for intracerebral haemorrhage trial (number I and number II)

Clinical Presentation

M67, without significant PMH, construction manager, lives on his own, suffered sudden severe h/a followed by decreased level of consciousness GCS E1 V1 M5 and R sided weakness and then deteriorated rapidly to E1 VT M2, I+V+S, PERLA 3+

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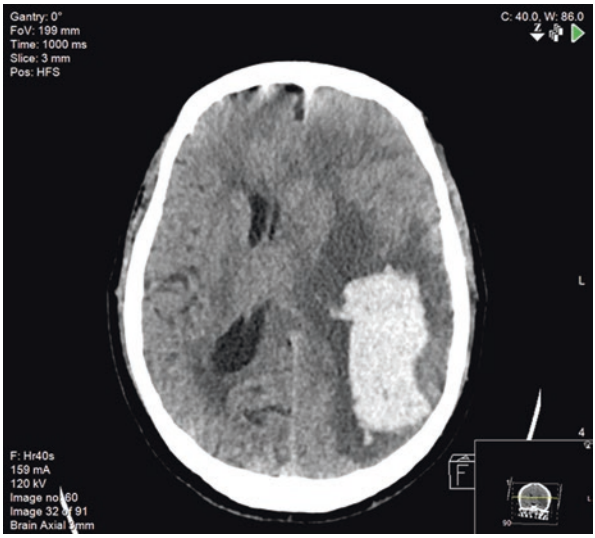
Differential Diagnosis

1. aneurysmal SAH with L MCA ICH
2. Spontaneous L ICH (HTN, AVM, Tumour, Vasculitis, Coagulopathy)
3. Spontaneous L ASDH (dAVF)

Investigations

CTH revealed large left parietal ICH with significant mass effect
CTA ruled out underlying related vascular malformation



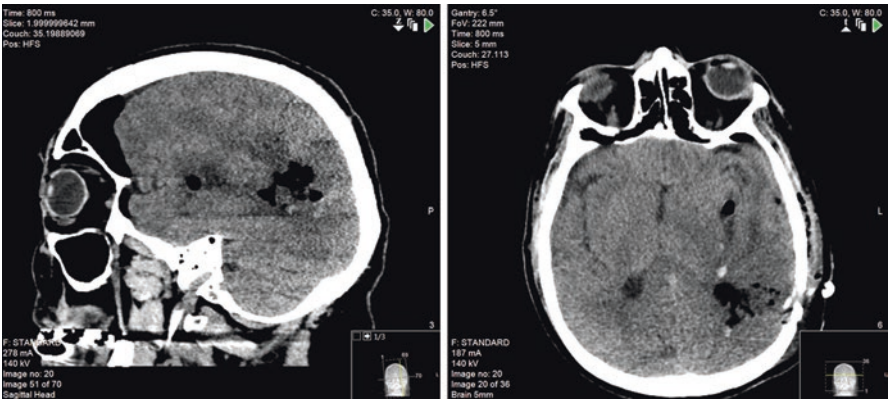
Management

Left Parietal Mini Craniotomy for Evacuation of ICH

Outcome

Complete removal of ICH and relief of mass effect

On transfer to neuro rehabilitation unit, he had a tracheostomy in situ and was opening his eyes spontaneously with localising motor response and right sided hemiplegia. CTA and DSA head showed only an incidental 1.2 cm ACommA aneurysm which was not the cause of this bleed.



Evidence

STICH I, II

Basal Ganglia ICH with Hydrocephalus

15

Abbreviations

AVM	Arteriovenous malformation
BP	Blood pressure
CSF	Cerebrospinal fluid
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
EVD	External ventricular drain
h/a	Headache
HCP	Hydrocephalus
HTN	Hypertension
ICH	Intracerebral haematoma
ICP	Intracranial pressure
IVH	Intraventricular haemorrhage
MCA	Middle cerebral artery
w	Weeks

Clinical Presentation

M47, RH, lives on his own, works in a restaurant, background of HTN, non compliant with medication, presents with sudden severe h/a, drowsiness, confusion and left sided hemiplegia, o/e GCS E3 V4 M6, left sided hemiplegia, gaze deviation to the right, PERLA 3+, BP 170/110

Right hemispheric acute vascular event with raised ICP

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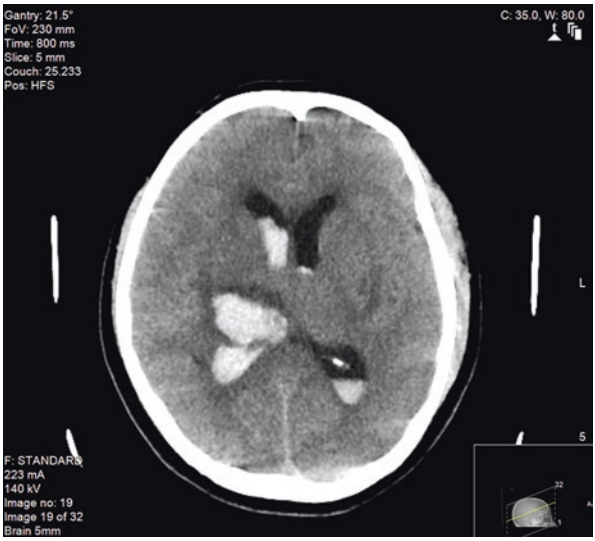
Differential Diagnosis

1. R Basal ganglia HTN ICH ± IVH and HCP
2. SAH and R sylvian ICH from R MCA aneurysm rupture
3. ICH from AVM, tumour

Investigations

CTH showed R thalamic ICH with intraventricular extension and HCP most likely of HTN aetiology

CTA and DSA did not demonstrate underlying vascular abnormality

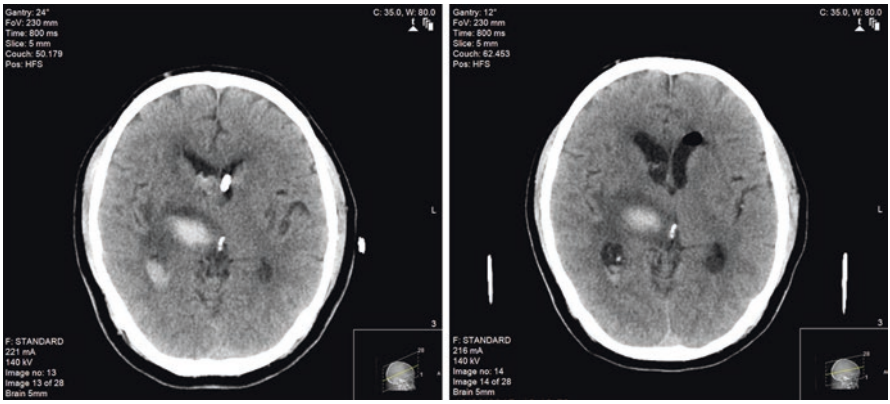


Management

EVD for HCP

Stroke management of HTN (do not overtreat to avoid secondary injury from hypoperfusion)

Deep seated basal ganglia ICH is not a favourable surgical target, evacuate clot only in young patients with significant mass effect as a life saving procedure and not for reversal of neurological deficit



Outcome

After 1w of external csf drainage the EVD was challenged and removed successfully without need for permanent csf diversion, patient remained with left sided hemiplegia

Posterior Fossa ICH with Hydrocephalus

16

Abbreviations

CTA	Cerebral tomography angiogram
CSF	Cerebrospinal fluid
h/a	Headache
HTN	Hypertension
HCP	Hydrocephalus
ICH	Intracerebral haematoma
IV	Intravenous
n/v	Nausea and vomiting
PF	Posterior fossa
R F EVD	Right frontal external ventricular drain
VPS	Ventriculoperitoneal shunt

Clinical Presentation

M71, RH, retired civil servant, non smoker, only medical background of HTN, presents with sudden severe h/a, n/v, ataxia and left sided incoordination with subsequent confusion and drowsiness, blood pressure 230/120

Sudden presentation with left cerebellar signs so probably vascular event in PF

Differential Diagnosis

1. Cerebellar ICH secondary to HTN or underlying vascular malformation with secondary HCP
2. Cerebellar ischaemic stroke with secondary HCP (usually h/a not presenting feature)

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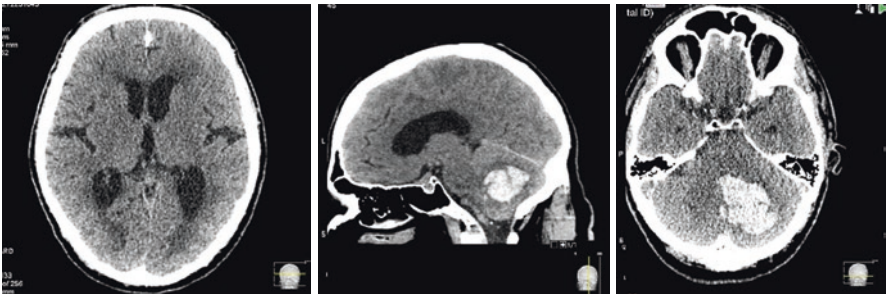
Investigations

CTH revealed PF ICH with obstructive HCP, CTA ruled out underlying vascular malformation



Management

Underwent urgent insertion of R F EVD and posterior fossa craniectomy and evacuation of haematoma

HTN titrated cautiously with iv labetalol infusion aiming BP 180/110

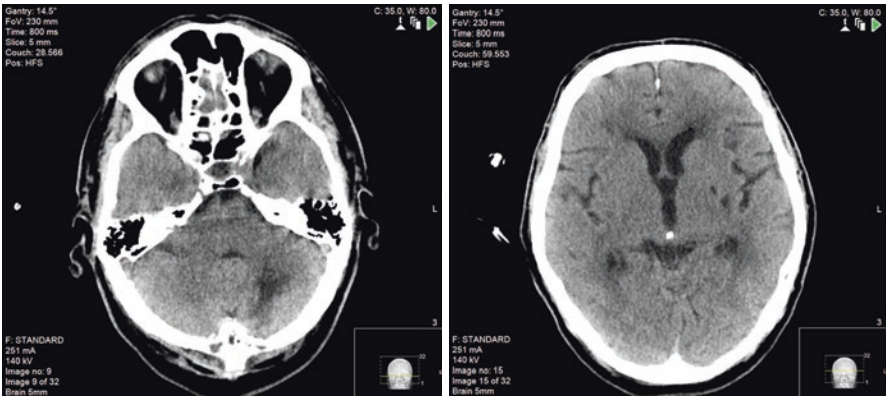
Be careful with only EVD for PF pathology as overdrainage can cause upward herniation, Also consider PF decompression and clot evacuation. Senior opinion is vital evaluate pre-operatively the perimesencephalic cisterns, especially quadrigeminal cistern

Outcome

Post op imaging revealed adequate decompression of posterior fossa and relief of HCP.

EVD was challenged and removed successfully without need for permanent csf diversion (VPS).

Recovered with residual balance and left sided co-ordination impairment.



Subarachnoid Haemorrhage WFNS II: ACommA Aneurysm

17

Abbreviations

AcommA	Anterior communicating artery aneurysm
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
h/a	Headache
hrly	Hourly
HDU	High dependency unit
HGV	Heavy goods vehicle
HTN	Hypertension
ICA	Internal carotid artery
ICH	Intra-cerebral haematoma
ISAT	International subarachnoid aneurysm trial
IV fluids	Intravenous fluids
o/e	On examination
MRA	Magnetic resonance angiogram
N/V	Nausea and vomiting
VA	Vertebral artery
VST	Venous sinus thrombosis
WFNS	World Federation of Neurosurgeons grading of sub-arachnoid haemorrhage

Clinical Presentation

41M RH HGV driver, heavy smoker and HTN, presents with sudden severe h/a and n/v, o/e GCS E3 V4 M6, no limb or visual deficit

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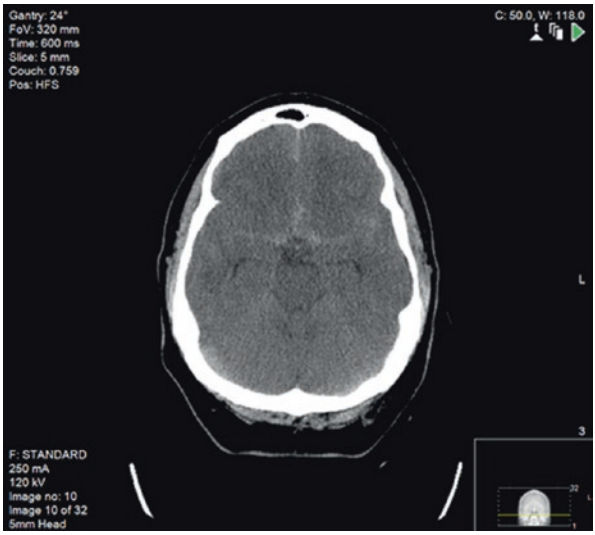
Differential Diagnosis

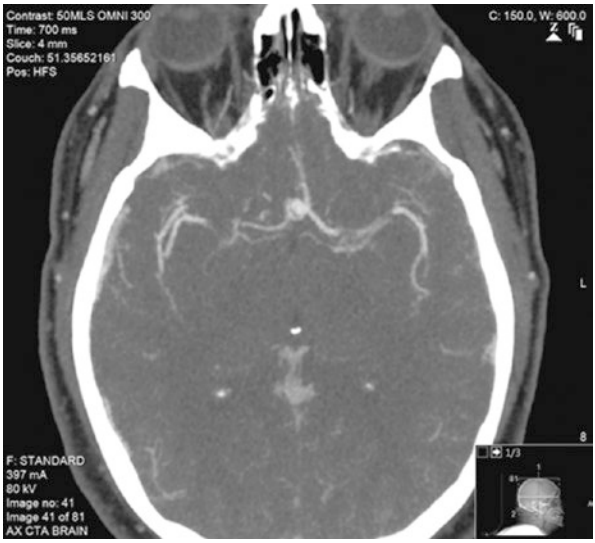
1. aneurysmal SAH
2. ICH (in non eloquent area since no lateralising signs)
3. VST
4. Pituitary apoplexy (but no visual deficit)
5. Haemorrhage into tumour
6. VA or ICA dissection

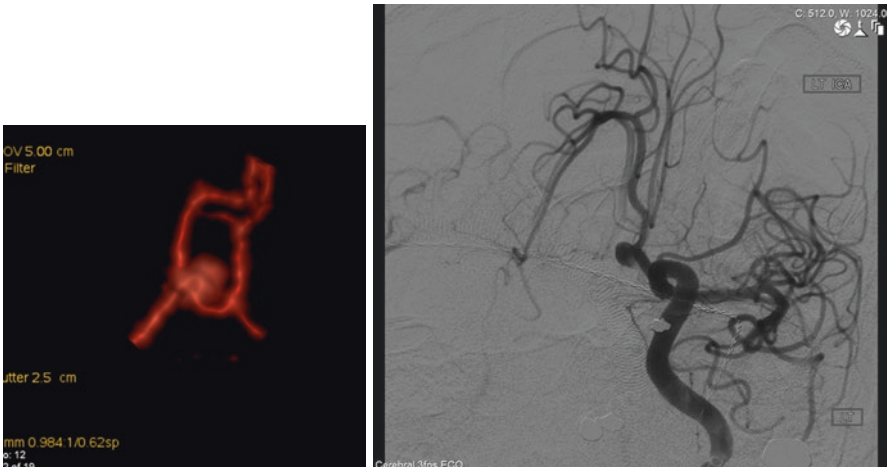
Investigations

CTH revealed basal SAH with extension in interhemispheric and sylvian fissures predominantly



CTA confirmed AcommA aneurysm





Management

SAH WFNS II

Admitted to a hdu bed, bed rest, iv fluids, analgesia, nimodipine 60 mg 4hrly

Underwent successful endovascular coil embolization of the ruptured aneurysm

Outcome

After 10D inpatient stay and gradual mobilisation after first week, he was eventually discharged home asymptomatic and intact on a total of 21D nimodipine po 60 mg 4hrly. He remained under follow up with the Neuro Vascular Service and surveillance imaging with MRA has not demonstrated re canalisation of the aneurysm.

Evidence

ISAT
- WFNS grading
- Fisher grading

Subarachnoid Haemorrhage WFNS V: MCA Aneurysm with Sylvian ICH

18

Abbreviations

ASDH	Acute sub-dural haematoma
BRAT	The Barrow Ruptured Aneurysm Trial
CARAT	Cerebral Aneurysm Rerupture After Treatment Study
CTA	Computed tomography angiogram
CTH	Computed tomography head
h/a	Headache
HTN	Hypertension
HTN BG ICH	Hypertensive basal ganglia intra-cerebral haematoma
ICH	Intra-cerebral haematoma
ICP	Intra-cranial pressure
ICU	Intensive care unit
ISAT	International Subarachnoid Aneurysm Trial
I+V+S	Intubated + ventilated + sedated
MCA	Middle cerebral artery
MLS	Midline shift
SAH	Sub-arachnoid haemorrhage

Clinical Presentation

M51, RH, police officer, lives with wife, pmh of HTN, presents with sudden severe h/a and rapid deterioration to GCS E1 V1 M4, R pupil fixed and dilated

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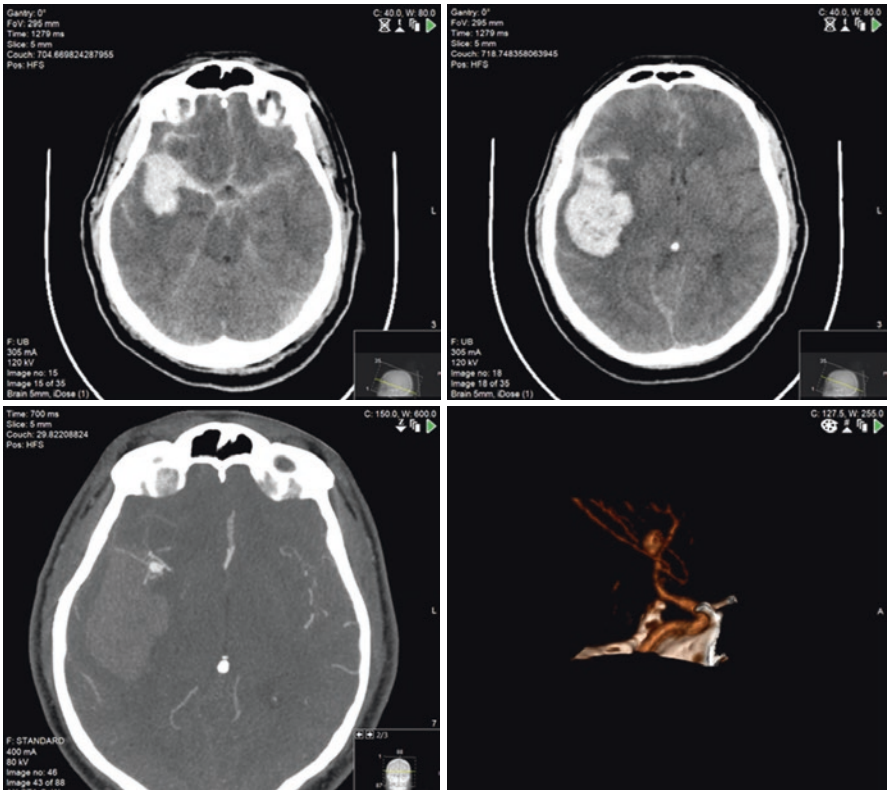
Differential Diagnosis

1. aneurysmal SAH
2. HTN R BG ICH
3. ICH in tumour
4. Spontaneous ASDH

Investigations

CTH, CTA demonstrated diffuse basal SAH with R sylvian ICH causing MLS secondary to ruptured R MCA bifurcation aneurysm

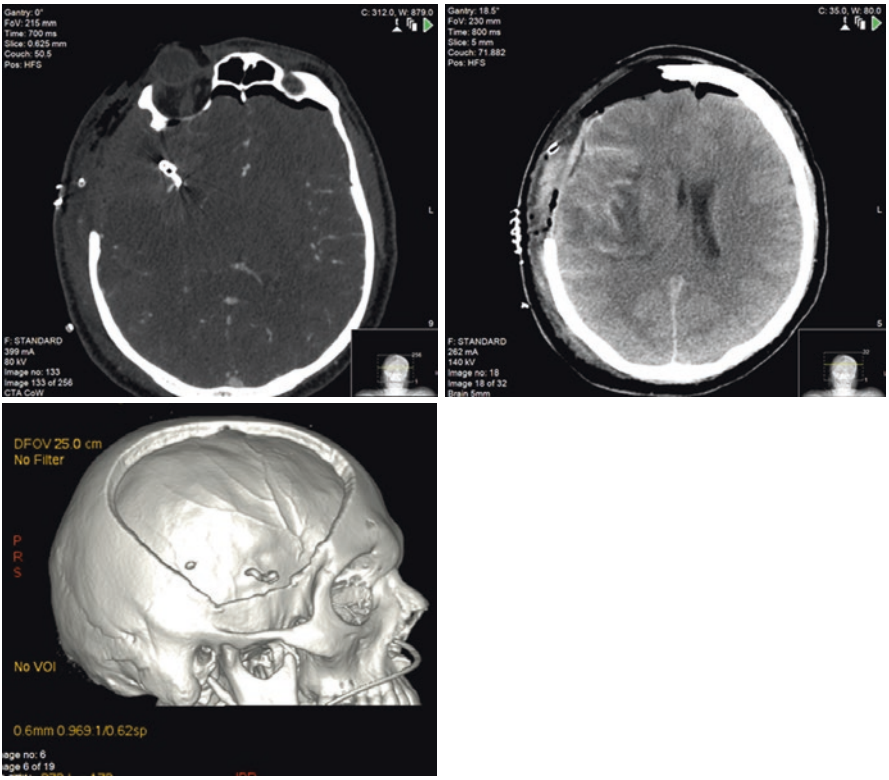


Management

– I+V+S, hyperosmotic therapy with mannitol or hypertonic saline
– Emergency Right sided Decompressive Craniectomy, evacuation of haematoma and clipping of ruptured MCA bifurcation aneurysm

Outcome

Post op imaging revealed adequate haematoma evacuation and secured aneurysm



Prolonged ICU stay for management of raised ICP and ventilation support, long term severe disability with cognitive impairment and left sided hemiparesis

Evidence

ISAT I

Molyneux AJ, Kerr RS, Stratton I, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomized trial. Lancet. 2002;360:1267–74.

Molyneux AJ, Kerr RS, Yu LM, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomized comparison of effects on survival, dependency, seizures, rebleeding, subgroups and aneurysm occlusion. Lancet. 2005;366:809–17.

Molyneux AJ, Kerr RS, Birks J, et al. ISAT Collaborators. Risk of recurrent subarachnoid haemorrhage, death, or dependence and standardized mortality ratios after clipping or coiling of an intracranial aneurysm in the International Subarachnoid Aneurysm Trial (ISAT): long-term follow-up. Lancet Neurol. 2009;8:427–433.

CARAT

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BRAT

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Painful Third Cranial Nerve Palsy: PCommA Aneurysm

19

Abbreviations

CC–fistula	Carotid cavernous fistula
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
DSA	Digital subtraction angiogram
ISAT	International subarachnoid aneurysm trial
ISUIA	International Study of Unruptured Intracranial Aneurysms
MCA	Middle cerebral artery
PcommA	Posterior communicating artery
SAH	Subarachnoid haemorrhage
SCA	Superior cerebellar artery

Clinical Presentation

F54, RH, lives with parents, carer of father, smoker, pmh of hypothyroidism on levothyroxine, presents with acute painful right sided ptosis, o/e, GCS E4 V5 M6, no limb deficit, complete R III nerve palsy with fixed and dilated pupil

Differential Diagnosis

1. Expanding R PcommA aneurysm, SCA, MCA
2. R cavernous sinus syndrome from C-C fistula, tumour (pituitary adenoma, meningioma)
3. diabetic neuropathy (painless)
4. partial ptosis and small pupil from Horner's syndrome
5. note the pupil is midsize in cavernous lesions due to compression on both sympathetic and parasympathetic fibers

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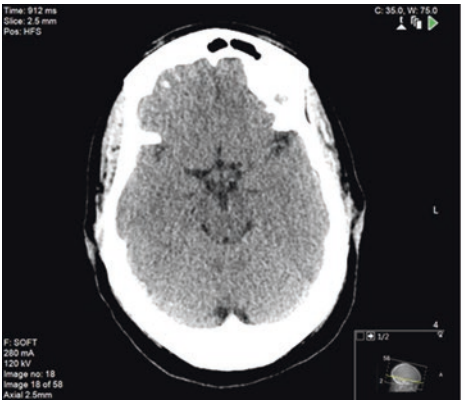
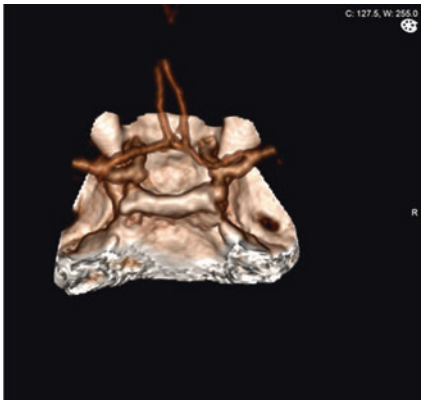
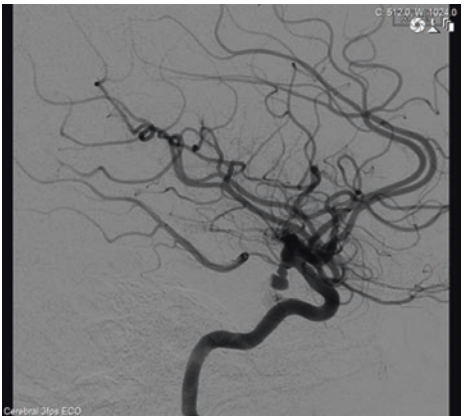
Investigations

– CTH did not show obvious SAH, LP did not show SAH (spectrophotometry for bilirubin)
– CTA and DSA showed 5.2 mm R PcommA aneurysm and mirror left 3 mm

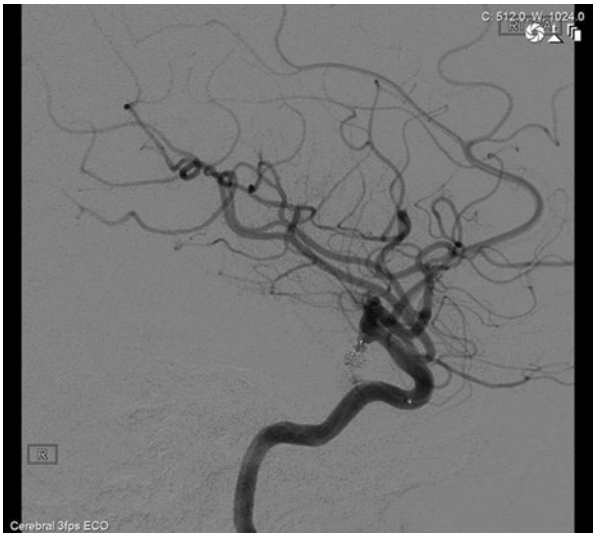






Management

Coil embolization of R PcommA aneurysm



Outcome

Neurovascular follow up with MRA shows stability of small left untreated PcommA unruptured aneurysm and securely occluded R PcommA aneurysm

The third cn palsy recovered partially and due to residual diplopia only on upward gaze. Ophthalmology did not proceed with surgery

Evidence

- ISAT
- ISUIA

Note the painful third cn palsy in the context of aneurysmal compression represents medical emergency as a-SAH and requires prompt treatment.

Arteriovenous Malformation

20

Abbreviations

ASDH	Acute sub-dural haematoma
AVM	Arteriovenous malformation
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
DSA	Digital subtraction angiogram
EtoH	Alcohol
EVD	External ventricular drain
h/a	Headache
HCP	Hydrocephalus
HDU	High dependency unit
HTN	Hypertension
ICH	Intra-cerebral haematoma
ICP	Intra-cranial pressure
MRC grading	Medical research council muscle grading
OT	Occupational therapy
PT	Physiotherapy
SRS	Stereotactic radiosurgery
VST	Venous sinus thrombosis

Clinical Presentation

M17, RH, student, previously fit and well, whilst at school suffered sudden severe h/a and subsequent decreased level consciousness (E1V1M5) and right sided weakness 4/5 MRC

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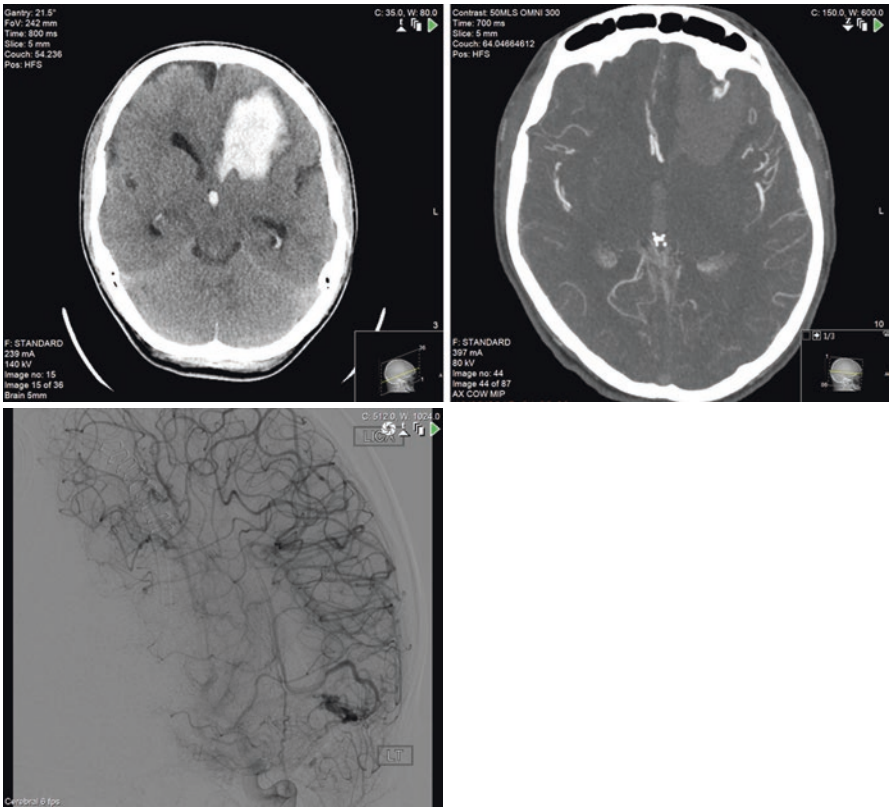
Differential Diagnosis

1. ICH (AVM, HTN, tumour, aneurysm, drugs, EtoH, VST, moya-moya, coagulopathy)
2. Spontaneous ASDH

Investigations

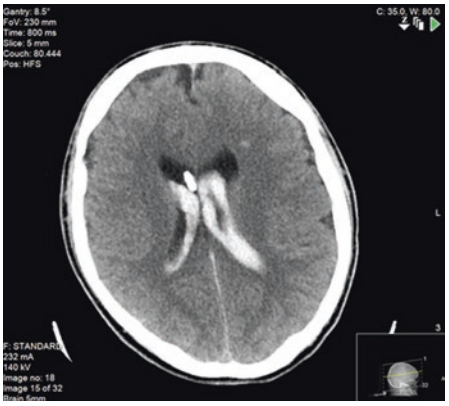
CTH, CTA, DSA revealed left frontal intracerebral haemorrhage with intraventricular extension and hydrocephalus secondary to a ruptured peripheral left frontal AVM.

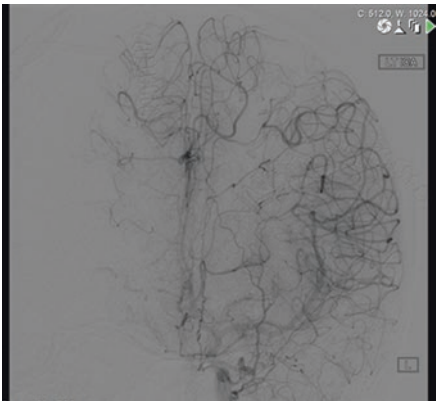


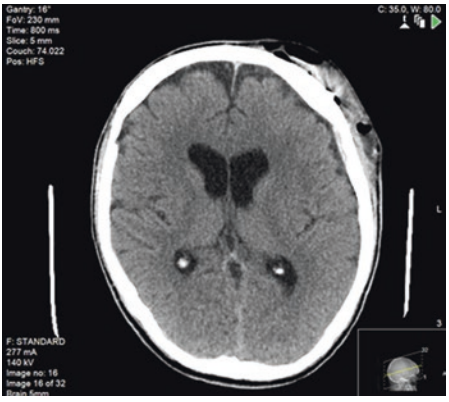
Management

1. Insertion of right frontal EVD in order to relieve HCP and control raised ICP
2. Left pterional craniotomy and excision of AVM
   - ABCDE protocol assessment
   - EVD for HCP or partial evacuation of ICH if patient significantly compromised by raised ICP
   - Neuro Vascular MDT as management of AVMs is a multi modality approach

   Surgery, Endovascular therapy, SRS or combination







Outcome

Post operatively he was transferred back to ITU initially, CTA and DSA confirmed complete excision of the AVM and gradually he was weaned off sedation and eventually extubated and stepped down to HDU where his EVD was successfully challenged and removed without clinical or radiological evidence of recurrent HCP. Under intense PT/OT/SALT he improved significantly being GCS E4 V5 M6 with residual mild expressive dysphasia and mild right sided weakness 4+/5 and he was transferred for further Neuro Rehabilitation.

After 6m he was neurologically intact and asymptomatic and f/u DSA did not show residual or recurrent A-V shunting.

Evidence[1]

ARUBA

Al-Shahi R, Bhattacharya JJ, Currie DG, Papanastassiou V, Ritchie VR, Roberts RC, Sellar RJ, Warlow CP, SIVMS Collaborators. Prospective, population-based detection of intracranial vascular malformations in adults: the Scottish Intracranial Vascular Malformation Study (SIVMS). Stroke. 2003;34:1163–9.

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[1] Grading systems (Spetzler-Martin, Spetzler-Ponce, Lawton-Young, Pollock-Flickinger)

Spinal Dural Arteriovenous Fistula

21

Abbreviations

AV	Aterio-venous
L1	First lumbar vertebrae
MRA	Magnetic resonance angiogram
MRI	Magnetic resonance imaging
MRI TRICKS	Magnetic resonance imaging with Time Resolved Imaging of Contrast KineticS, an MRI sequence that provides MR angiography with excellent spatial and temporal resolution
mscc	Metastatic spinal cord compression
T6	'Sixth' thoracic vertebrae (same applies to other T'$' to corresponding number)

Clinical Presentation

F73, retired pharmacist, pmh of lymphoedema and bilateral hip replacements, presents with 4m of gradually worsening mobility and sphincteric dysfunction. Last month more rapid deterioration and last week double incontinence and unsteadiness o/e lower thoracic severe myelopathy with loss of proprioception, weakness 4/5, double incontinence, can feel catheter tug

Thoracic myelopathy

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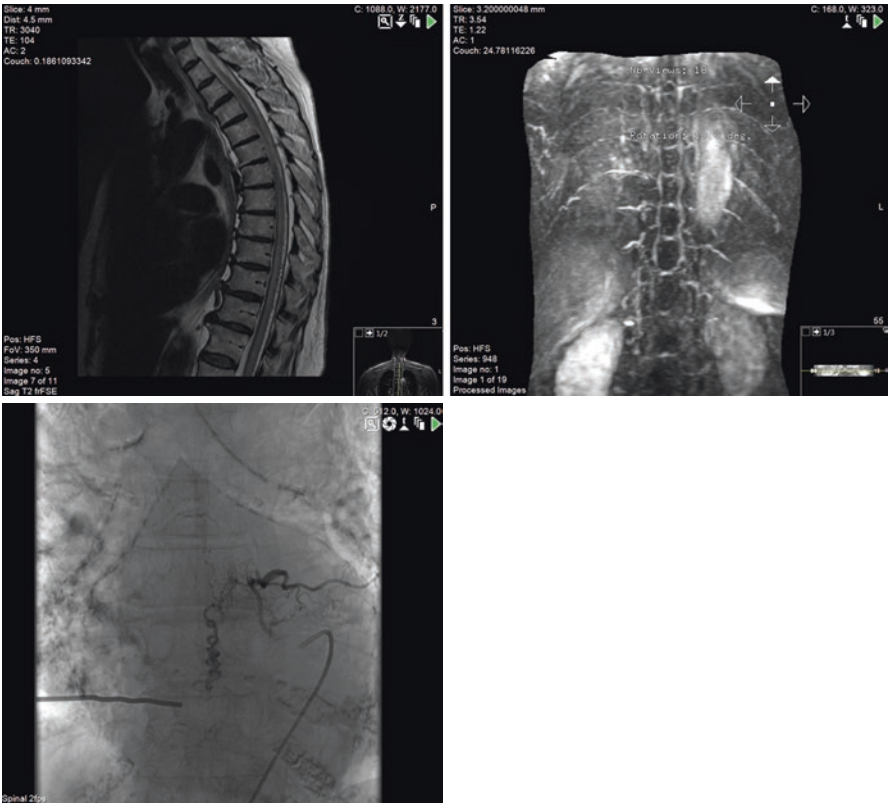
Differential Diagnosis

1. Thoracic spine MSCC
2. Thoracic spine meningioma
3. Thoracic disc prolapse or stenosis
4. Thoracic dural A-V fistula
5. Inflammatory/demyelination process

Investigations

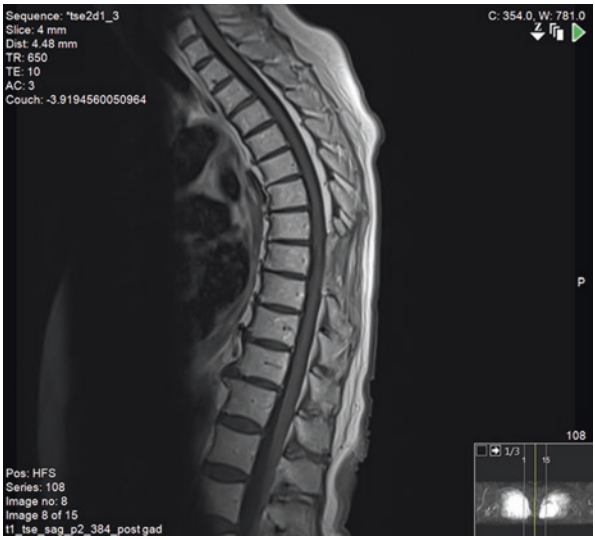
MRI spine revealed cord signal change consistent with oedema secondary to venous hypertension from T6 to L1 and numerous punctate flow voids predominantly at T9–10, appearances consistent with dural arterio-venous fistula. MRI supplemented with TRICKS sequence which demonstrated early filling caudally directed draining vein with the point of fistulation likely at T8 level on the left which was confirmed with selective spinal catheter angiogram



Management

Underwent uneventful T8 laminectomy and disconnection of dural A-V fistula.

Post op imaging with MRI/MRA confirmed no residual A-V shunting and improvement of cord oedema.



Outcome

After 2w of inpatient neuro rehabilitation the thoracic myelopathy improved significantly and she was discharged home mobilising with one stick and in 3m post op independently with social bladder control.

American-English-French Connection classification (I—dAVF, II—intramedullary spinal glomus AVM, III—juvenile spinal AVM, IV—perimedullary AVM)

Coup de poignard of Michon (sudden severe back pain with SAH)

Foix-Alajouanine syndrome (subacute necrotic myelopathy)

Evidence

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Malignant MCA Syndrome

22

Abbreviations

ACA	Anterior cerebral artery
A+E	Accident and emergency department
ASDH	Acute subdural haematoma
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
CT Perfusion	Computed tomography perfusion scan (helps identify areas of ischaemic penumbra from established areas of infarct)
DSA	Digital subtraction angiogram
EtoH	Alcohol
GH	Growth hormone
HASU	Hyper-acute stroke unit
HIV	Human immunodeficiency virus
HTN	Hypertension
ICH	Intracerebral haematoma
IHD	Ischaemic heart disease
INR	International normalized ratio (measurement of extrinsic pathway clotting)
ICU	Intensive care unit
MCA	Middle cerebral artery
MI	Myocardial infarction
MLS	Midline shift
MRA	Magnetic resonance angiogram
MRC grading	Medical research council muscle grading
MRI	Magnetic resonance imaging
MR perfusion	Magnetic resonance perfusion imaging (helps identify areas of ischaemic penumbra from established areas of infarct)

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MRI DWI	Diffusion weighted magnetic resonance imaging (looks at restricted movement of hydrogen molecules and is useful in identifying infarct, intracerebral abscess and tumours with high cellularity)
NIHSS	National Institutes of Health Stroke Scale
PCA	Posterior cerebral artery
PERLA	Pupils equal and reactive to light and accommodation
SAH	Subarachnoid haemorrhage

Clinical Presentation

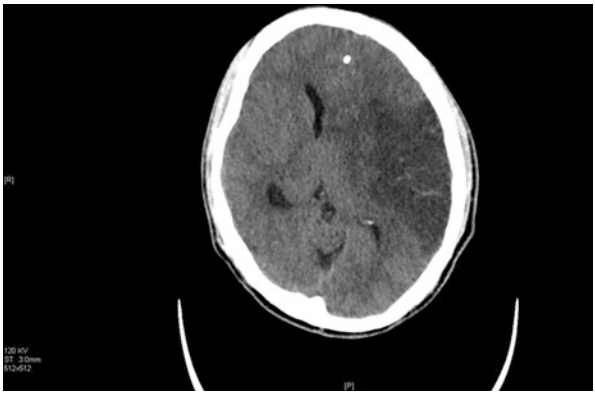
M35, RH, unemployed, with background of HIV, anti-phospholipid syndrome and IHD/MI 1 year ago, found unresponsive in his house after use of recreational drugs and EtoH, upon admission at A+E GCS E4 V aphasic M6 with Right sided weakness, normally on warfarin and clopidogrel but non compliant with medication, INR 1.2, deteriorated rapidly to GCS E3 V aphasic M5 with right sided hemiplegia, PERLA (3 mm)

Differential Diagnosis

1. Malignant L MCA stroke
2. Left sided ICH, ASDH, aneurysmal SAH with left sided ICH
3. Left sided intracranial abscess or subdural empyema/epidural abscess

Investigations

CTH revealed large wedge shaped left fronto-temporal hypodense area consistent with malignant L MCA stroke causing significant mass effect and MLS

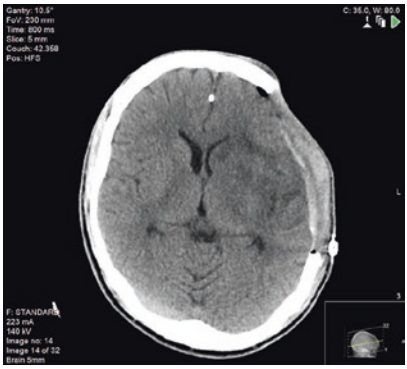


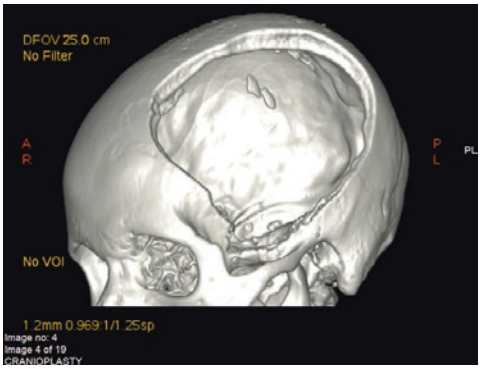
CT, CTA, CT perfusion, MRI, MRI perfusion, DSA
Early CT findings in hyperacute stroke (<6 h);

- hyperdense artery sign (string sign)
- loss of insular ribbon
- attenuation of lentiform nucleus
- loss of grey-white matter interface

Management

Urgent left sided decompressive hemicraniectomy performed followed by ICU stay for 48 h and then step down to level 2 HASU bed for further management under the stroke team. Therapeutic anticoagulation commenced on D3 post op.





Outcome

After prolonged neuro rehabilitation and titanium cranioplasty he recovered significantly with residual expressive dysphasia but good comprehension and mild Right sided hemiparesis m MRC grade 4/5.

Appendix

NICE Guidelines: https://www.nice.org.uk/guidance/cg68/chapter/1-Guidance#specialist-care-for-people-with-acute-stroke

Surgical Referral for Decompressive Hemicraniectomy

People with middle cerebral artery infarction who meet all of the criteria below should be considered for decompressive hemicraniectomy. They should be referred within 24 h of onset of symptoms and treated within a maximum of 48 h.

- Aged 60 years or under.
- Clinical deficits suggestive of infarction in the territory of the middle cerebral artery, with a NIHSS score of above 15.
- Decrease in the level of consciousness to give a score of 1 or more on item 1a of the NIHSS.
- Signs on CTH of an infarct of at least 50% of the MCA territory, with or without additional infarction in the territory of the ACA or PCA on the same side, or infarct volume >145 cm^3 as shown on MRI DWI.

Evidence

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Part IV

Hydrocephalus

Prematurity Related Intracranial Haemorrhage

23

Abbreviations

AF	Anterior fontanelle
AqS	Aqueductal stenosis
EVD	External ventricular drain
ETV	Endoscopic third ventriculostomy
HCP	Hydrocephalus
ICH	Intracerebral haematoma
IVH	Intraventricular haemorrhage
MRI 'feed and wrap'	MRI in a neonate where the infant is fed and then swaddled to encourage it to sleep for the scan
OFC	Occipital frontal circumference
VAS	Ventriculo-arterial shunt
VPS	Ventriculo-peritoneal shunt

Clinical Presentation

New born baby girl at 34w, tense bulging AF, OFC >97th centile, distended scalp veins, managed in the Neonatal Intensive Care Unit, respiratory distress, transient neonatal thrombocytopenia, neonatal hypoglycaemia

Differential Diagnosis

1. Prematurity related germinal matrix haemorrhage (ICH, IVH and HCP)
2. HCP from AqS
3. Dandy Walker syndrome and variants

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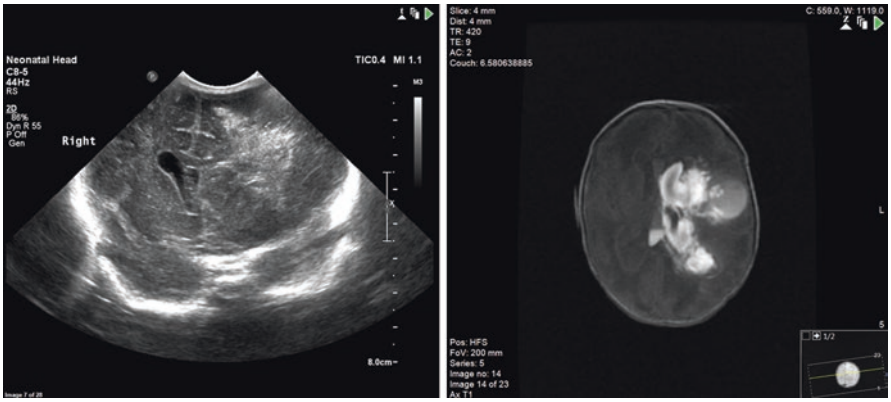
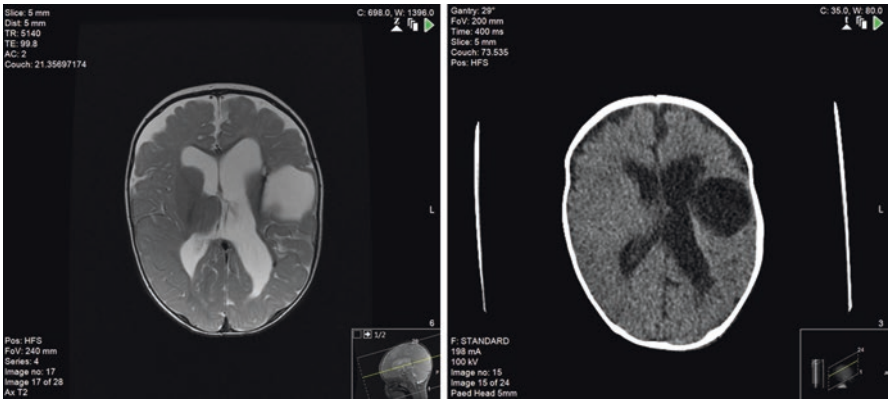
Investigations

– U/S; ventricular indices, MRI feed and wrap revealed left caudate germinal matrix haemorrhage with intraventricular extension and HCP



Management

– serial transfontanelle taps in this case (with or without U/S guidance)
– EVD
– ventriculo-subgaleal shunt until >1.5 kg weight
– shunt procedures (VPS, VAS)
– endoscopic wash out and ETV



Outcome

In this case the progression of HCP was prevented only with serial transfontanelle taps, radiological surveillance ruled out worsening ventriculomegaly and revealed area of encephalomalacia at previous ICH site.

OFC followed normal charts

Mild developmental delay

Evidence

Papile grading system

Chiari II Malformation

24

Abbreviations

aFP	Alpha fetoprotein
AF	Anterior fontanelle
CSF	Cerebrospinal fluid
CTH	Computed tomography head
LL	Lower limb
MRI 'feed and wrap'	MRI in a neonate where the infant is fed and then swaddled to encourage it to sleep for the scan
OFC	Occipital frontal circumference
U/S	Ultrasound
VPS	Ventriculo-peritoneal shunt

Clinical Presentation

New born baby girl, delivered @ 38 + 4 due to premature rupture of the membranes

Antenatal diagnosis of Chiari II [Spinal Bifida, Myelomeningocele, Hydrocephalus (OFC 38 cm)]

O/E Full tense AF, 6 × 5 cm lumbar defect with csf leak, breathing spontaneously but no obvious LL movement

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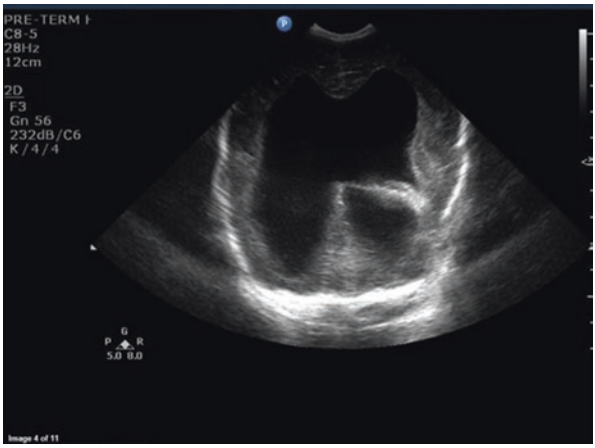
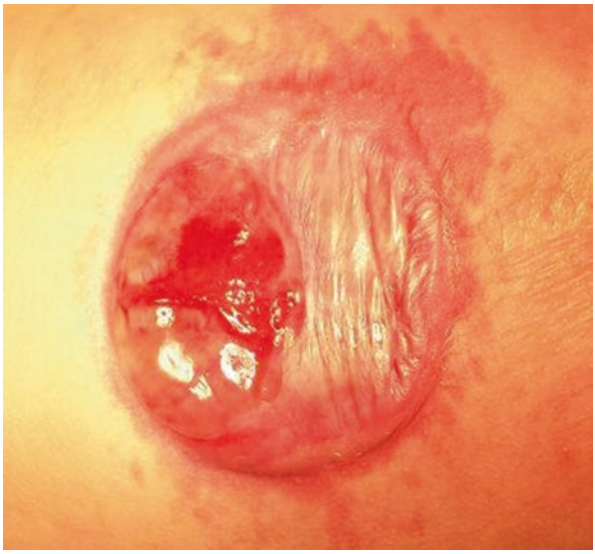
Differential Diagnosis

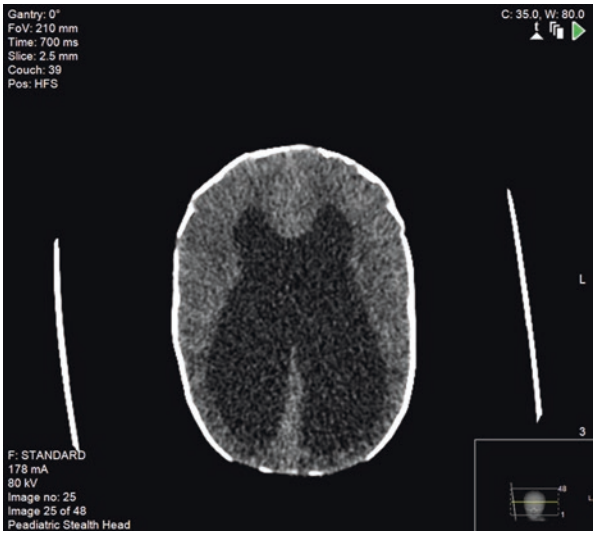
1. Neural tube defects—spina bifida overta (myelomeningocoele, limomeningocoele, dermal sinus)

Investigations

– Antenatal diagnostic tests, fetal MRI, serum aFP, amniocentesis
– Cranial U/S, CTH





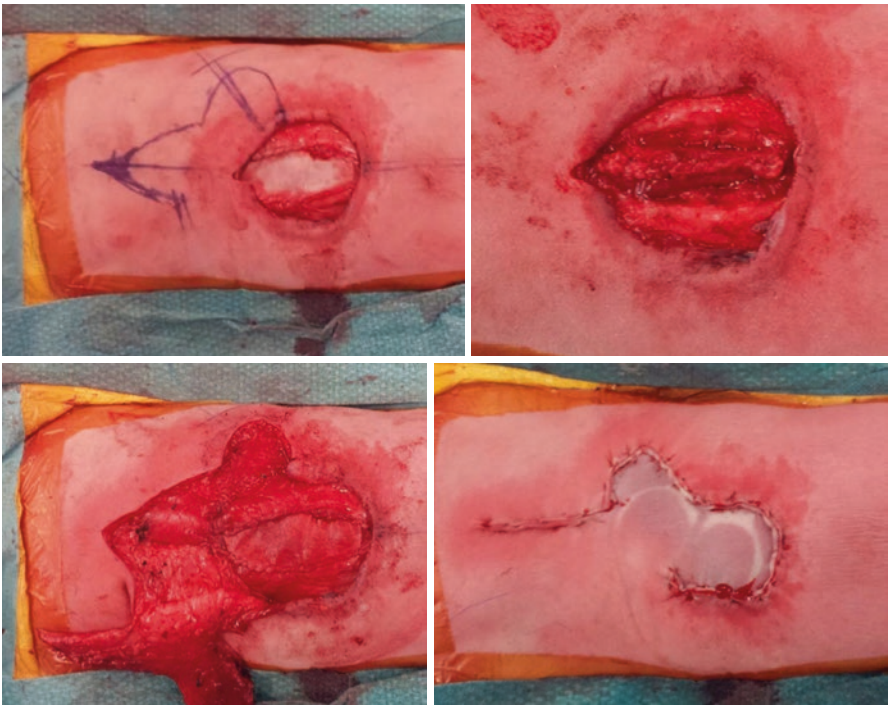


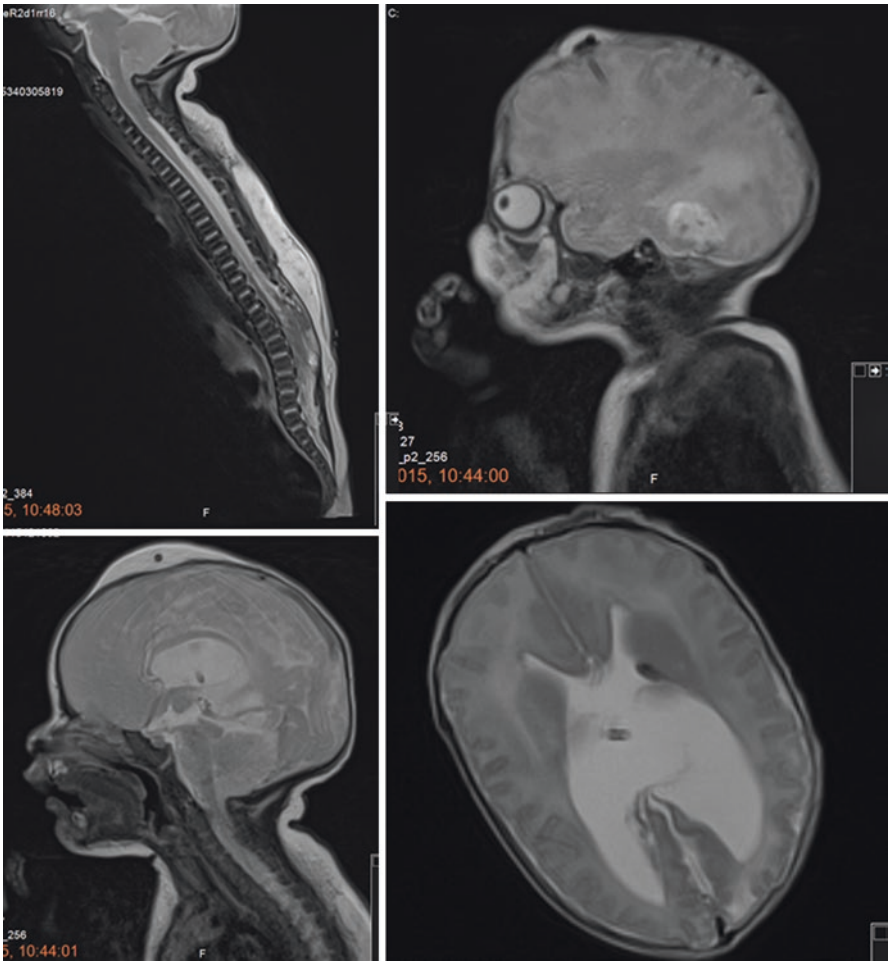
Management

Repair of Myelomeningocele and bi-lobed skin flap and i/o VPS

Whole neuraxis MRI feed and wrap confirmed the Chiari Type II Malformation manifestations;

- hydrocephalus (80%)
- low attachment of tentorium
- tectal beaking and medullary bending
- deficiency of the septum pellucidum
- crowding in the posterior fossa with tonsillar ectopia





Outcome

The lumbar wound healed well without complications

Developmental delay, neuropathic bladder, impaired mobility but crawls efficiently

VPS blocked expressed with lethargy, decreased feeding, vomiting, tense bulging fontanelle, sun setting, confirmed radiologically with increased ventricular size and revised successfully, radiological post op confirmation with decrease in ventricular size and appropriate catheter placement

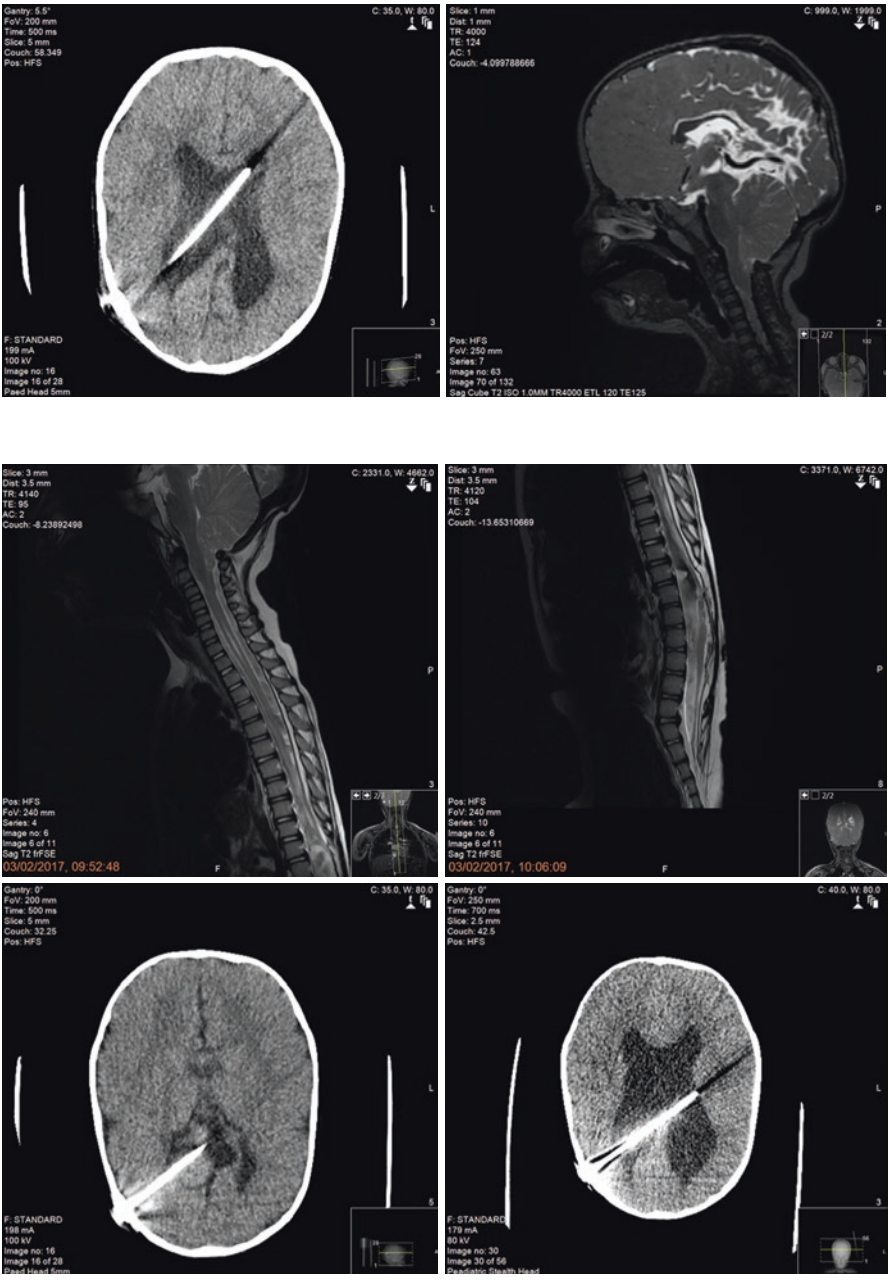

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Image 30 of 56

Aqueduct Stenosis

25

Abbreviations

AqS	Aqueductal stenosis
CSF	Cerebrospinal fluid
ETV	Endoscopic third ventriculostomy
hx	History
h/a	Headache
IIH	Idiopathic intracranial hypertension
ICP	Intracranial pressure
n/v	Nausea and vomiting
MRI	Magnetic resonance imaging
MRI (T2 Sagittal CISS)	Magnetic resonance T2 weighted imaging with a constructive interference in steady state (type of CSF flow study)
VPS	Ventriculo-peritoneal shunt

Clinical Presentation

F43, RH, dog walker, lives on her own, no significant pmh, non smoker, presents with 1 year hx gradually worsening h/a, worse in the morning and relieved with vomiting, accompanied the last month by visual disturbances in the form of flashing lights, o/e intact neurologically except bilateral papilloedema

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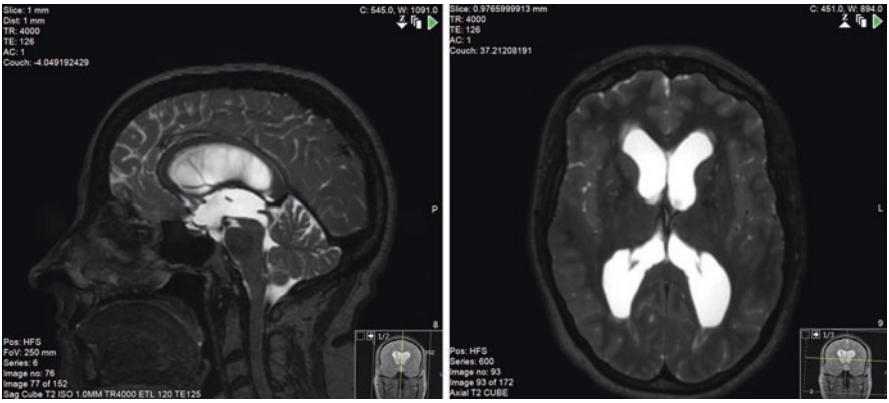
Differential Diagnosis

1. HCP secondary to AqS
2. IIH
3. Midline neoplastic process, intraventricular tumour, frontal meningioma

Investigations

Brain MRI revealed longstanding triventricular HCP secondary to AqS with periventricular lucency

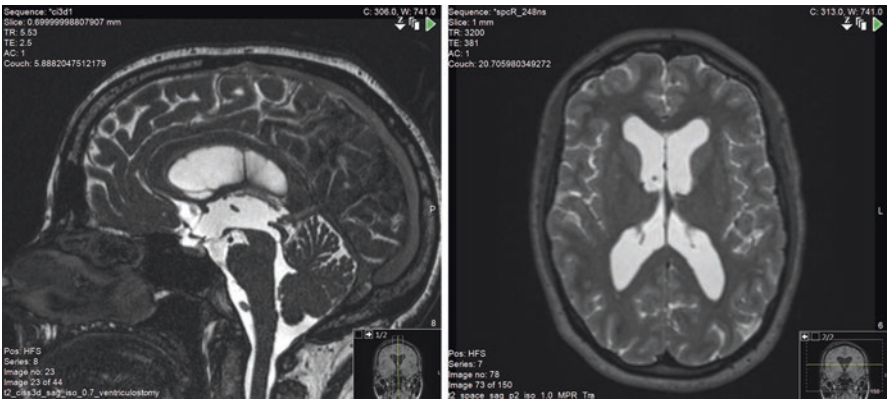


Management

CSF diversion procedure in the form of ETV or VPS

She underwent Image Guided ETV and i/o Ommaya Reservoir

Post op MRI (Sag T2 Ciss) confirmed resolution of the HCP and patent third ventriculostomy



Outcome

Patient improved post operatively with resolution of raised ICP symptomatology (h/a, n/v) and discharged home neurologically intact without papilloedema

Evidence

Kulkarni AV, Riva-Cambrin J, Browd SR. Use of the ETV Success Score to explain the variation in reported endoscopic third ventriculostomy success rates among published case series of childhood hydrocephalus. J Neurosurg Pediatr. 2011;7(2):143–6. https://doi.org/10.3171/2010.11.PEDS10296.

Colloid Cyst

26

Clinical Presentation

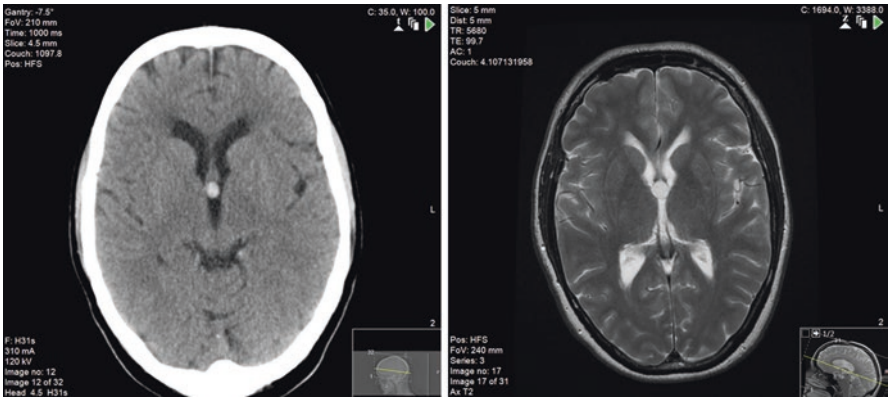
F52, RH, cardiac nurse, healthy, presents with 6m h/o h/a, last 2w c/o blurred vision and unsteadiness, o/e intact level of consciousness, bilateral papilloedema, mild memory deficit

Differential Diagnosis

1. HCP secondary to AqS, Colloid Cyst
2. IIH
3. Midline neoplastic process, intraventricular tumour, frontal meningioma

Investigations

CT and MRI revealed obstructive HCP with PVL secondary to CC



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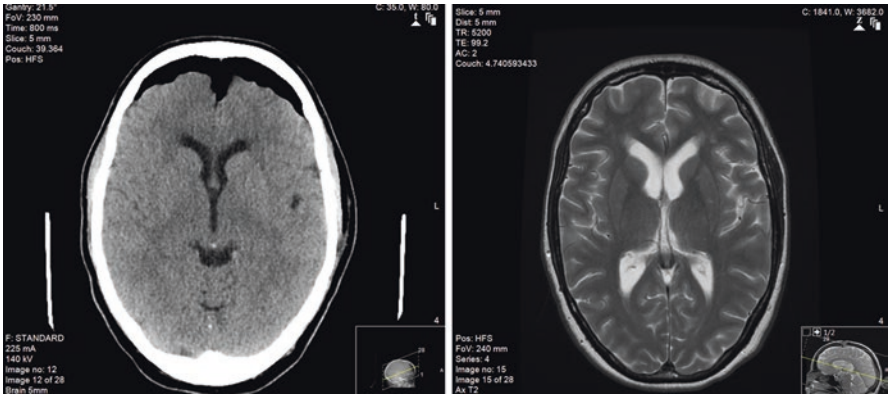
Management

– If patient in extremis from HCP for urgent bilateral EVDs
– Small (<10 mm), asymptomatic, without HCP for radiological surveillance
– If longstanding HCP, symptomatic or >10 mm for excision of colloid cyst
  • transcortical transventricular (especially if large ventricles, risk of Epilepsy 5%)
  • interhemispheric transcallosal (especially if small ventricles, risk of venous infarct or forniceal injury)
  • endoscopic transcortical transventricular resection
– Neuro psychological (cognition) assessment and Neuro Ophthalmology (fundoscopy)



Outcome

EVD for 24–48 h post op
Full recovery
Radiological surveillance

Evidence

Mayo clinic

Idiopathic Intracranial Hypertension

27

Abbreviations

AqS	Aqueductal stenosis
CC	Colloid cyst
CTH	Computed tomography head
h/a	Headache
HCP	Hydrocephalus
IIH	Idiopathic intracranial hypertension
IV	Intra venous
LP	Lumbar puncture
MRI	Magnetic resonance imaging
N/V	Nausea and vomiting
VF	Visual fields
VPS	Ventriculo-peritoneal shunt
VST	Venous sinus thrombosis
w	Weeks

Clinical Presentation

F35, RH, previously healthy, non smoker, on contraceptive pill, presents 3w ago with sudden severe h/a associated with n/v and subsequent 1w ago visual impairment. Referred to NS and blue lighted to the unit. Currently GCS E4 V5 M6, no FND, visual findings as above with peripheral VF restriction and reduced visual acuity

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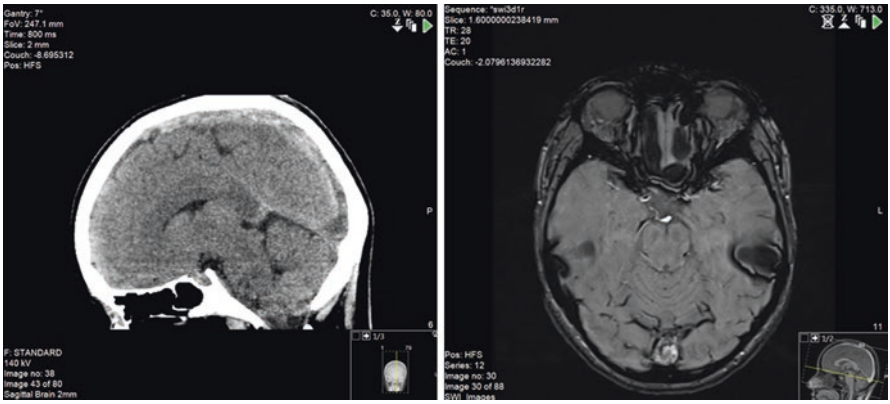
Differential Diagnosis

1. HCP secondary to AqS, CC
2. IIH
3. Midline neoplastic process, intraventricular tumour, frontal meningioma

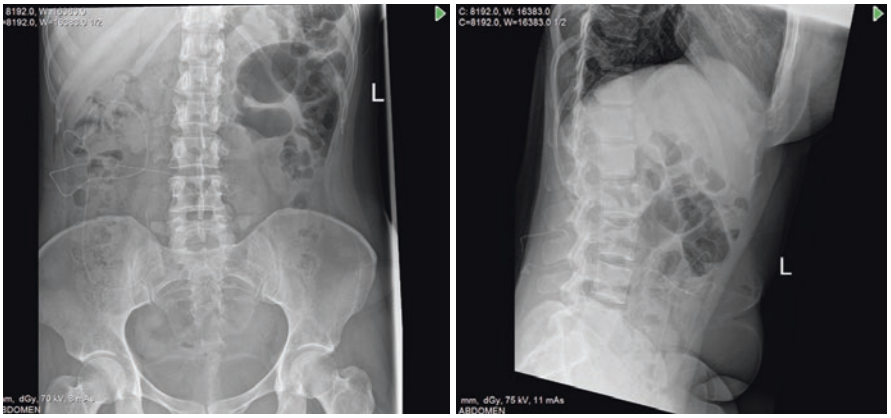
Investigations

Brain imaging with CTH and MRI revealed VST and LP OP above 40, also local ophthalmological assessment revealed bilateral papilloedema and retinal haemorrhages, note in IIH the ventricles are normal size or slit



Management

– improvement of h/a on acetazolamide and started on iv heparin infusion
– insertion of Lumbo–Peritoneal Shunt
– Other options include:
  • VPS
  • bilateral subtemporal decompression
  • optic nerve fenestration



Outcome

- resolution of h/a and normal vision
- initially low pressure syndrome (manage conservatively)
- shunt series reveal appropriate catheter placement in lumbar canal and peritoneum
- upon recurrence of symptoms perform shunt series to r/o malposition/disconnection, LP to measure OP and r/o csf infection, on brain imaging did not expect ventriculomegaly but iatrogenic chiari (tonsillar descent), subdural hygromas or haematomas if overdrainage
- therapeutic anticoagulation or endovascular venous sinus stent for VST

Blocked Shunt

28

Abbreviations

CSF	Cerebrospinal fluid
CTH	Computed tomography head
ETV	Endoscopic third ventriculostomy
FND	Focal neurological deficit
VPS	Ventriculo-peritoneal shunt

Clinical Presentation

M16. Hx of fourth ventricular rosette-forming glioneuronal tumour debulked 7 years ago. Shunt dependent (initially had ETV which failed, multiple revisions of VPS, the last 3w ago). Re-presented with increasingly tense sub-occipital wound pseudomeningocoele and csf leak from shunt site, o/e GCS E3 V4 M6, disorientated and drowsy, apyrexial and not meningitic, no other FND

Differential Diagnosis

1. Shunt malfunction/Blocked shunt

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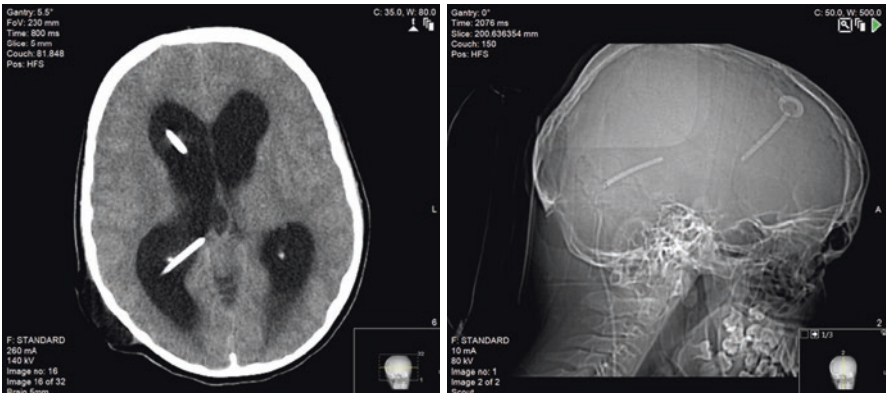
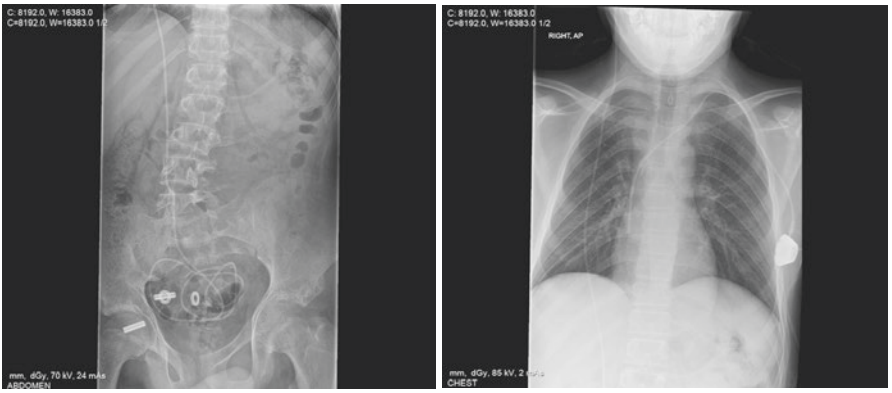
Investigations

CTH revealed enlarged ventricular system indicative of blocked shunt



Shunt series did not show obvious disconnection or malposition

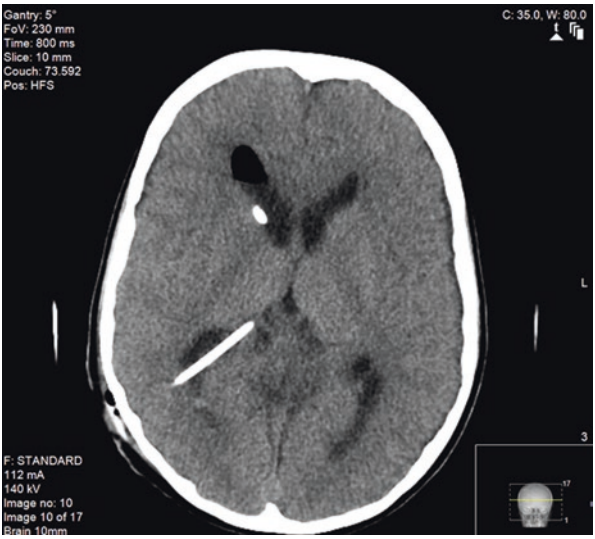


CSF not infected from tap of the collection

Management

Revision of VPS (ventricular catheter and low pressure valve)

Post op CTH revealed appropriate placement of ventricular catheter and decompressed ventricular system



Outcome

Improved clinically and pseudomeningocoele resolved

Part V

Tumor

Paediatric Posterior Fossa Tumor

29

Abbreviations

CSF	Cerebrospinal fluid
ETV	Endoscopic third ventriculostomy
EVD	External ventricular drain
h/a	Headache
Hx	History
Mets	Metastasis
MRI	Magnetic resonance imaging
N/V	Nausea and vomiting
PF	Posterior fossa
PROM	Premature rupture of membranes
PPI	Proton pump inhibitor
U/S	Ultrasound
WHO	World Health Organisation classification of brain tumours (2016)

Clinical Presentation

Nine years old boy, PROM at 28/40 and induced vaginal delivery at 35/40 +4, with background of complex partial seizures on medical treatment, presents with 2m hx worsening h/a, n/v, weight loss, poor co-ordination and imbalance, o/e nystagmus, dysarthria and ataxia with mild papilloedema

Clinical picture of cerebellar dysfunction and hydrocephalus

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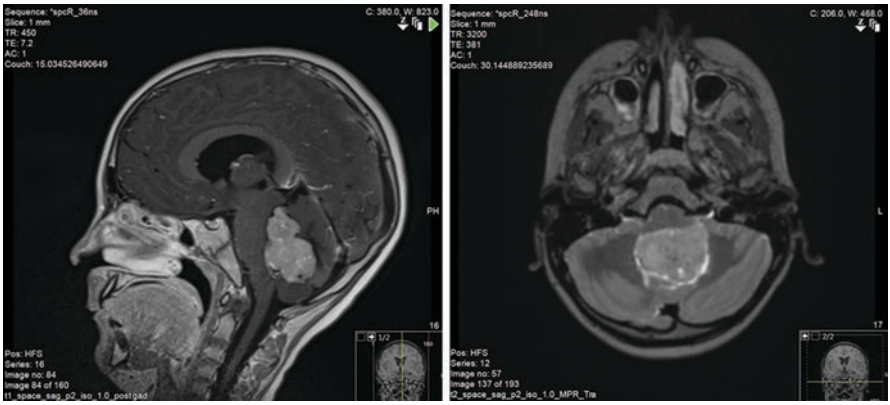
Differential Diagnosis

1. Cerebellar Tumour with secondary HCP (Pilocytic astrocytoma, ependymoma, medulloblastoma)
2. Brainstem tumours tend to present with lower cranial neuropathy, long tract findings and HCP

Investigations

Whole neuraxis MRI demonstrated fourth ventricular tumour with obstructive HCP without drop mets



Management

– dexamethasone with ppi cover
– ETV to relieve the HCP (in rare case in extremis from HCP for EVD to ensure external csf drainage)
– PF craniotomy with neuro monitoring and intra-operative U/S assistance for debulking of tumour

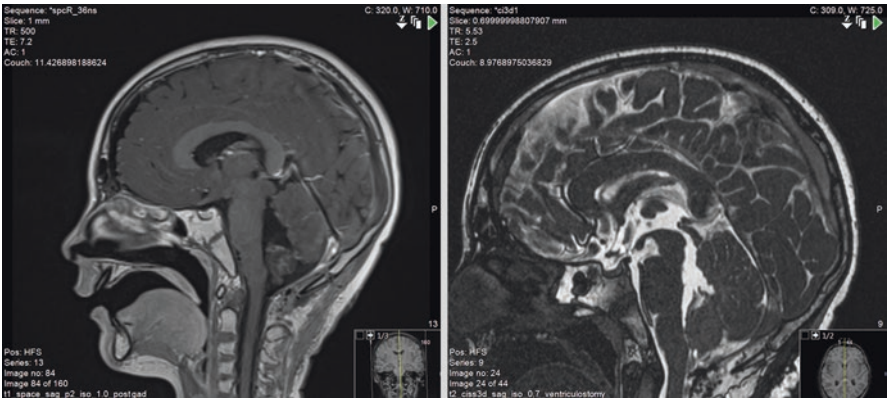
Outcome

Partial improvement of pre operative symptomatology

Complete resection of tumour achieved

Histology revealed Medulloblastoma with focal anaplasia, WHO grade IV

Non-WNT/non-SHH (group 3 or 4) subtype therefore adjuvant chemo radiotherapy instituted



Craniopharyngioma

30

Abbreviations

MDT	Multi-disciplinary team
MRI	Magnetic resonance imaging
T1WI	T1 weighted MRI imaging sequence
T1WI +c (Gad)	T1 weighted MRI imaging sequence with contrast (gadolinium)
T2WI	T2 weighted MRI imaging sequence

Clinical Presentation

12Y/M with short stature presented with increased frequency and worsening intensity of generalised, throbbing headaches on the background of occasional headaches since 1–2 years duration; visited the optician for eye routine eye screen which highlighted reduced visual acuity (6/60); associated with photophobia, blurred vision; no history of vomiting, seizures, polyuria or polydipsia; immunised as per the national schedule; no family history of brain tumours

On examination, GCS: 15/15, bitemporal hemianopia

Differential Diagnosis

Sellar/suprasellar lesion

1. Craniopharyngioma
2. Rathke cleft cyst
3. pituitary macroadenoma (with cystic degeneration or necrosis)

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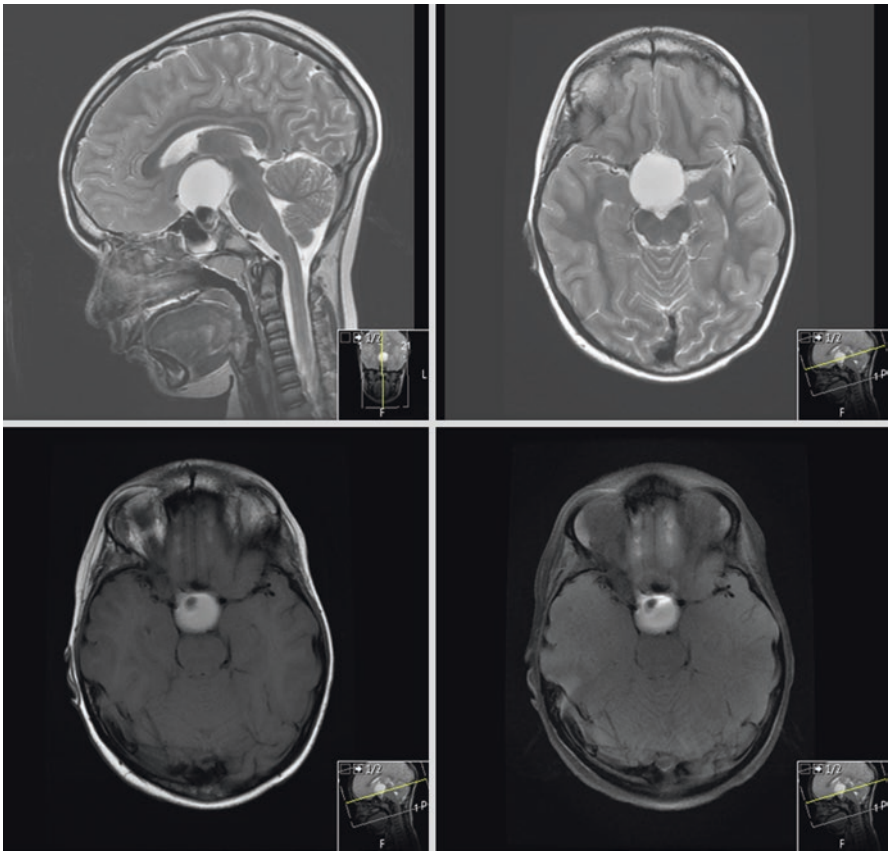
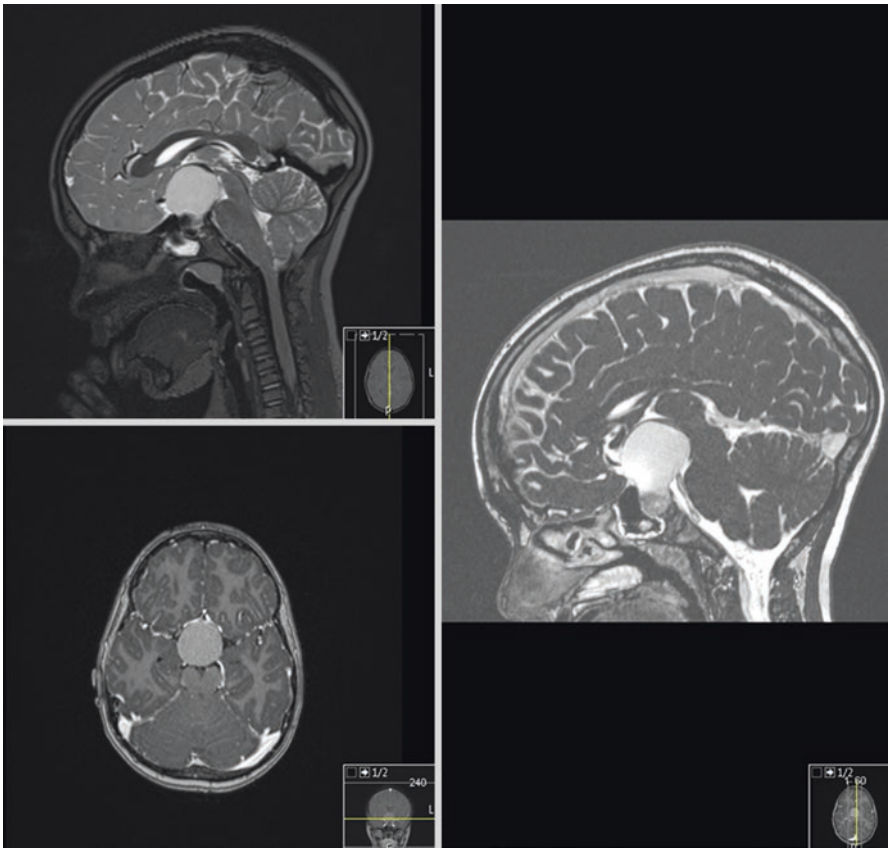
Investigations

MRI



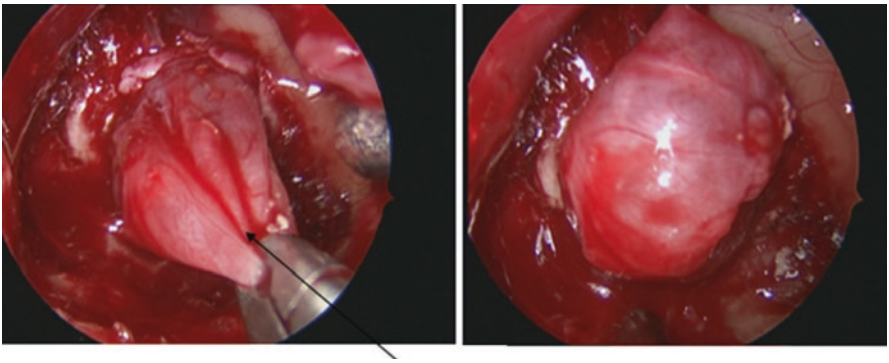


- cysts T1WI: iso- to hyperintense to brain (due to high protein content "machinery oil cysts")
  - T2WI: variable but ~80% are mostly or partly T2WI hyperintense
- solid component T1WI C+ (Gd): vivid enhancement
  - T2WI: variable or mixed

Management

1. Pituitary hormone profile
2. Endocrine advice
3. Insertion of lumbar drain and transsphenoidal resection of sellar/suprasellar lesion (image guided)

Histology: adamantinomatous craniopharyngioma, with no evidence of atypia



Outcome

Panhypopituitarism requiring supplementation, Pituitary MDT, regular follow up with endocrine, interval MRI scan at 3m

Follow up: 3m MRI, Follow up with Endocrine

Evidence

Garrè ML, Cama A. Craniopharyngioma: modern concepts in pathogenesis and treatment. Curr Opin Pediatr. 2007;19(4):471–9.

Reddy GD, Hansen D, Patel A, Lin Y, Jea A, Lam S. Treatment options for pediatric craniopharyngioma. Surg Neurol Int. 2016;7(Suppl 6):S174–8.

Pineal Tumour

31

Clinical Presentation

F12, previously healthy, presents with a 2w history of headache nausea and vomiting, Reduced appetite, Increased Thirst and Lethargy

On examination she was alert and orientated with ataxia and mild papilloedema

Differential Diagnosis

1. HCP secondary to PF tumour, pineal tumour, suprasellar tumour (craniopharyngioma, germinoma)
2. Supratentorial diencephalic tumour

Investigations

Whole neuraxis MRI revealed pineal region tumor with HCP, no spinal drop mets (spinal MRI should be performed prior to any intervention if possible to prevent confusion with blood products)

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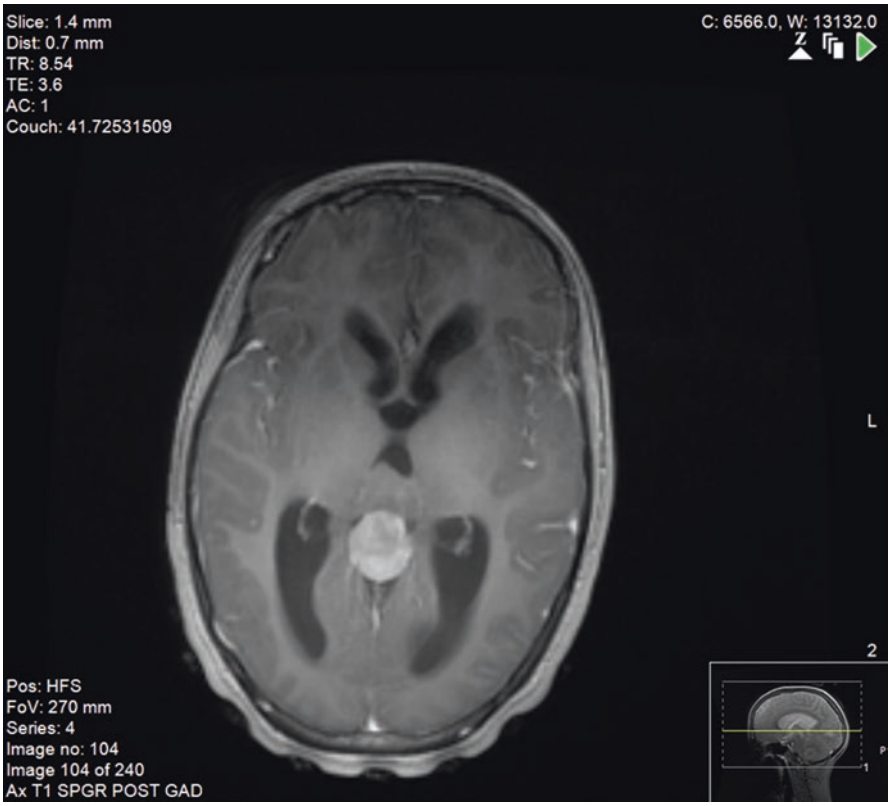


Serum and csf tumour markers (b-HCG, AFP, PLAP)

Pineal Region Tumours

- germ cell tumours(germinoma 40%, teratoma, choriocarcinoma, embryonal carcinoma, yolk sac tumour)
- pineocytoma and pineoblastoma
- glioma
- meningioma
- ependymoma
- metastasis
- pineal cyst
- (epi-)dermoid cyst
- cysticercosis
- VOM

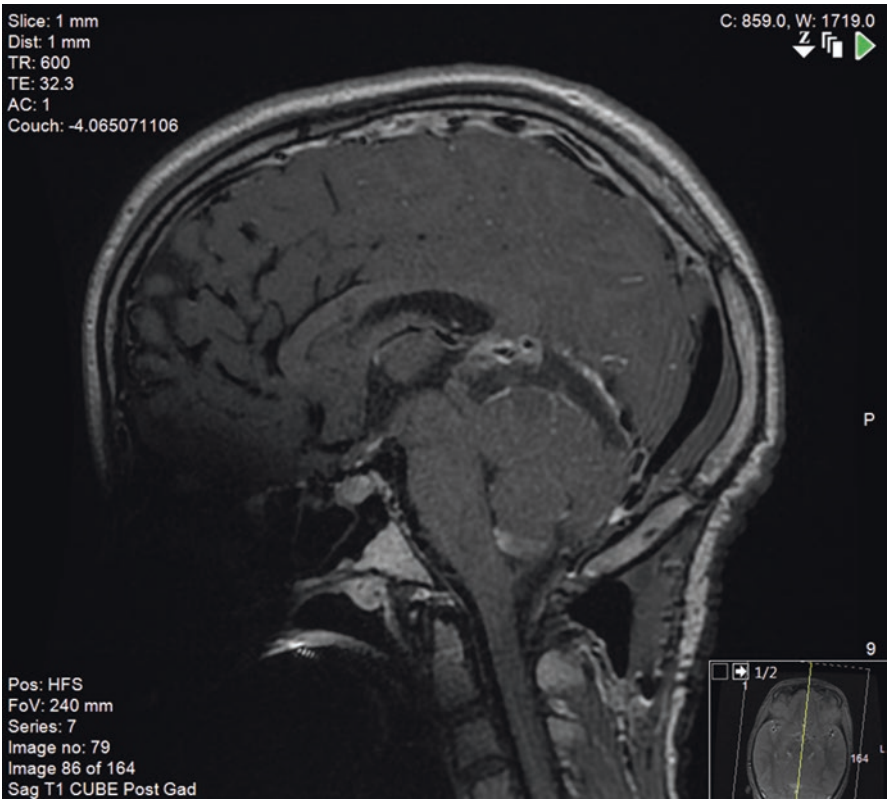
Germinoma highly sensitive to radiotherapy—usually require only csf diversion procedure

Synchronous germ cell tumours [suprasellar (mainly females) and pineal (mainly males)]

Management

- steroids
- endoscopic third ventriculostomy + insertion of rickham reservoir

posterior fossa craniotomy resection of tumour—pineoblastoma



Followed by craniospinal irradiation standard protocol
36 Gy in 20#, delivered over 4/52 using a 3D conformal technique
Followed by 18 Gy in 10# VMAT boost to site of pineal primary tumour, over 2/52

Outcome

Full recovery, under paediatric neuro oncology surveillance

Left Temporal High Grade Glioma

32

Abbreviations

co	Complaining of
CSDH	Chronic subdural haematoma
D	Days
GTR	Gross total resection
h/a	Headache
HGG	High grade glioma
MRI	Magnetic resonance imaging
PPI	Proton pump inhibitor
UMN	Upper motor neurone

Clinical Presentation

F60, RH, previously healthy and still very active, non smoker, IT school teacher, lives with her husband, 4w ago presented with right sided facial and limb weakness and focal seizure activity after 2D of behavioural change and mild cognitive decline. Currently no c/o h/a, intact neurologically except mild cognitive impairment and subtle Right UMN facial weakness, no speech impairment or pronator drift

Differential Diagnosis

1. Left Frontal/Temporal Tumour (Glioma, Metastasis, Sphenoid Wing Meningioma)
2. Left sided CSDH
3. Intracranial infection (abscess)

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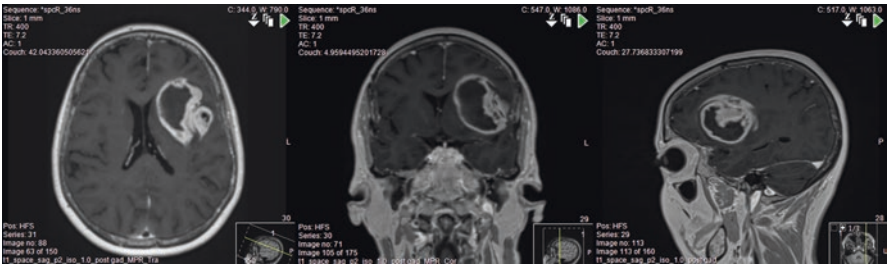
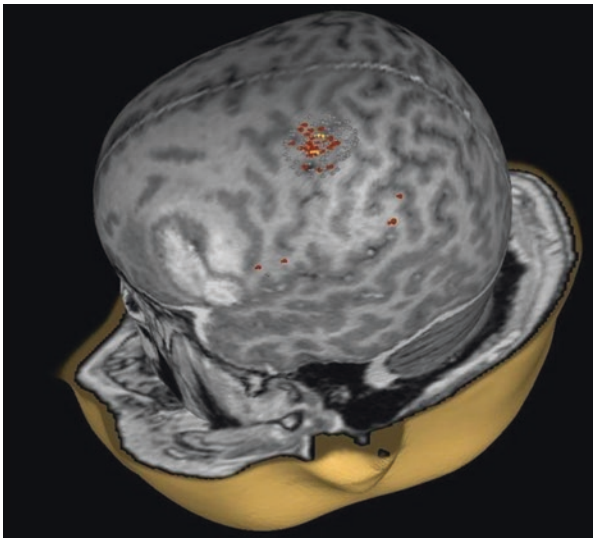
Investigations

MRI Brain revealed left insular HGG



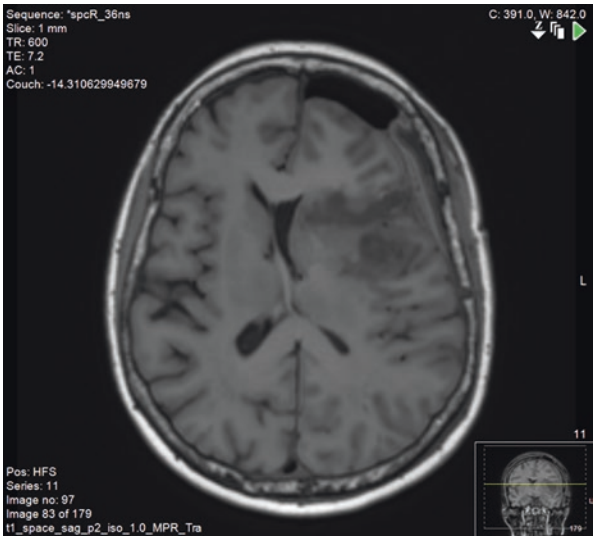
Management

Commenced on anti-epileptic and steroid therapy with ppi cover, underwent neuropsychology assessment and pre operative navigated transcranial magnetic stimulation for motor and speech mapping followed by awake image guided craniotomy with neuro monitoring and gliolan



Outcome

Recovered fully without neurological deficit and in specific without speech or motor impairment. Post op MRI confirmed GTR, histopathology revealed Glioblastoma, IDH 1 wild type (IHC)—(WHO grade IV), ATRX staining preserved (no mutation), Methylated MGMT promoter and completed adjuvant chemoradiotherapy.



Evidence

- Stupp protocol
- Molecular markers and prognosis

Olfactory Groove Meningioma

33

Abbreviations

ACA	Anterior cerebral artery
AqS	Aqueductal stenosis
D	Days
h/a	Headache
HCP	Hydrocephalus
Hx	History
MRI	Magnetic resonance imaging
NPH	Normal pressure hydrocephalus
PMH	Past medical history
SOL	Space occupying lesion

Clinical Presentation

M61, RH, lorry driver, lives with wife, ex smoker (stopped 5 years ago), no other significant PMH, presents with 1 year hx gradually worsening h/a, behavioural changes and vacant episodes (absent seizures) on a background of a 3 year anosmia, o/e mild cognitive impairment and short term memory deficit, anosmia, normal vision and fundoscopy

Frontal lobe syndrome

Differential Diagnosis

1. Frontal neoplastic SOL (extra axial—meningioma, intra axial—glioma)
2. NPH, longstanding HCP from AqS
3. Dementia

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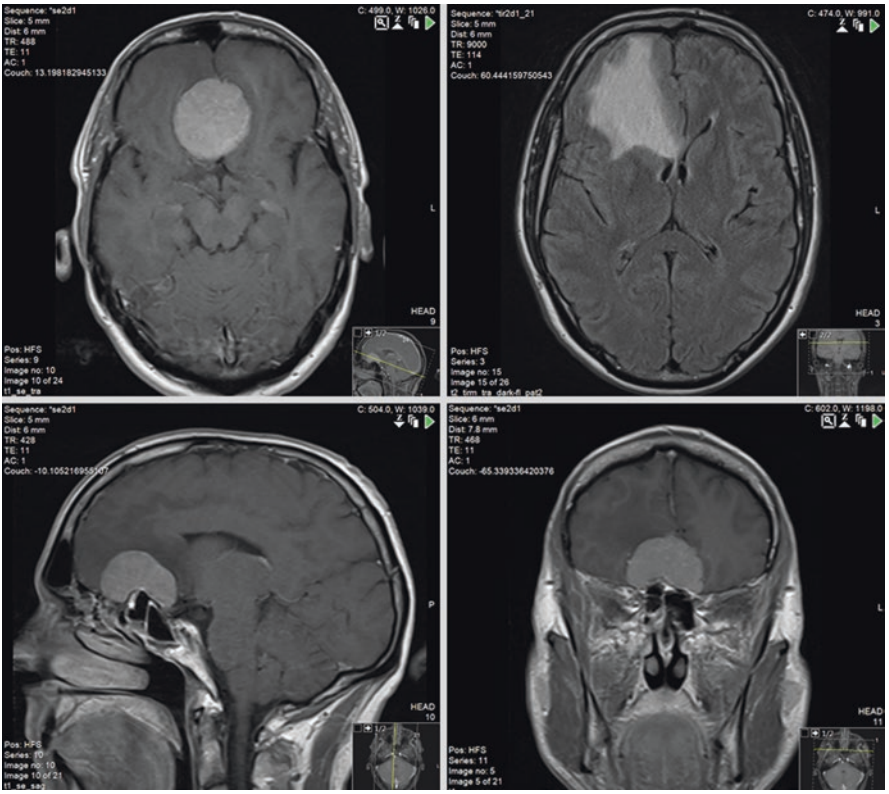
Investigations

MRI Brain revealed olfactory groove meningioma with associated peri-tumoral oedema.



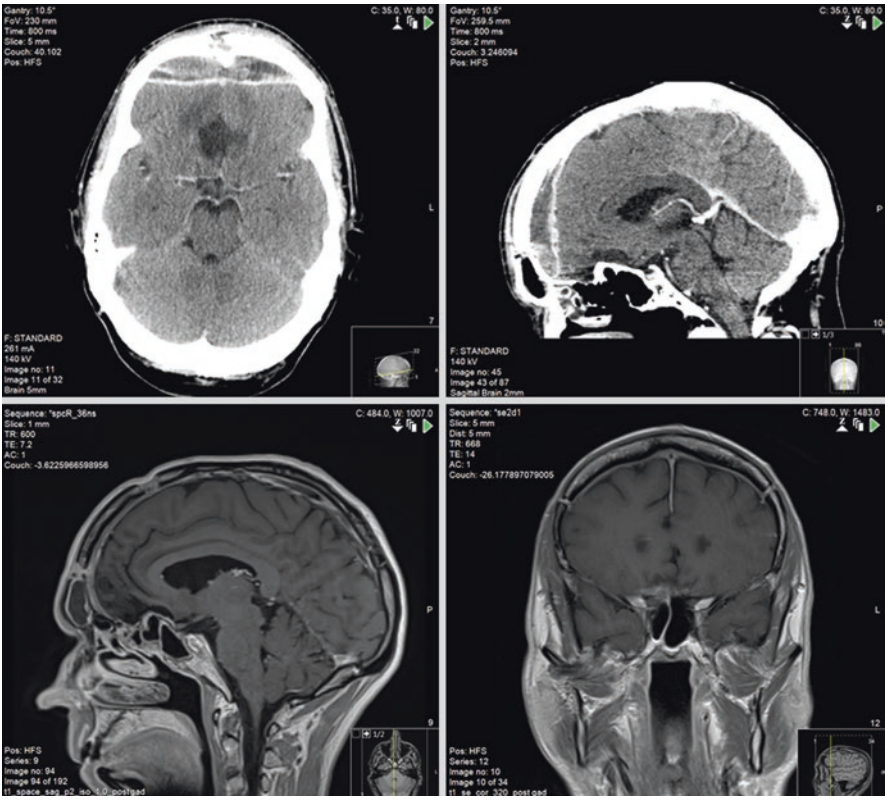
Management

Administration of steroids with ppi cover and anti-epileptics. A preoperative CTA is often performed to document the relation of the ACAs to the tumour.

Underwent electively Bifrontal craniotomy and resection of olfactory groove meningioma.

Outcome

Post op imaging confirmed complete resection.
Discharged home with residual anosmia and mild cognitive impairment D4 post op.



Posterior Fossa Metastasis

34

Abbreviations

CT CAP	Computed tomography chest abdomen and pelvis
CTH	Computed tomography head
HCP	Hydrocephalus
m	Months
MDT	Multi-disciplinary team
Mets	Metastasis
MRI	Magnetic resonance imaging
PF	Posterior fossa
RF EVD	Right frontal external ventricular drain
TNM	Tumour node metastasis cancer grading system
SCC	Squamous cell carcinoma

Clinical Presentation

M61, RH, lives with friend, ex smoker, no etoh, background of Hypopharynx SCC—T4A N2C M0, resected 1 year ago followed by adjuvant chemoradiotherapy, presents with 1m hx h/a, ataxia, left sided dysmetria and visual impairment secondary to papilloedema and deteriorates rapidly to GCS E2 V tracheostomy M4

Differential Diagnosis

1. HCP secondary to PF metastatic disease or carcinomatous meningitis
2. Multiple intracranial metastases
3. Intracranial infection due to immunosuppression
4 Intracranial haemorrhage due to treatment related coagulopathy

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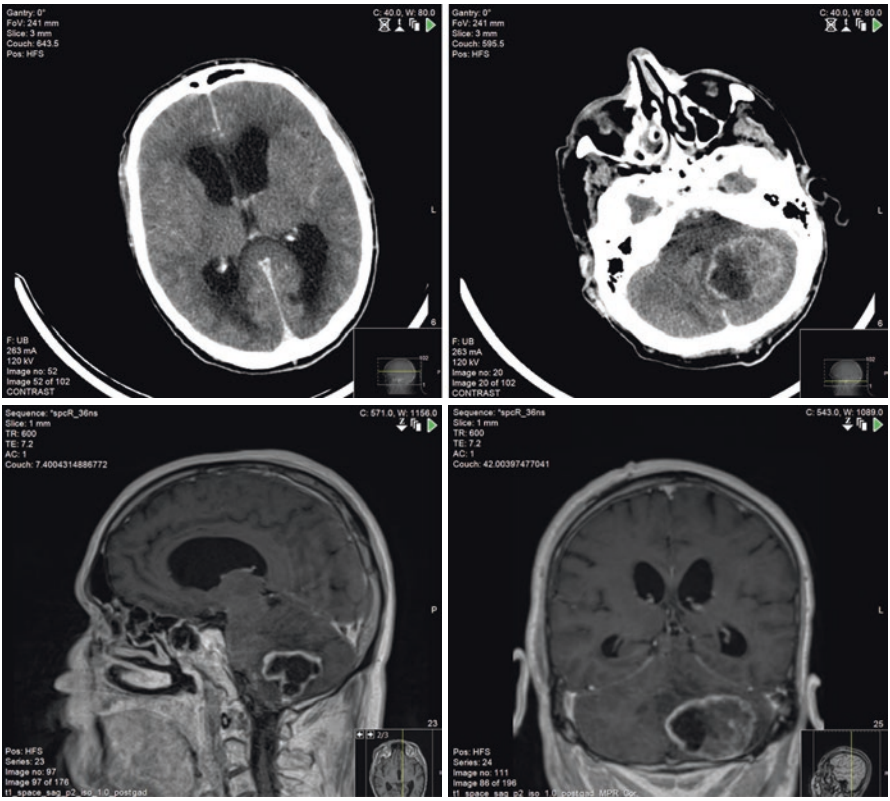
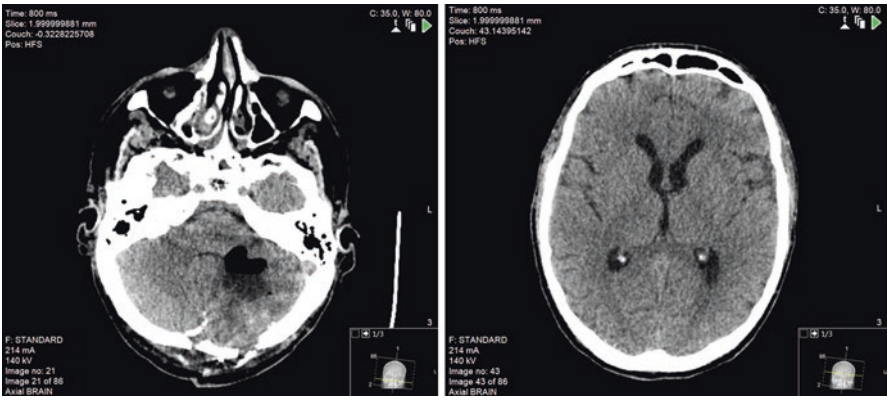
Investigations

Urgent CTH pre and post contrast revealed HCP secondary to left cerebellum metastasis, MRI confirmed solitary deposit, CT CAP excluded metastatic disease elsewhere



Management

Underwent urgent insertion of R F EVD to relieve the HCP and recovered to GCS E4 VT M6, commenced on high dose steroids with ppi cover which was weaned down gradually post op, since no disease elsewhere MDT decided to proceed with resection of cerebellar metastasis through posterior fossa craniotomy and subsequently EVD removed 72 h after.



Outcome

Recovered with mild residual left sided in coordination and balance impairment.

Evidence

Cerebral metastases are the most common brain tumour seen clinically
Cerebellar lesion in an adult is a metastasis until proven otherwise
15–30% of patients with cancer will develop cerebral metastasis
Primary sites; lung, breast, kidney, gastro-intestinal, melanoma, undetermined

Pituitary Apoplexy

35

Abbreviations

ACTH	Adrenocorticotropic hormone
CTA	Cerebral tomography angiogram
CTH	Computed tomography head
FSH	Follicle stimulating hormone
GH	Growth hormone
h/a	Headache
HCP	Hydrocephalus
IGF-1	Insulin like growth factor
MRI	Magnetic resonance imaging
Na	Sodium
N/V	Nausea and vomiting
PMH	Past medical history
PRL	Prolactin
SAH	Subarachnoid haemorrhage
T4	Thyroxine
TDS	Three times daily
TSH	Thyroid stimulating hormone
VF	Visual fields

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Clinical Presentation

M56, RH, fireman, non smoker, lives with wife and two children, no significant pmh, reports libido loss over the last 6m, presents with sudden severe h/a with associated neck pain and n/v and blurred vision, o/e drowsy but orientated and obeying commands with bitemporal hemianopia and visual acuity R 6/12 (corrected, glasses), L finger counting

Differential Diagnosis

1. Pituitary apoplexy (adenoma)
2. Craniopharyngioma with HCP
3. Aneurysmal SAH

Investigations

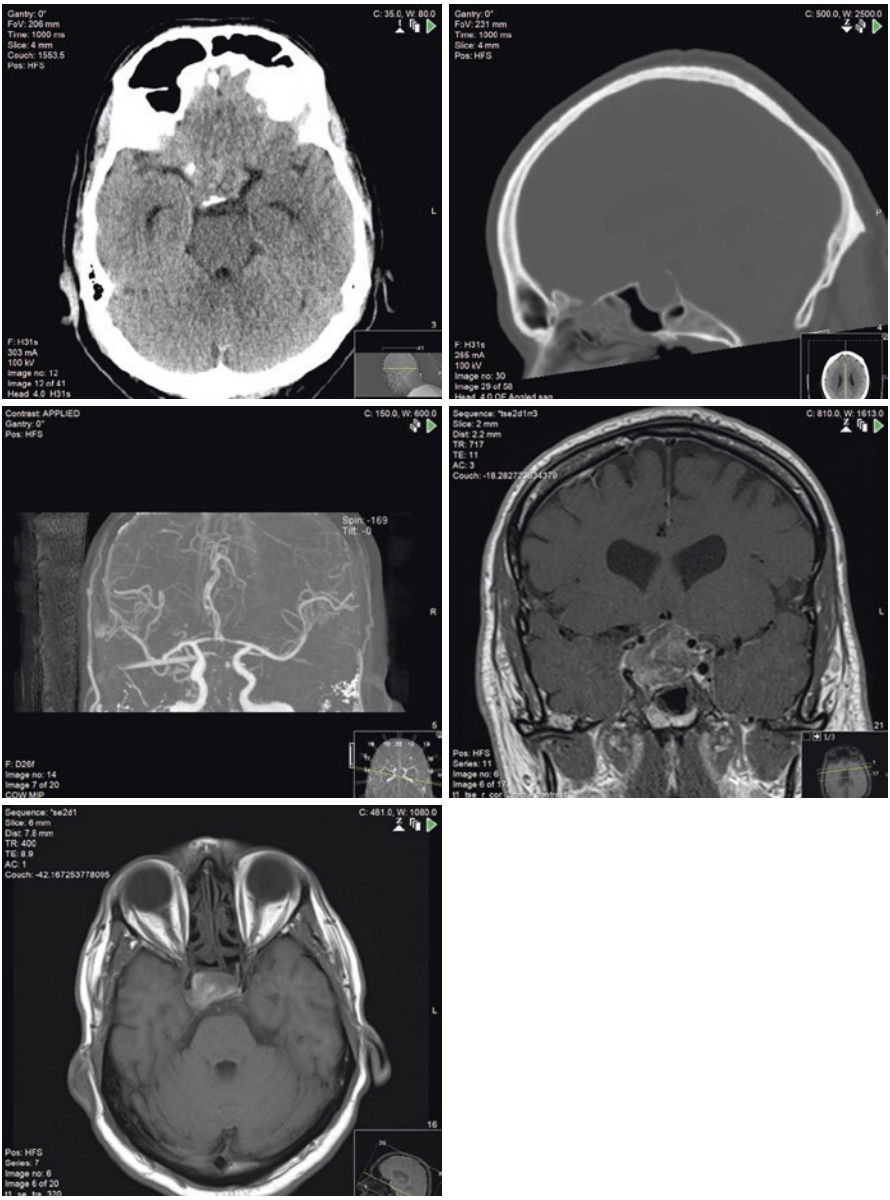
– Pituitary profile
– Formal VF
– CTH, CTA, Brain MRI

CTH revealed pituitary apoplexy secondary to suprasellar lesion, most likely adenoma

Note expansion and thinning of bony sella consistent with pituitary adenoma

CTA ruled out underlying vascular abnormality

MRI confirmed pituitary macroadenoma with haemorrhagic and necrotic component secondary to apoplexy and compression of the optic chiasm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Pos: HFS
Series: 7
Image no: 6
Image 6 of 20

Management

– After pituitary profile sent from peripheral blood commence on steroid replacement therapy with Hydrocortisone 100 mg tds
– Endoscopic Transnasal Transphenoidal Resection of Pituitary Adenoma

General

- pituitary adenomas usually present with VF deficit and hormone oversecretion (PRL-amenorrhea-galactorrhea-loss of libido, GH-acromegaly, ACTH-Cushing's, TSH-Hyperthyroidism) or Panhypopituitarism
- also can cause h/a from dural irritation secondary to diaphragma sella stretching
- Pituitary profile includes; 8 am cortisol, 24 h urine free cortisol, T4, TSH, PRL, FSH,LH, IGF-1, fasting blood glucose
- Prolactinoma can be managed with dopamine agonists (bromocriptine, cabergoline), Acromegaly with dopamine agonists, somatostatin analogue (octreotide), GH antagonist (pegvisomant), Cushing's with ketoconazole, metyrapone

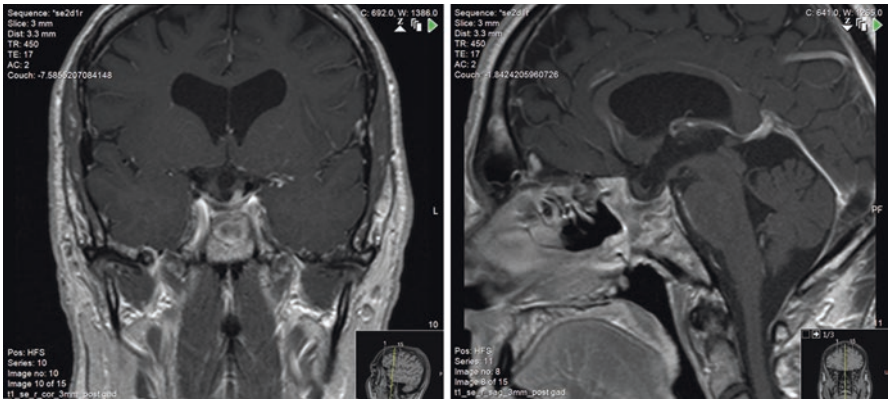
Outcome

Histology confirmed pituitary adenoma with LH and FSH positivity

Biochemistry revealed evidence of panhypopituitarism which require cortisone, testosterone, growth hormone and thyroxine replacement

Post op MRI confirmed total resection of tumour and decompression of optic chiasm

Vision improved back to pre morbid baseline



Evidence

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Albani A, Ferraù F, Angileri FF, et al. Multidisciplinary management of pituitary apoplexy. Int J Endocrinol. 2016;2016:7951536. https://doi.org/10.1155/2016/7951536.

Rajasekaran S, Vanderpump B, Drake W, Reddy N, Lanyon M, Markey A, Plant G, Powell M, Sinha S, Wass J. UK guidelines for the management of pituitary apoplexy Pituitary Apoplexy Guidelines Development Group: May 2010. Clin Endocrinol. 2011;74:9–20.

Turgut M, Ozsunar Y, Başak S, Güney E, Kir E, Meteoğlu I. Pituitary apoplexy: an overview of 186 cases published during the last century. Acta Neurochir (Wien). 2010;152(5):749–61.

Metastatic Malignant Spinal Cord Compression

36

Abbreviations

AVF	Arteriovenous fistula
Ca	Cancer
CT CAP	Computed tomography chest abdomen and pelvis
EDEM	Extradural extramedullary tumour
Hx	History
IDEM	Intradural extramedullary tumour
IDIM	Intradural intramedullary tumour
L2	Second lumbar vertebrae
LL	Lower limb
m	Months
MSCC	Metastatic spinal cord compression
MRC grading	Medical research council muscle grading
MRI	Magnetic resonance imaging
PPI	Proton pump inhibitor
PSA	Prostate specific antigen
T4	Fourth thoracic vertebrae
XRT	Radiotherapy

Clinical Presentation

F72 with background of breast Ca, mastectomy and adjuvant chemoradiotherapy 15 years ago, presents with 2m hx upper thoracic back pain worse at night and 3w hx progressive lower limb weakness and numbness, o/e sensory level at T4 with hypoaesthesia, bilateral spastic LL weakness MRC grade 4/5 with increased reflexes, bilateral clonus and reduced proprioception, no sphincteric disturbance

Upper thoracic myelopathy

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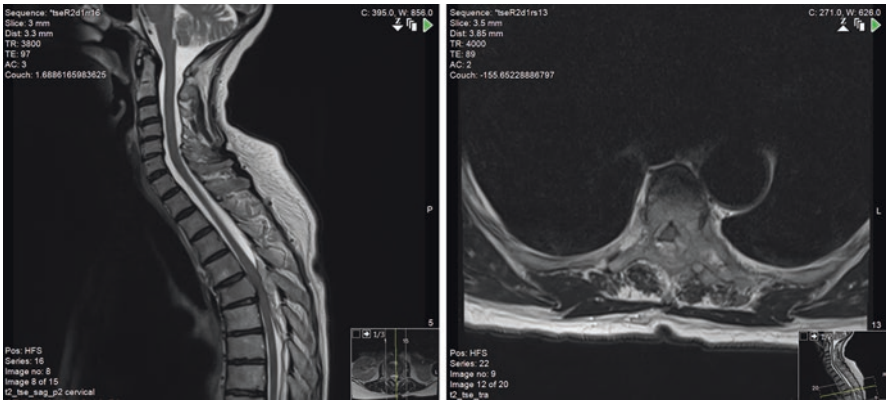
Differential Diagnosis

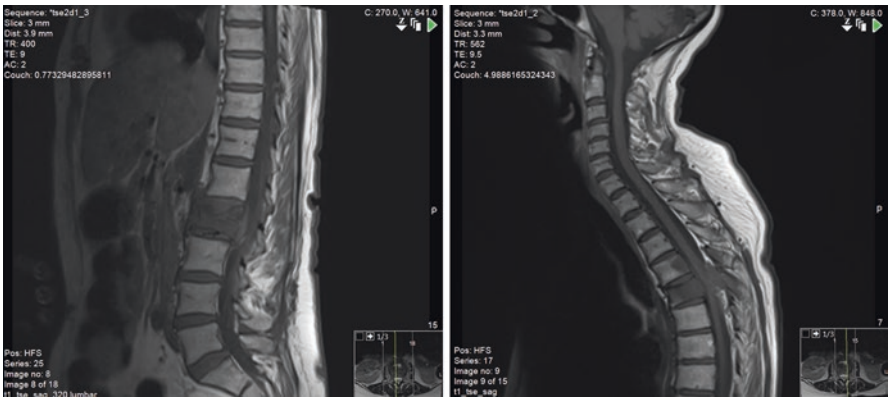
1. MSCC
2. Thoracic meningioma or ependymoma
3. Spinal dural AVF
4. Transverse myelitis

Investigations

MRI whole spine showed T5 MSCC with cord compression and infiltration of the pedicles, also non compressive L2 metastatic deposit



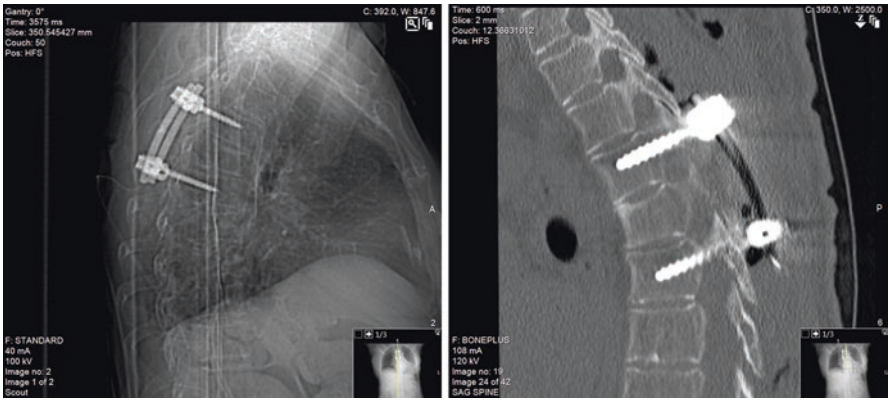
Note that bacterial infection usually involves the disc space and produces picture of vertebral osteomyelitis, neoplastic process usually spares the disc space and involves the pedicles with expansion and infiltration



CT CAP did not show metastatic disease elsewhere

Management

Dexamethasone with PPI cover
T5 laminectomy and T4–6 pedicle screw fixation



Outcome

Followed by XRT

Evidence

Spinal Tumours
- 55% extradural (metastatic, aneurysmal bone cyst, giant cell tumour, Ewing's sarcoma, chordoma, osteoma, osteoblastoma, haemangioma)
- 40% ID-EM (meningioma, nerve sheath tumours)
- 5% ID-IM (30% ependymoma, 30% astrocytoma, 30% miscellaneous)

MSCC
- osteolytic (lymphoma, lung, breast, prostate)
- osteoblastic (prostate, breast)

General Management
- patients with extensive metastatic disease and poor prognosis or total paralysis for >24 h for palliative oncological management
- patients with radiosensitive tumours (Multiple Myeloma, lymphoma) and no neurological compromise for XRT

– patients with new deficit, instability, unknown primary, favourable prognosis or radioresistant tumours (Renal Cell Carcinoma, melanoma) for surgical decompression/stabilisation
– metastatic work up (CT CAP, PSA, mammogram, serum and urine protein electrophoresis)

Thoracic Meningioma

37

Abbreviations

Ca	Cancer
EDEM	Extradural extramedullary tumour
IDEM	Intra-dural extramedullary tumour
IDIM	Intradural intramedullary tumour
IOM	Intra operative monitoring
MDI	Myelopathy disability index
mJOA	Modified Japanese orthopaedic association scale
MS	Multiple sclerosis
T2	Second thoracic vertebrae (same applies to T4, T1–3)

Clinical Presentation

39F, RH, mother of two, 6m history of altered sensation in legs and difficulty walking. O/E MRC grade 5/5 power legs, hyperreflexic, altered sensation to T4 level

Upper thoracic myelopathy

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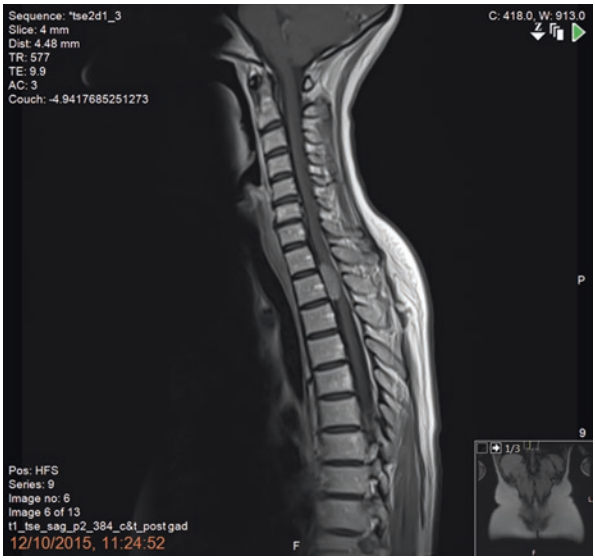
Differential Diagnosis

1. Thoracic spine neoplastic process (EDEM, IDEM, IDIM)
   a. Primary: nerve sheath tumour, meningioma, ependymoma, astrocytoma, sarcoma
   b. Secondary: lymphoma, multiple myeloma, breast Ca
2. Developmental (arachnoid cyst)
3. Degenerative (thoracic disc prolapse)
4. Inflammatory (MS, transverse myelitis)

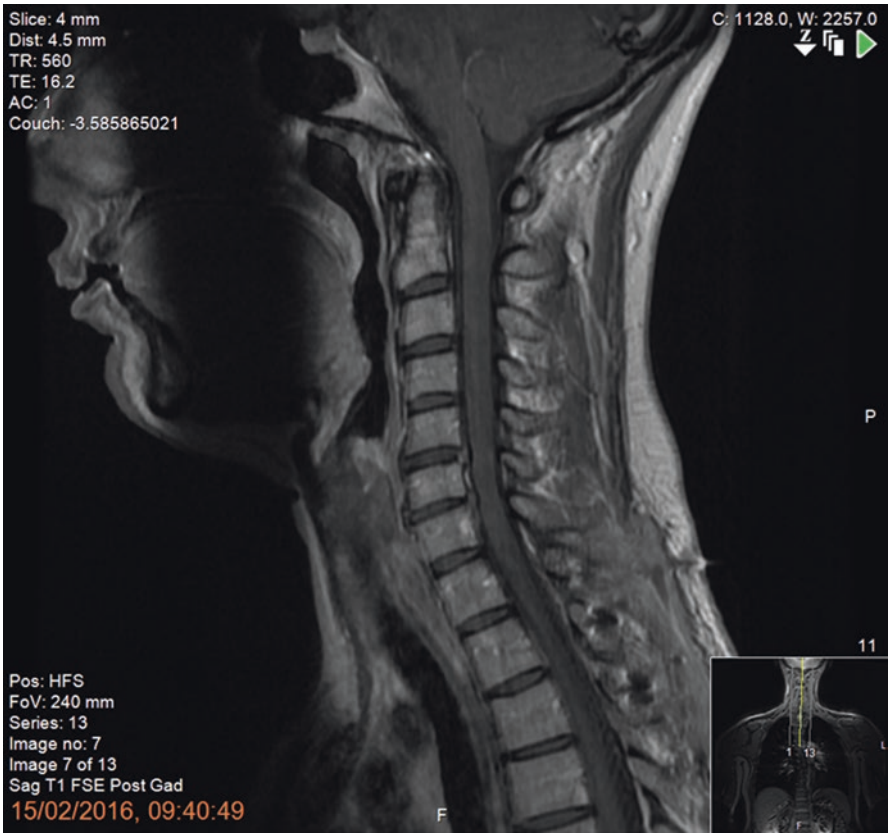
Investigations

MRI revealed T2 ventral durally based homogeneously enhancing lesion consistent with meningioma



Management

T1–3 laminotomy and excision of thoracic (T2) meningioma with IOM



Outcome

Excellent recovery although she mentions that she has some numbness around the axilla and some loss of position sense in her limbs. She is now walking independently and has improved significantly compared to pre-operatively.

Myelopathy Grading systems: MDI, mJOA

Evidence

MDI

Casey ATH, Bland JM, Crockard HA. Development of a functional scoring system for rheumatoid arthritis patients with cervical myelopathy. Ann Rheum Dis. 1996;55:901–6.

mJOA

Japanese Orthopaedic Association. Scoring system for cervical myelopathy. J Jpn Orthop Assoc. 1994;68:490–503.

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Spinal Cord Intradural Intramedullary Tumour

38

Abbreviations

Ca	Cancer
CT CAP	Computed tomography chest abdomen and pelvis
EDEM	Extradural extramedullary tumour
Hx	History
IDEM	Intra-dural extramedullary tumour
IDIM	Intradural intramedullary tumour
IOM	Intra operative monitoring
LL	Lower limb
m	Months
MEPs	Motor evoked potentials
MRC grading	Medical research council muscle grading
MS	Multiple sclerosis
SOL	Space occupying lesion
SSEPS	Somatosensory evoked potentials
T4	Fourth thoracic vertebrae
UL	Upper limb
WHO grade	World Health Organisation classification of brain tumours (2016)
XRT	Radiotherapy

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Clinical Presentation

F28, RH, smoker, social EtoH intake, no significant pmh, presents with 2m hx gradually worsening mid thoracic pain worse at night, unsteadiness, LL weakness and ascending loss of sensation, 24 h prior to admission developed insensate urinary retention requiring bladder catheterisation, o/e spastic LL weakness MRC 4/5, increased tone and bilateral LL clonus, sensory level at T4 will all modalities affected (pin prick, pain, temperature, proprioception) and no bladder sensation, normal UL and cranial examination

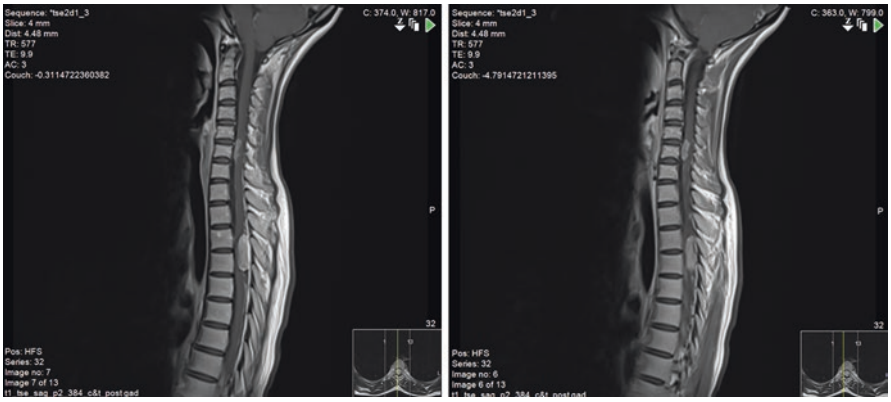
Upper thoracic myelopathy

Differential Diagnosis

1. Thoracic spine neoplastic process (EDEM, IDEM, IDIM)
   a. Primary: nerve sheath tumour, meningioma, ependymoma, astrocytoma, sarcoma
   b. Secondary: lymphoma, multiple myeloma, breast Ca
2. Developmental (arachnoid cyst)
3. Degenerative (thoracic disc prolapse)
4. Inflammatory (MS, transverse myelitis)

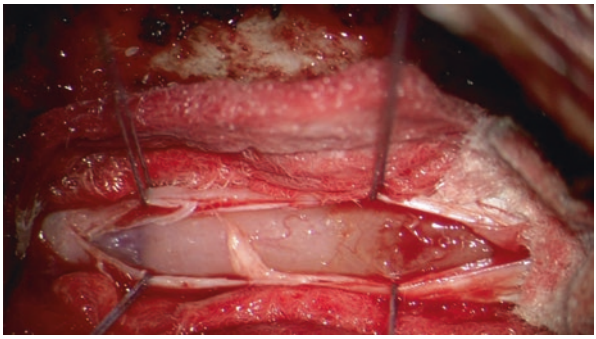
Investigations

– Whole Neuraxis MRI revealed dorsally located T4–5 IDIM SOL causing severe spinal cord compression, ventral C5–6 IDIM SOL, no obvious brain pathology and CT CAP showed small volume lymphadenopathy



Management

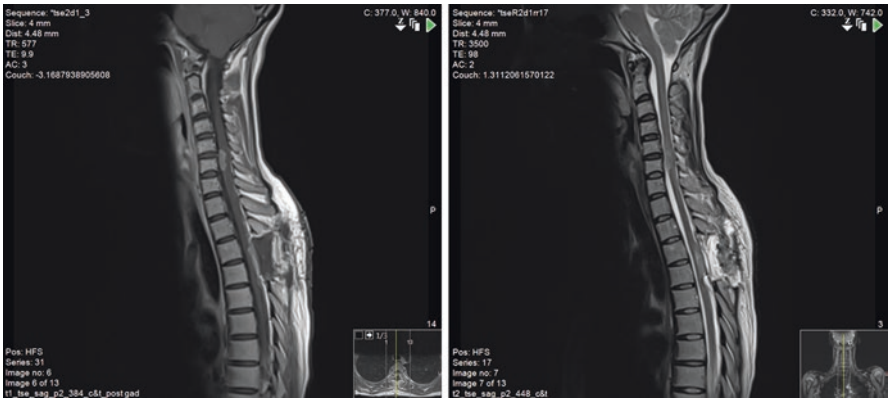
Commenced on steroids and underwent urgent T3 4 laminectomy and resection of intradural lesion with intraoperative neuro monitoring (MEPs, SSEPs)



Outcome

She was discharged eventually home with significant improvement of the pre existing sensori-motor deficit and mobilising independently but with residual bladder impairment requiring self catheterisations. Post op MRI confirmed adequate debulking and spinal cord decompression.

Histology revealed anaplastic Ependymoma (WHO grade III) and underwent cranio-spinal XRT.



Part VI

Infections

Intracerebral Abscess

39

Abbreviations

Abx	Antibiotics
CSF	Cerebrospinal fluid
CTH	Computed tomography head
ENT	Ear nose and throat specialists
EtoH	Alcohol
EVD	External ventricular drain
h/a	Headache
HCP	Hydrocephalus
IT	Intrathecal
IV	Intra venous
MRI	Magnetic resonance imaging
MRI ADC Map	Apparent diffusion coefficient magnetic resonance imaging sequence (used with DWI to verify if there is evidence of restricted diffusion i.e. free movement of hydrogen)
MRI DWI	Diffusion weighted magnetic resonance imaging (looks at restricted movement of hydrogen molecules and is useful in identifying infarct, intracerebral abscess and tumours with high cellularity)

Clinical Presentation

M22, RH, student, lives with parents, healthy, non smoker, no etoh, presents with 3m hx h/a and paranasal sinusitis on oral abx at the community, admitted urgently with meningism, confusion, headache and systemic sepsis without major focal neurological deficit

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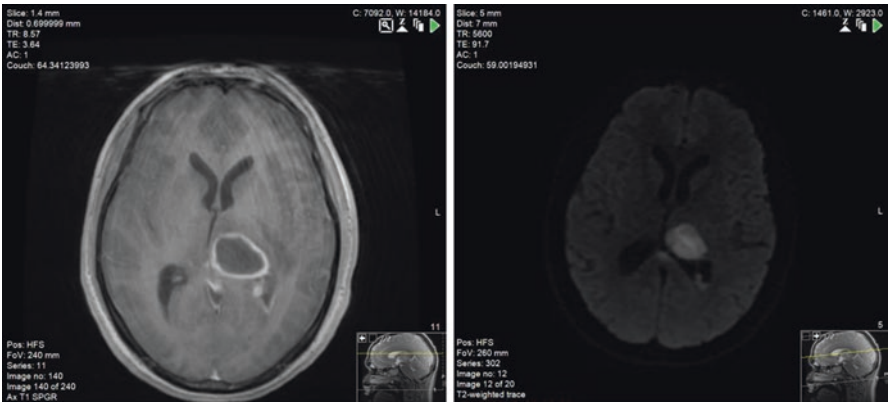
Differential Diagnosis

1. Intracranial infection (meningitis, abscess, empyema, ventriculitis, secondary HCP)

Investigations

CTH and MRI Brain demonstrates paranasal sinusitis, left thalamic abscess with intraventricular extension and HCP, ring enhancing lesion in the left thalamus with restricted diffusion on DWI (always correlate with ADC map-as a rule of thumb infection returns opposite signal from CSF on DWI/ADC)

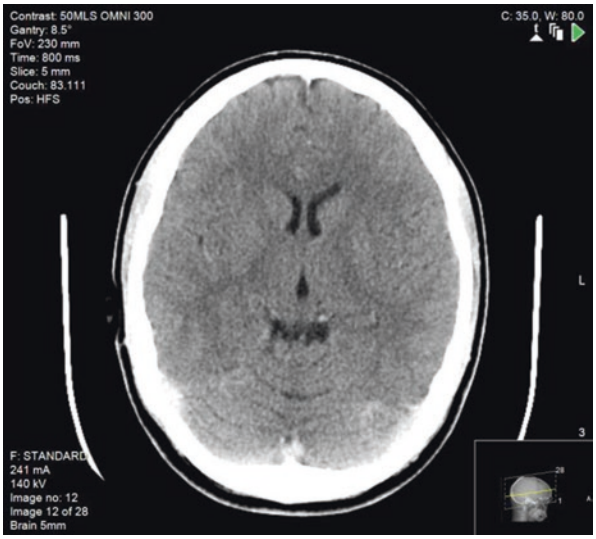


Management

Emergency image guided burr hole aspiration of left thalamic abscess and insertion of EVD followed by 6w iv abx therapy with Vancomycin and Metronidazole and 2w IT Vancomycin for streptococcus intermedius infection, also sinus wash out by ENT team, subsequently the EVD was challenged and removed.

Outcome

Recovered fully without residual symptomatology or neurological deficit



Subdural Empyema and Epidural Abscess

40

Abbreviations

Abx	Antibiotics
CTH	Computed tomography head
IV	Intra venous
MRI	Magnetic resonance imaging
MRI DWI	Diffusion weighted magnetic resonance imaging (looks at restricted movement of hydrogen molecules and is useful in identifying infarct, intracerebral abscess and tumours with high cellularity)

Clinical Presentation

M17, RH, student, non smoker, previously healthy, presents with 1w hx R periorbital cellulitis, commenced on oral abx at the community, admitted urgently at local hospital with pyrexia, confusion and drowsiness, deteriorated rapidly to GCS E2 V2 M4 with left sided hemiparesis and fixed and dilated R pupil, I+V urgently, hyperosmotic therapy with mannitol commenced and blue lighted to neurosurgical operating theatre.

Differential Diagnosis

1. Intracranial infection (extradural abscess, subdural empyema, intracerebral abscess)
2. Pott's puffy tumour

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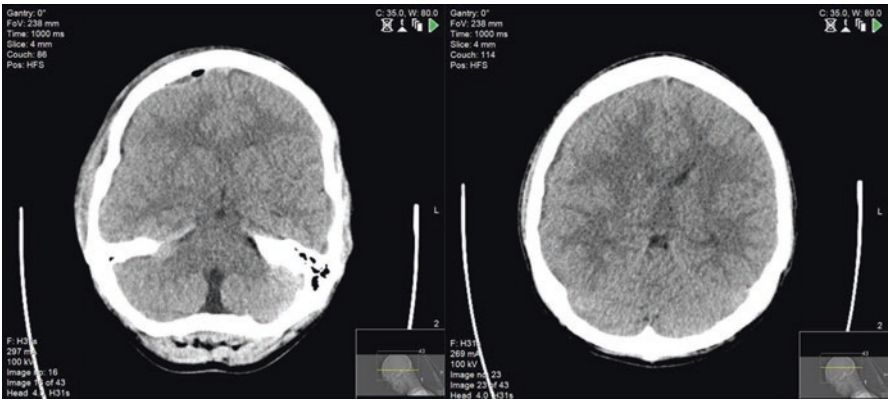
Investigations

CTH revealed right sided subdural empyema and extradural abscess causing mass effect secondary to frontal sinusitis and periorbital cellulitis

(note that imaging usually underestimates extent of subdural empyema)

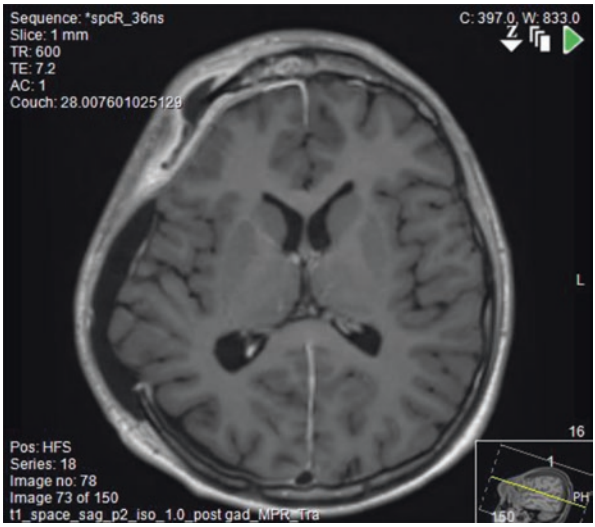


Management

Underwent urgent Right sided Fronto-Temporal Craniectomy for Drainage of Extradural Abscess and Subdural Empyema plus Fiber optic Endoscopic Sinus Surgery and washout of Frontal Sinus followed by 6w administration of iv abx therapy ceftriaxone and vancomycin (most common pathogen is streptococcus milleri, in this occasion fusobacterium necrophorum was isolated), also commenced on prophylactic anti-epileptic therapy since intracranial infection imposes high risk of epilepsy 40–80%.

Outcome

Patient recovered gradually back to normal without major residual deficit



Ventriculitis and Post Operative Spinal Infection

41

Clinical Presentation

M83, RH, previously healthy and independent, lives with wife, non smoker, no etoh, underwent elective lumbar laminectomy for lumbar canal stenosis expressed with neurogenic claudication, complicated by intraoperative dural tear and csf leak which was primarily repaired, re presented with persistent lumbar wound csf leak followed by pyrexia, confusion and double incontinence, rapidly deteriorated to GCS E2 V1 M3 and systemic sepsis

Differential Diagnosis

1. CNS Infection (extension of spinal infection in intracranial space in the form of meningitis, empyema, abscess, ventriculitis)
2. Subdural hygromas or haematomas secondary to spinal csf leak plus systemic infection

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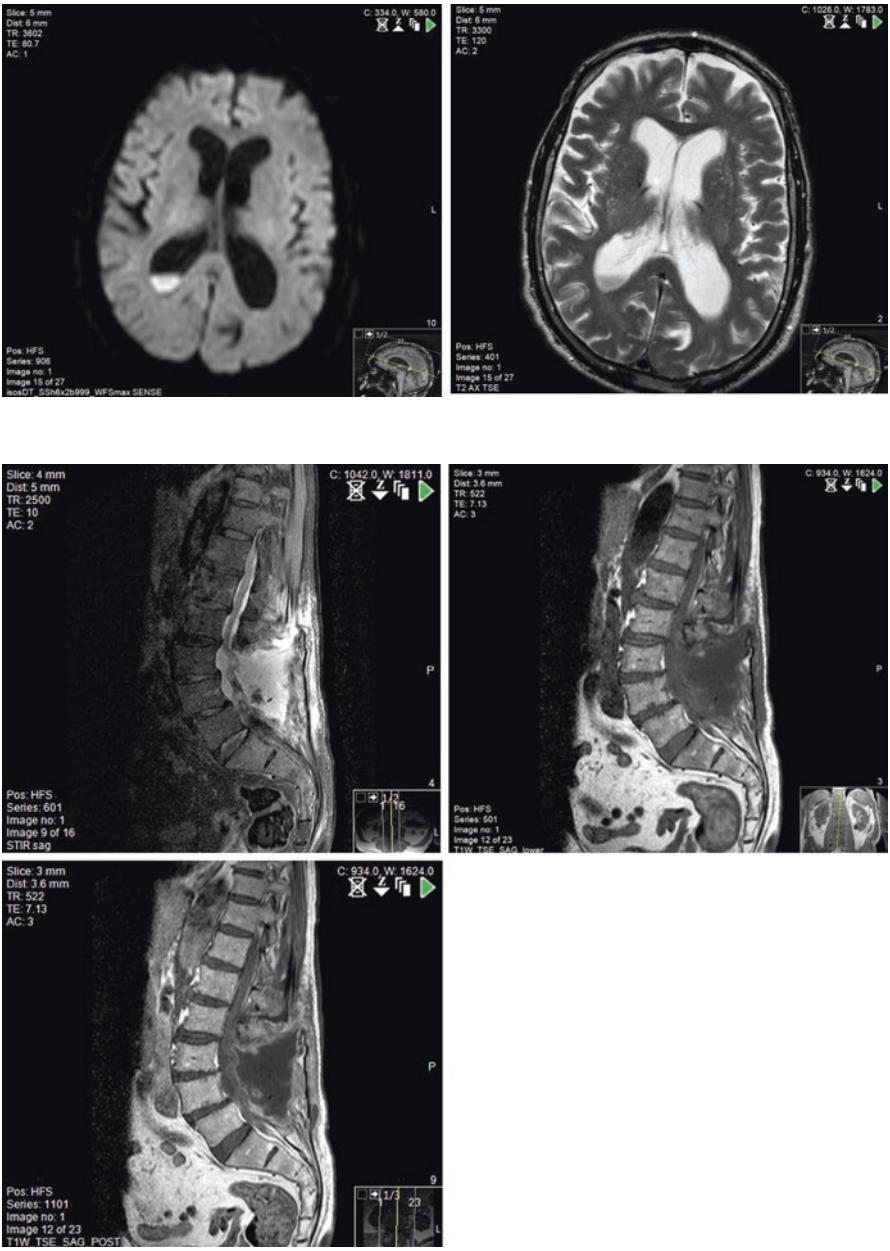
Investigations

CT Head and whole neuraxis MRI demonstrated infective lumbar collection and HCP with ventriculitis, area of restricted diffusion in right occipital horn on DWI and fluid level on T2WI



Management

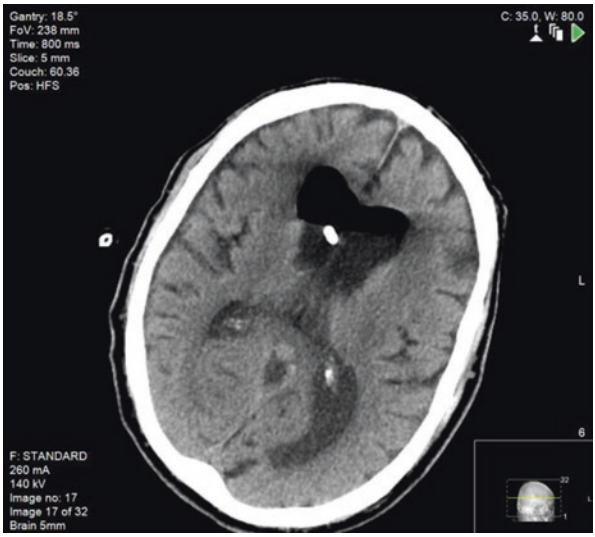
Patient underwent emergency i/o RF EVD for relief of HCP and IT access for abx, commenced on iv abx therapy with meropenem and vancomycin for 6w, IT gentamycin for 2w, prophylactic anti-epileptic therapy, lumbar wound wash out and repair of dural tear. E coli infection.

Outcome

Recovered partially with significant cognitive and mobility impairment.

Ventriculitis especially in elderly population carries significant mortality and morbidity.

Post op CTH revealed expected intraventricular air, appropriate placement of EVD and decreased ventricular size



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Thoracic Epidural Abscess

42

Abbreviations

Abx	Antibiotics
CRP	C reactive protein
CT CAP	Computed tomography chest abdomen and pelvis
ECHO	Echocardiogram
Gram +	Gram positive bacteria
Hx	History
IV	Intra venous
LL	Lower limb
MSCC	Metastatic spinal cord compression
MRC grading	Medical research council muscle grading
MRI	Magnetic resonance imaging
OPG	Orthopantomogram
PICC line	Peripherally inserted central catheter
PT	Physiotherapy
QDS	Four times daily
T3–6	Third to sixth thoracic vertebrae
TB	Tuberculosis
w	Weeks
WBC	White blood cell count

Clinical Presentation

M79, RH, only PMH of HTN and high cholesterol, active and independent, lives with wife, presents with 2w hx worsening back pain, malaise, loss of appetite and weight, night sweats and low grade pyrexia, o/e mid thoracic myelopathy expressed with spastic weakness of LL MRC grading 4/5 and T6 sensory level

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Differential Diagnosis

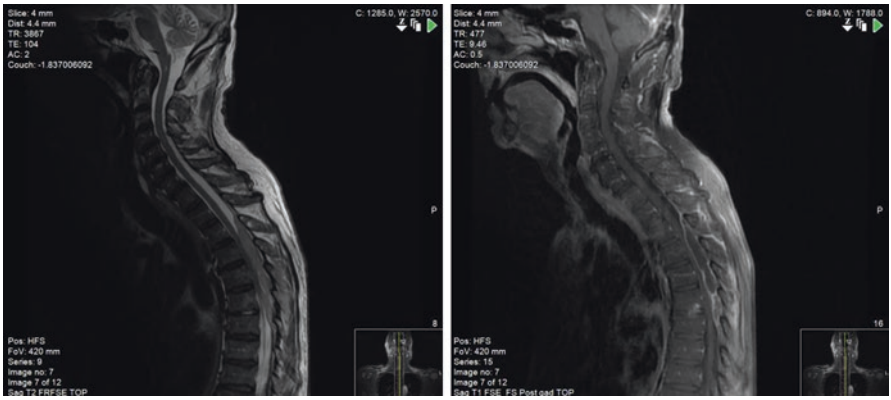
1 Thoracic spine bacterial infection or TB
2. Thoracic spine MSCC
3. Inflammatory/demyelination process

Investigations

MRI spine revealed T3–6 epidural enhancing collection consistent with abscess causing significant spinal cord compression.

Full septic screen (blood, sputum, urine cultures, CT CAP, Cardiac ECHO, OPG, skin evaluation)

WCC 23.84, CRP 224.3



Management

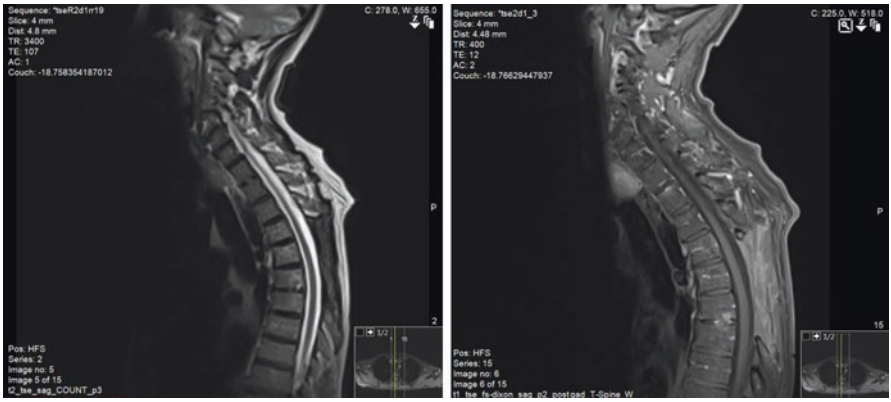
Underwent emergency T3–6 laminectomies and drainage of epidural abscess

Post op imaging with MRI confirmed adequate abscess evacuation and spinal cord decompression

Blood cultures and pus sample revealed gram + cocci, staph aureus, sensitive to flucloxacillin which he received at high dose (2 g QDS) for 6w through PICC line.

Outcome

He had a satisfactory post-operative recovery with significant improvement of the myelopathy and regaining LL power MRC grade 4+/5 and he followed routine PT/OT assessments. Eventually he was transferred to a neuro rehabilitation unit and at 3m review he was neurologically intact with mild residual back pain.



Post Operative Cranial Wound Infection

43

Abbreviations

Abx	Antibiotics
Ca	Cancer
CRP	C reactive protein
CSF	Cerebrospinal fluid
CTH	Computed tomography head
h/a	Headache
IV	Intra venous
Mets	Metastasis
MRI	Magnetic resonance imaging
Na	Sodium
PICC line	Peripherally inserted central catheter
r/o	Rule out
TIIDM	Type 2 diabetes mellitus
WBC	White blood cell count
XRT	Radiotherapy

Clinical Presentation

F56, RH, heavy smoker, TIIDM on metformin, undergoes uneventful Right occipital craniotomy for breast Ca metastasis expressed with h/a and left sided hemianopia, 7 years earlier Right breast Ca 2010: mastectomy + node clearance + chemoradiotherapy (XRT for 6w then 18m herceptin), presents 6w post op with pus discharge from cranial wound, worsening h/a and low grade pyrexia, WBC 9.75, CRP 8.9, Na 132

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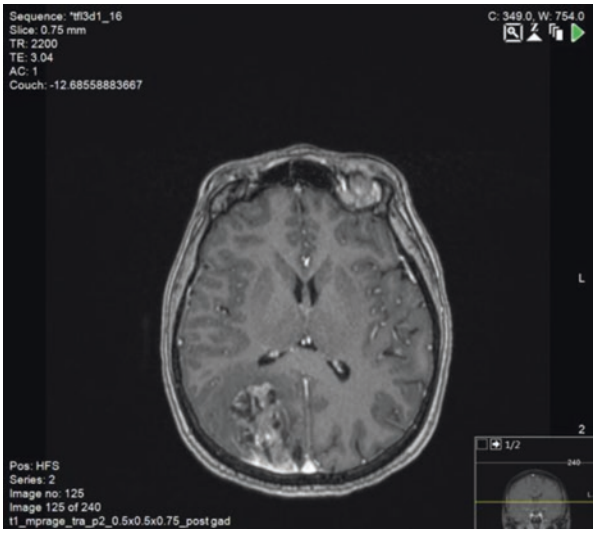
Differential Diagnosis

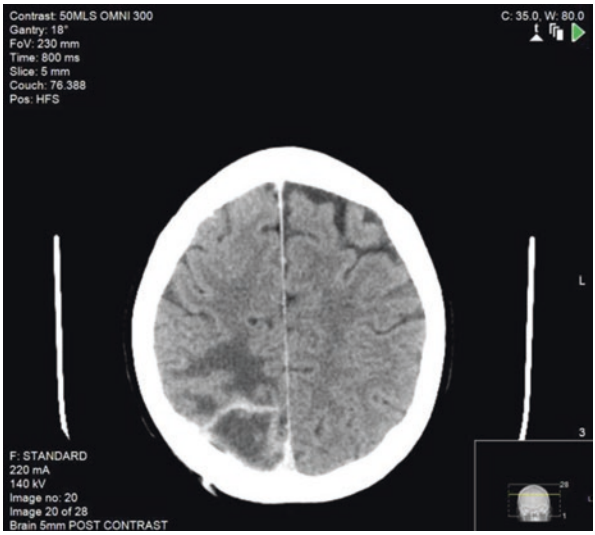
1. post op cranial wound infection
2. if you see post op wound csf leak you need to r/o hydrocephalus and infection

Investigations

MRI demonstrating R occipital breast Ca metastasis



CTH pre and post contrast revealed enhancing collection in tumour bed consistent with infection, no signs of bone flap osteomyelitis on bone window which is usually a late sign



Management

Underwent removal of infected bone flap, drainage of abscess and wound wash out, microbiology confirmed mixed growth of Haemophilus Parainfluenzae and Enterobacter Cloacae and received 6w iv abx therapy through PICC line with Meropenem and Vancomycin.

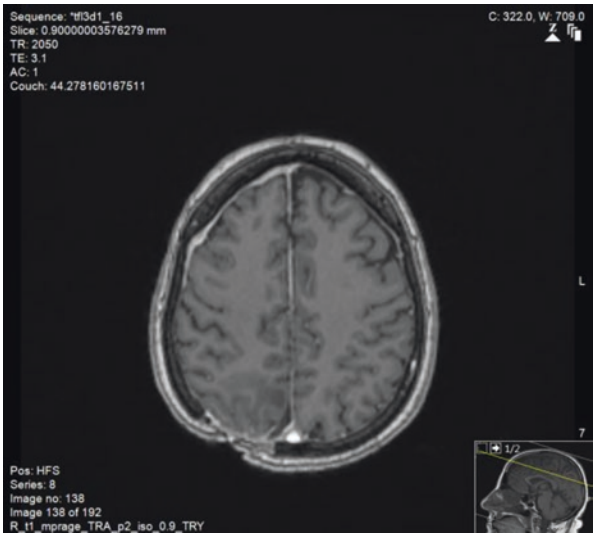
If only superficial wound infection can be managed with abx

Definitive treatment is removal of bone flap

Usual pathogens are staph epidermidis and staph aureus

Outcome

She recovered fully back to her normal baseline and awaiting titanium cranioplasty.



Part VII

Degenerative

Cervical Canal Stenosis

44

Abbreviations

C3–6	Third to sixth cervical vertebral level
HTN	Hypertension
MRC grading	Medical research council muscle grading
PMH	Past medical history
w	Weeks

Clinical Presentation

F78, RH, retired homecare assistant, independent with PMH of HTN presents with 5w hx neck pain and rapidly worsening mobility and dexterity, o/e bilaterally positive Hoffman's, mild spastic weakness of all four limbs MRC grade 4/5, bilaterally positive Babinski, diffusely reduced pin prick sensation of upper limbs

Rapidly progressing cervical myelopathy

Differential Diagnosis

1. Degenerative cervical canal stenosis
2. Cervical spine tumor primary or secondary
3. Infection
4. Inflammation/Demyelination

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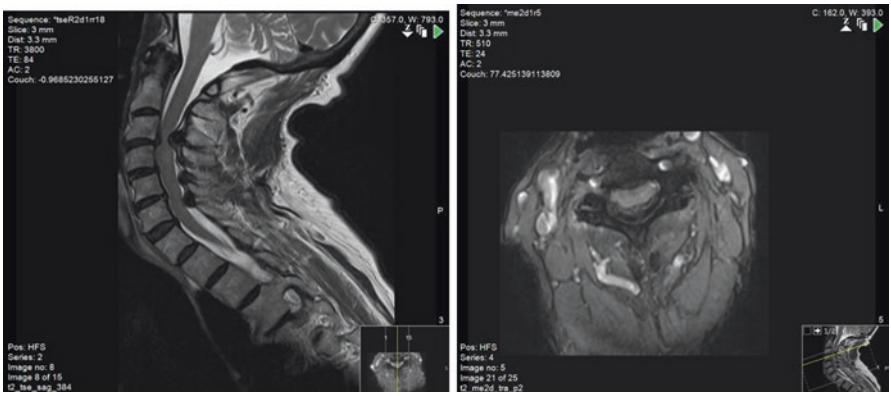
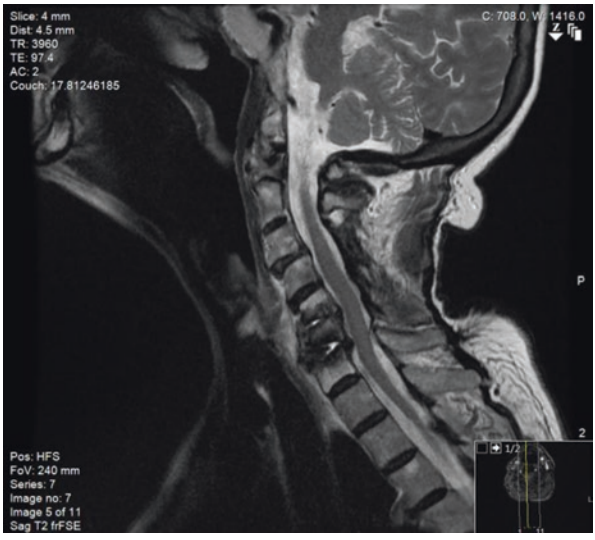
Investigations

MRI C spine revealed multilevel cervical canal stenosis C3–6 with cord compression worse at C3–4



Management

Underwent uneventful C3–6 laminectomies followed by neuro rehabilitation physiotherapy programme. Post operative imaging confirmed adequate neuronal decompression and revealed disc signal changes at the C5–6 and C6–7 levels.



Outcome

The decompressive surgery prevented further neurological deterioration and provided partial improvement of the cervical myelopathy.

Evidence

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Cauda Equina Syndrome

45

Abbreviations

ADF	Ankle dorsiflexion
AVM	Arteriovenous malformation
CES	Cauda equina syndrome
EHL	Extensor hallucis longus
HE	Hip extension
HF	Hip flexion
HLD	Herniated lumbar disc
KE	Knee extension
KF	Knee flexion
L3	Third lumbar vertebrae
LBP	Lower back pain
LL	Lower limb
LLL	Left lower limb
MRC grading	Medical research council muscle grading
MRI	Magnetic resonance imaging
S1	First sacral vertebrae
SLR	Straight leg raise
TB	Tuberculosis

Clinical Presentation

33M Hx chronic LBP presents with 6w of R sciatica, worse over last 2w, right LL weakness, erectile dysfunction, three episodes of urinary incontinence and pain radiating to LLL as well with bilateral paresthesias and dysesthesias and reduced perineal sensation.

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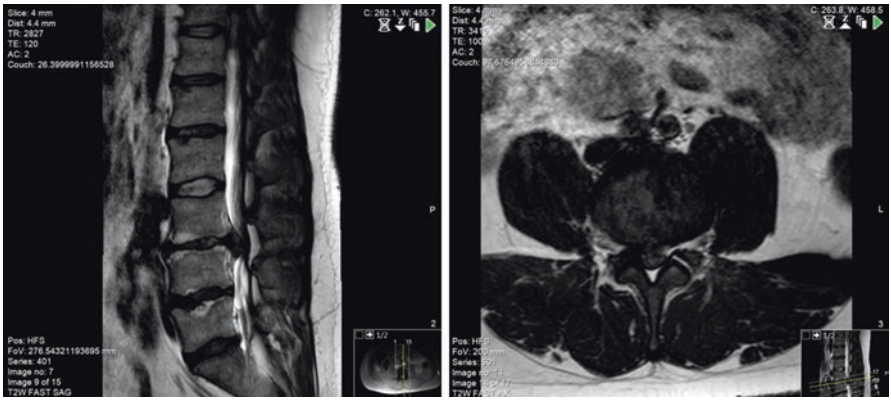
o/e SLR 10° bilaterally, Right lower limb power (MRC grade) EHL and ADF 2/5, KF/KE 3/5, HF/HE 4/5, reduced sensation from L3 dermatomes to S1. Absent knee and ankle reflex. Left lower limb 5/5, no sensory loss, absent ankle reflex. Reduced perineal sensation on the right.

Differential Diagnosis

1. Degenerative (HLD)
2. Neoplastic (nerve sheath tumour or metastatic)
3. Vascular (spinal AVM)
4. Inflammatory
5. Infectious (TB)

Investigations

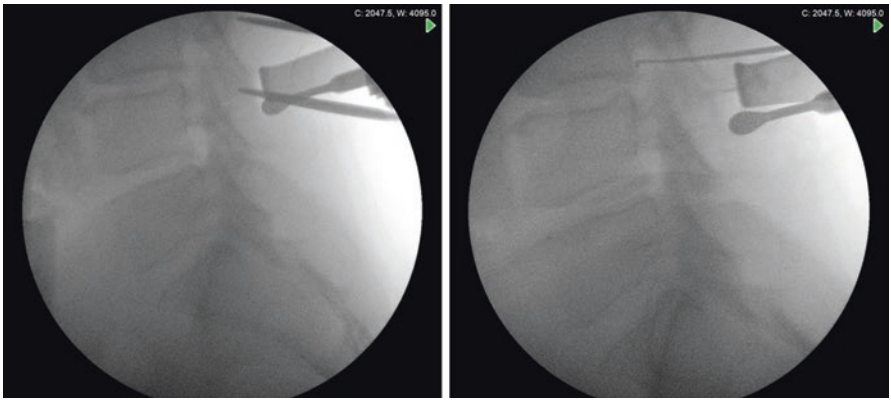
MRI L-S spine revealed large L3–4 disc prolapse causing cauda equina compression

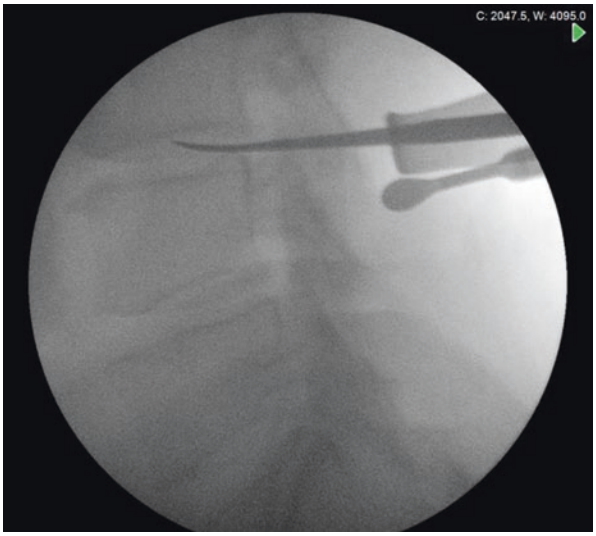


Management

Urgent L3–4 discectomy

Intra op level localisation with xray





Outcome

Significant post op improvement, regained R LL power and perineal sensation, resolved sciatica

Evidence

Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP. Cauda equina syndrome secondary to lumbar disc herniation—a meta-analysis of surgical outcomes. Spine. 2000;25(12):1515–22.

Coscia M, Leipzig T, Cooper D. Acute cauda equina syndrome: diagnostic advantage of MRI. Spine. 1994;19:475–8.

Crocker M, Fraser G, Boyd E, Wilson A, Chitnavis BP, Thomas NW. The value of interhospital transfer and emergency MRI for suspected cauda equina syndrome: a 2-year retrospective study. Ann R Coll Surg Engl. 2008;90:513–6.

DeLong WB, Polissar N, Neradilek B. Timing of surgery in cauda equina syndrome with urinary retention: meta-analysis of observational studies. J Neurosurg Spine. 2008;8:305–20.

Gardner A, Gardner E, Morley T. Cauda equina syndrome: a review of the current clinical and medico-legal position. Eur Spine J. 2011;20(5):690–7.

Hussain SA, Gullan RW, Chitnavis BP. Cauda equina syndrome: outcome and implications for management. Br J Neurosurg. 2003;17(2):164–7.

Kostuik JP. Medico-legal consequences of cauda equina syndrome: an overview. J Neurosurg Neurosurg Focus. 2004;16:39–41.

SBNSguidelines. http://www.spinedragon.com/docs/professional_guidelines/guidelines_ces_sbns_standardsofcarecaudaequinapolicyv4.pdf

Foot Drop

46

Abbreviations

ADF	Ankle dorsiflexion
BMI	Body mass index
EHL	Extensor hallucis longus
HLD	Herniated lumbar disc
Hx	History
L5 distribution	Fifth lumbar nerve root
m	Months
SLR	Straight leg raise

Clinical Presentation

F30, RH, background of high BMI and eczema, mother of three, presented with 2m hx severe intractable R sided sciatica, last 24 h complicated by R foot drop, normal perineal sensation and bladder function, o/e R SLR+ at 30°, absent R ankle reflex, decreased R L5 dermatomal sensation, R EHL/ ADF 0/5

Differential Diagnosis

1. Degenerative (disc prolapse)
2. Neoplastic (nerve sheath tumour or metastatic)
3. If spastic foot drop without sciatica consider parafalcine meningioma
4. Peroneal neuropathy
5. Lumbosacral plexus neuropathy

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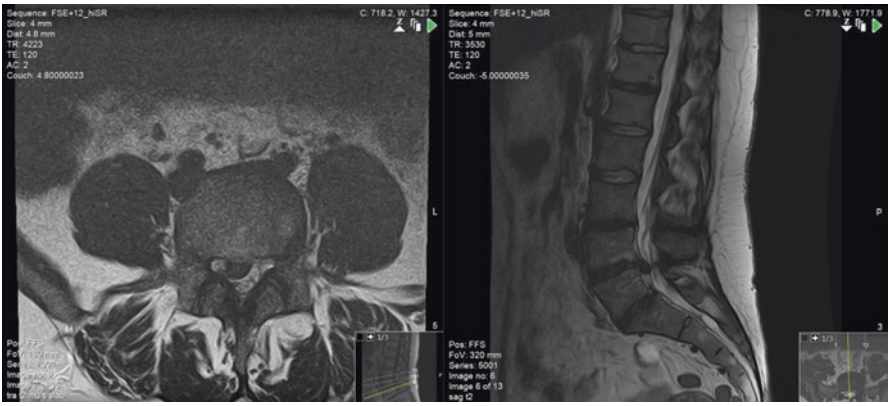
6. Diabetic painless neuropathy
7. Charcot-Marie-Tooth
8 Muscular dystrophy

Investigations

MRI L-S spine revealed large R L4–5 HLD with compression on the R L5 nerve root

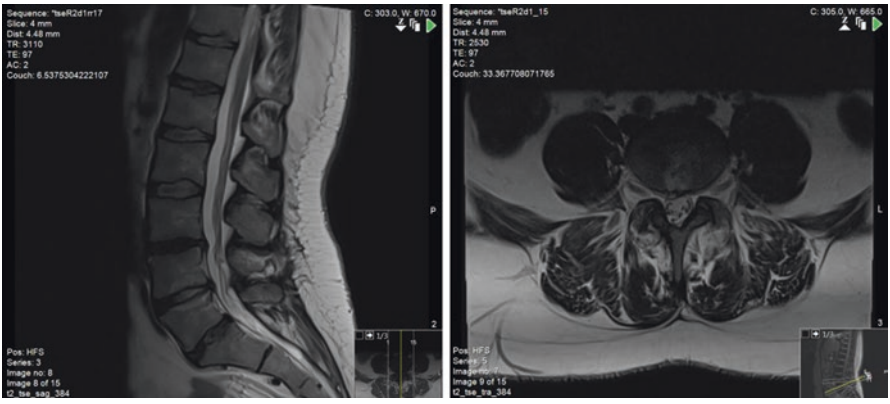


Management

Urgent R L4–5 micro discectomy

Outcome

Significant post op improvement, regained normal power and resolved sciatica
Post op MRI confirmed adequate neuronal decompression



Evidence

Aono H, Nagamoto Y, Tobimatsu H, Takenaka S, Iwasaki M. Degenerative lumbar spondylolisthesis: cohort of 670 patients, and proposal of a new classification. Surgical outcomes for painless drop foot due to degenerative lumbar disorders. J Spinal Disord Tech. 2014;27(7):E258–61. https://doi.org/10.1097/BSD.0000000000000102.

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Gille O, Challier V, Parent H, Cavagna R, Poignard A, Faline A, Fuentes S, Ricart O, Ferrero E, Ould Slimane M, French Society of Spine Surgery (SFCR). Degenerative lumbar spondylolisthesis: cohort of 670 patients, and proposal of a new classification. Orthop Traumatol Surg Res. 2014;100(6 Suppl):S311–5. https://doi.org/10.1016/j.otsr.2014.07.006. Epub 2014 Sep 5

Liu K, Zhu W, Shi J, et al. Foot drop caused by lumbar degenerative disease: clinical features, prognostic factors of surgical outcome and clinical stage. PLoS One. 2013;8(11):e80375. https://doi.org/10.1371/journal.pone.0080375.

Macki M, Syeda S, Kerezoudis P, Gokaslan ZL, Bydon A, Bydon M. Preoperative motor strength and time to surgery are the most important predictors of improvement in foot drop due to degenerative lumbar disease. J Neurol Sci. 2016;361:133–6. https://doi.org/10.1016/j.jns.2015.12.035. Epub 2015 Dec 23

Thoracic Disc Prolapse

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Abbreviations

Ca	Cancer
CT	Computed tomography
EDEM	Extradural extramedullary tumour
IDEM	Intra-dural extramedullary tumour
IDIM	Intradural intramedullary tumour
m	Months
MS	Multiple sclerosis
MRC grading	Medical research council muscle grading
PT	Physiotherapy
T12	12th thoracic vertebrae (same applies to T10–11)

Clinical Presentation

F47, RH, receptionist, lives with family, hx obesity, presents with 3m hx back pain and after a minor fall lower limb weakness and numbness, o/e hypoaesthesia below T12, normal sphincteric function, spastic weakness MRC grade 4/5 of both lower limbs with increased reflexes and clonus

Lower thoracic myelopathy

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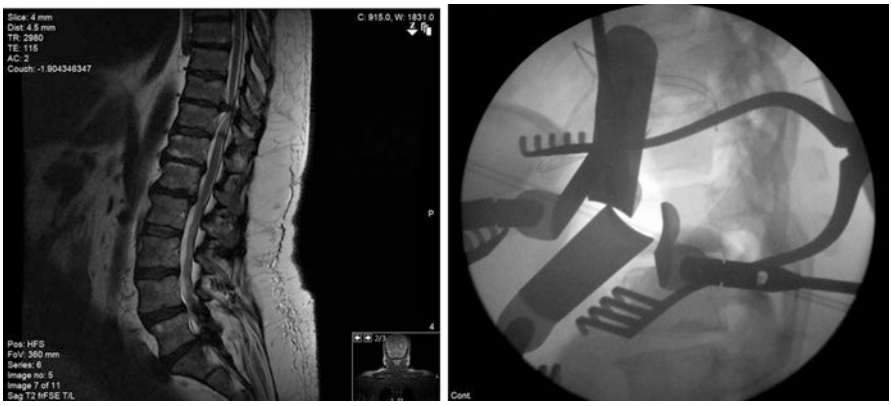
Differential Diagnosis

1. Thoracic spine neoplastic process (EDEM, IDEM, IDIM)
   a. Primary: nerve sheath tumour, meningioma, ependymoma, astrocytoma, sarcoma
   b. Secondary: lymphoma, multiple myeloma, breast Ca
2. Developmental (arachnoid cyst)
3. Degenerative (thoracic disc prolapse)
4. Inflammatory (MS, transverse myelitis)

Investigations

MRI revealed T10–11 disc prolapse causing significant cord compression, CT is useful to assess degree of calcification

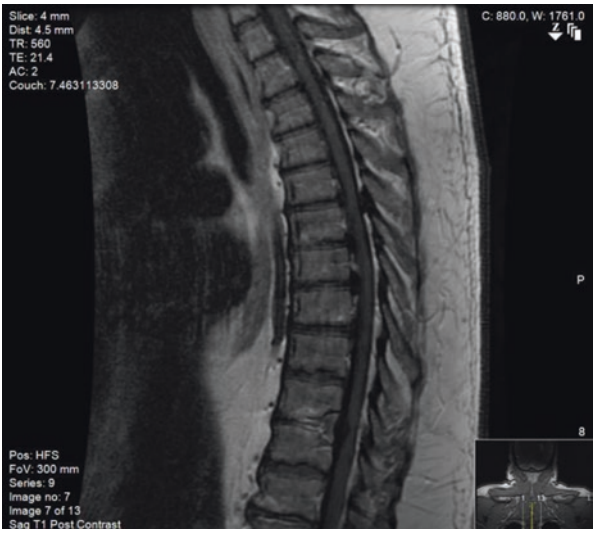


Management

T10/11 Transthoracic discectomy in this case

Other surgical options are transthoracic approach with thoracoscopy, posterolateral approach with costotransversectomy, transpedicular approach

Posterior approach with laminectomy is not suggested due to worsening neurological deficit in 50%

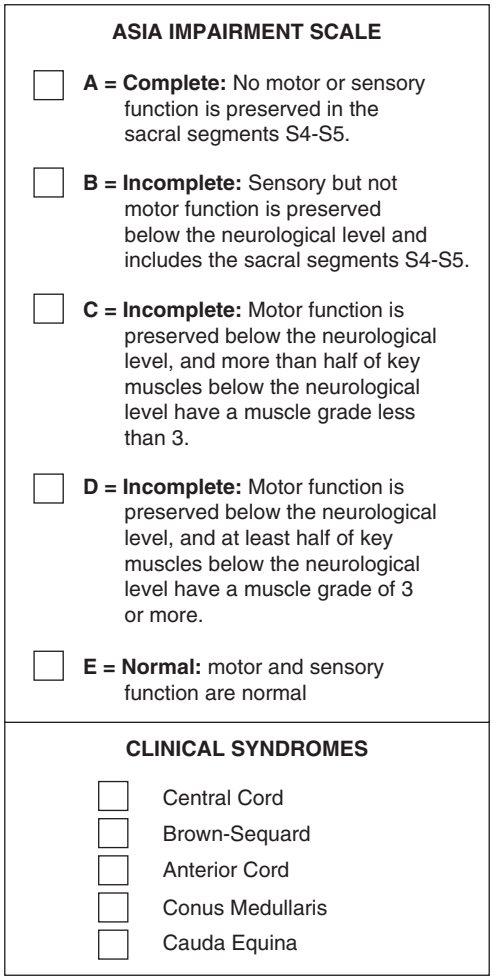


Outcome

Recovered eventually after prolonged PT programme with mild residual mobility impairment but independent

Appendix

Figures



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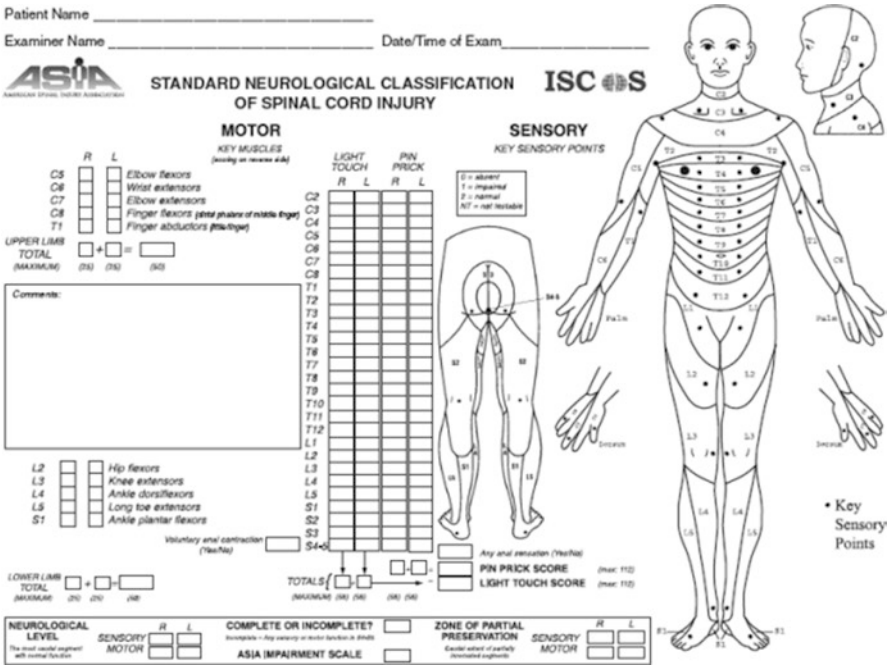


ECOG	Description
0	Fully active, able to carry on all pre-disease performance without restriction.
1	Restricted in physically strenuous activity but ambulatory and able to carry out work of a light or sedentary nature, e.g., light house work, office work.
2	Ambulatory and capable of all selfcare but unable to carry out any work activities. Up and about more than 50% of waking hours.
3	Capable of only limited selfcare, confined to bed or chair more than 50% of waking hours.
4	Completely disabled. Cannot carry on selfcare. Totally confined to bed or chair

Fisher CT Grading Scale

Fisher Group	Blood Pattern on Nonenhanced CT
1	No subarachnoid blood detected
2	Diffuse or vertical layers <1mm thick*
3	Localized clot or vertical layers ≥1 mm thick
4	Intracerebral or IV clot with diffuse or no SAH

"Vertical" cistems: interhemispheric, insular, and ambient.

GLASGOW COMA SCORE

Eye(s) Opening	
Spontaneous	4
To speech	3
To pain	2
No response	1
Verbal Response	
Oriented to time, place, person	5
Confused/disorientated	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Best Motor Response	
Obeys commands	6
Moves to localised pain	5
Flexion withdraws from pain	4
Abnormal flexion	3
Abnormal extension	2
No response	1
Best response	15
Comatose patient	8 or less
Totally unresponsive	3

GOS score	Functional status
5	Resumption of normal life; there may be minor neurologic and/or psychological deficits
4	Able to work in a sheltered environment and travel by public transportation
3	Dependent for daily support by reason of mental or physical disability or both
2	Unresponsive for weeks or months or until death
1	Death

GOS: Glasgow outcome score

Grade	Description	Characteristics
I	Normal	Normal facial function in all areas
II	Mild dysfunction	Slight weakness noticeable on close inspection; may have very slight synkinesis
III	Moderate dysfunction	Obvious, but not disfiguring, difference between 2 sides; noticeable, but not severe, synkinesis, contracture, or hemifacial spasm; complete eye closure with effort
IV	Moderately severe dysfunction	Obvious weakness or disfiguring asymmetry; normal symmetry and tone at rest; incomplete eye closure
V	Severe dysfunction	Only barely perceptible motion; asymmetry at rest
VI	Total paralysis	No movement

Component	ICH score points
GCS score at presentation	
13-15	0
5-12	1
3-4	2
ICH volume (cm^3)	
≥30	1
<30	0
IVH	
Yes	1
No	0
Origin of ICH	
Infratentorial	1
Supratentorial	0
Age	
≥80	1
<80	0
Total ICH score	0-6

ICH-Intracerebral hemorrhage, IVH-Intraventricular hemorrhage, GCS-Glasgow Coma Scale

100	Normal; no complaints; no evidence of disease
90	Able to carry on normal activity; minor signs or symptoms of disease
80	Normal activity with effort; some signs or symptoms of disease
70	Cares for self; unable to carry on normal activity or to do active work
60	Requires occasional assistance but is able to care for most personal needs
50	Requires considerable assistance and frequent medical care
40	Disabled; requires special care and assistance
30	Severely disabled; hospitalisation is indicated, although death not imminent
20	Very sick; hospitalisation necessary; active support treatment is necessary
10	Moribund; fatal processes
0	Dead

Modified Rankin Scale (MRS)	
0	No symptoms
1	No significant disability, despite symptoms; able to perform all usual duties and activities
2	Slight disability, unable to perform all previous activities but able to look after own affairs without assistance
3	Moderate disability; requires some help, but able to walk without assistance
4	Moderately severe disability; unble to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent, and requires constant nursing care and attention
6	Death

Name _____

Date _____

Myelopathy Disability Index

Rising are you able to stand up from an armless straight chair?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Rising are you able to get in and out of bed?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Eating are you able to cut your meat?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Eating are you able to lift a full cup or glass to your mouth?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Walking are you able to walk outdoors on flat ground?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Walking are you able to climb up five steps?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Hygiene are you able to wash and dry your entire body?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Hygiene are you able to get on and off the toilet?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Grip are you able to: Open jars which have been previously opened?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Activities are you able to: Get in and out of a car?

- Without ANY difficulty ☐
- With SOME difficulty ☐
- With MUCH difficulty / assistance required ☐
- Unable to do ☐

Casey ATH, Bland JM, Crockard HA. Development of a functional scoring system for rheumatoid arthritis patients with cervical myelopathy. Ann Rheum Dis 1996;55:901-906

PEDIATRIC GLASGOW COMA SCALE (PGCS)				
	> 1 Year		**< 1 Year**	**Score**
EYE OPENING	Spontaneously		Spontaneously	4
	To verbal command		To shout	3
	To pain		To pain	2
	No response		No response	1
MOTOR RESPONSE	Obeys		Spontaneous	6
	Localizes pain		Localizes pain	5
	Flexion-withdrawal		Flexion-withdrawal	4
	Flexion-abnormal (decorticate rigidity)		Flexion-abnormal (decorticate rigidity)	3
	Extension (decerebrate rigidity)		Extension (decerebrate rigidity)	2
	No response		No response	1
	> 5 Years	**2-5 Years**	**0-23 months**	
VERBAL RESPONSE	Oriented	Appropriate words/phrases	Smiles/coos appropriately	5
	Disoriented/confused	Inappropriate words	Cries and is consolable	4
	Inappropriate words	Persistent cries and screams	Persistent inappropriate crying and/or screaming	3
	Incomprehensible sounds	Grunts	Grunts, agitated, and restless	2
	No response	No response	No response	1
TOTAL PEDIACTRIC GLASGOW COMA SCORE (3-15):				

Grading SAH

WFNS SAH Grade		
WFNS Grade	**GCS Score**	**Major Focal Deficit**
0**		
1	15	-
2	13-14	-
3	13-14	+
4	7-12	+ or -
5	3-6	+ or -
*aphasia, hemiparesis or hemiplegia ** intact anseurysm		

Spetzler-Martin Grading	Points	Supplementary Grading
Size, cm		Age, y
<3	1	<20
3-6	2	20-40
>6	3	>40
Venous drainage		Bleeding
Superficial	0	Yes
Deep	1	No
Eloquence		Compactness
No	0	Yes
Yes	1	No
Total	5	

Scoring Systems

Table A.1 Japanese Orthopaedic Association score (English translation)

Motor function	
Fingers	
0	Unable to feed oneself with any tableware including chopsticks, spoon, or fork, and/or unable to fasten buttons of any size
1	Can manage to feed oneself with a spoon and/or fork but not with chopsticks
2	Either chopstick-feeding or writing is possible but not practical, and/or large buttons can be fastened
3	Either chopstick-feeding or writing is clumsy but practical, and/or cuff buttons can be fastened
4	Normal
Shoulder and elbow (evaluated by MMT score of the deltoid or biceps muscles, whichever is weaker)	
−2	MMT 2 or less
−1	MMT 3
−0.5	MMT 4
0	MMT 5
Lower extremity	
0	Unable to stand up and walk by any means
0.5	Able to stand up but unable to walk
1	Unable to walk without a cane or other support on a level
1.5	Able to walk without support but with a clumsy gait
2	Walks independently on a level but needs support on stairs
2.5	Able to walk independently when going upstairs, but needs support when going downstairs
3	Capable of fast but clumsy walking
4	Normal

Table A.1 (continued)

Motor function	
Sensory function	
Upper extremity	
0	Complete loss of touch and pain sensation
0.5	50% or less normal sensation and/or severe pain or numbness
1	More than 60% normal sensation and/or moderate pain or numbness
1.5	Subjective numbness of slight degree without any objective sensory deficit
2	Normal
Trunk	
0	Complete loss of touch and pain sensation
0.5	50% or less normal sensation and/or severe pain or numbness
1	More than 60% normal sensation and/or moderate pain or numbness
1.5	Subjective numbness of slight degree without any objective sensory deficit
2	Normal
Lower extremity	
0	Complete loss of touch and pain sensation
0.5	50% or less normal sensation and/or severe pain or numbness
1	More than 60% normal sensation and/or moderate pain or numbness
1.5	Subjective numbness of slight degree without any objective sensory deficit
2	Normal
Bladder function	
0	Urinary retention and/or incontinence
1	Sense of retention and/or dribbling and/or thin stream and/or incomplete continence
2	Urinary retardation and/or pollakiuria
3	Normal

Table A.2 Modified Japanese Orthopaedic Association score

Motor dysfunction score of the upper extremities	
0	Inability to move hands
1	Inability to eat with a spoon, but able to move hands
2	Inability to button shirt, but able to eat with a spoon
3	Able to button shirt with great difficulty
4	Able to button shirt with slight difficulty
5	No dysfunction
Motor dysfunction score of the lower extremities	
0	Complete loss of motor and sensory function
1	Sensory preservation without ability to move legs
2	Able to move legs, but unable to walk
3	Able to walk on flat floor with a walking aid (i.e., cane or crutch)
4	Able to walk up and/or down stairs with hand rail

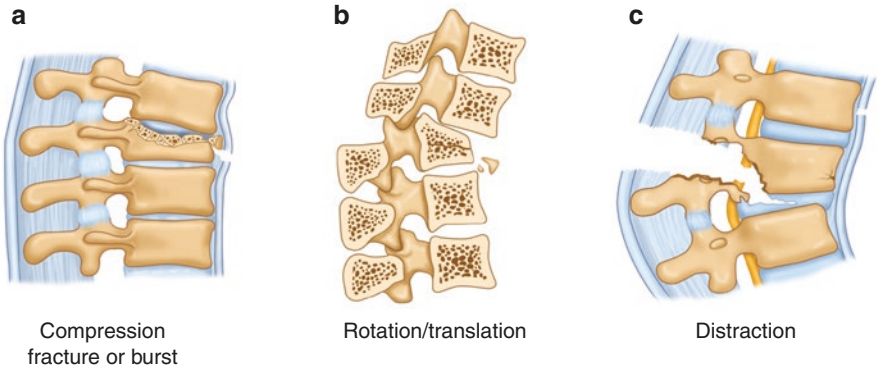
(continued)

Table A.2 (continued)

Motor dysfunction score of the upper extremities	
5	Moderate to significant lack of stability, but able to walk up and/or down stairs without hand rail
6	Mild lack of stability but walks with smooth reciprocation unaided
7	No dysfunction
Sensory dysfunction score of the upper extremities	
0	Complete loss of hand sensation
1	Severe sensory loss or pain
2	Mild sensory loss
3	No sensory loss
Sphincter dysfunction score	
0	Inability to micturate voluntarily
1	Marked difficulty with micturition
2	Mild to moderate difficulty with micturition
3	Normal micturition

TLICS scoring

Parameter	Points
Morphology	
Compression fracture	1
Burst fracture	2
Translational/rotational	3
Distraction	4
Neurologic involvement	
Intact	0
Nerve root	2
Cord, conus medullaris	
Incomplete	3
Complete	2
Cauda equina	3
Posterior ligamentous complex	
Intact	0
Injury suspected/indeterminate	2
Injured	3



Morphology. (a) Compression (compression fracture or burst). (b) Rotation/translation. (c) Distraction

Management as per TLICS score

Management	Points
Nonoperative	0–3
Nonoperative or operative	4
Operative	≥5

Mnemonics

Common Neurosurgical Mnemonics

1. Pituitary Region Mass

   SATCHMO

- **S:** sarcoid, sellar tumour (pituitary adenoma)
- **A:** aneurysm
- **T:** teratoma or tuberculosis (and other granulomatous diseases)
- **C:** craniopharyngioma, cleft cyst (Rathke), chordoma
- **H:** hypothalamic glioma, hamartoma of tuber cinereum, histiocytosis
- **M:** meningioma, metastasis
- **O:** optic nerve glioma

   E at the end and moving 'histiocytosis' to this last letter as:

- **E:** eosinophilic granuloma or epidermoid/dermoid/teratoma

   MOuSTACHE

- **M:** meningioma, **m**etastasis
- **O:** optic nerve glioma
- **S:** sarcoid, sellar tumour (pituitary adenoma)
- **T:** teratoma or tuberculosis (and other granulomatous diseases)
- **A:** aneurysm
- **C:** craniopharyngioma, cleft cyst (Rathke), chordoma
- **H:** hypothalamic glioma, hamartoma of tuber cinereum
- **E:** eosinophilic granuloma or epidermoid/dermoid/teratoma

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2. Corpus callosum hyperintensity

   I MADE A PHD

- **I:** infections (e.g. tuberculosis, varicella, rotavirus, HSV)
- **M:** Marchiafava-Bignami syndrome
- **A:** AIDS encephalopathy
- **D:** diffuse axonal injury and diffuse vascular injury
- **E:** epilepsy
- **A:** autoimmune disorders: haemolytic uremic syndrome-related encephalopathy
- **P:** posterior reversible encephalopathy syndrome
- **H:** hypoglycemia
- **D:** demyelination (e.g. multiple sclerosis, ADEM)

3. Foramen ovale

   OVALE

- **O:** otic ganglion (inferior)
- **V:** V3 cranial nerve (mandibular division of the trigeminal nerve)
- **A:** accessory meningeal artery
- **L:** lesser petrosal nerve
- **E:** emissary veins

4. Tumours of the posterior fossa in children

   BEAM

- **B:** brainstem glioma
- **E:** ependymoma
- **A:** astrocytoma (pilocytic) (85%)
- **M:** medulloblastoma

5. The internal carotid artery segments, according to the Bouthillier classification

   C'mon Please Learn Carotid Clinical Organizing Classification

- **C:** cervical segment
- **P:** petrous segment
- **L:** lacerum segment
- **C:** cavernous segment
- **C:** clinoid segment
- **O:** ophthalmic segment
- **C:** communicating segment

6. Contents of Cavernous Sinus

   O TOM CAT

- **O:** oculomotor nerve
- **T:** trochlear nerve
- **O:** ophthalmic branch of trigeminal nerve
- **M:** maxillary branch of trigeminal nerve
- **C:** internal carotid artery
- **A:** abducens nerve
- **T:** trochlear nerve

7. Cerebellopontine angle mass

   AMEN or MEAN

- **A:** acoustic schwannoma (~80%)
- **M:** meningioma (~10%)
- **E:** ependymoma (~5%)
- **N:** neuroepithelial cyst (arachnoid/epidermoid) (~5%)

   SAME

- **S:** schwannoma
  - acoustic schwannoma much more common than a trigeminal schwannoma
- **A:** aneurysm, arachnoid cyst
- **M:** meningioma, metastasis
- **E:** epidermoid cyst, ependymoma

8. Cerebral ring enhancing lesions

   MAGIC DR or DR MAGIC

- **M:** metastasis
- **A:** abscess
- **G:** glioblastoma
- **I:** infarct (subacute phase)
- **C:** contusion
- **D:** demyelinating disease
- **R:** radiation necrosis or resolving haematoma

DR MAGIC L or MAGICAL DR
"L" and "A" for lymphoma and AIDS-related CNS disease.

- **D:** demyelinating disease
- **R:** radiation necrosis or resolving haematoma
- **M:** metastasis
- **A:** abscess
- **G:** glioblastoma
- **I:** infarct (subacute phase)
- **C:** contusion
- **A:** AIDS
- **L:** lymphoma

9. Parts of Corpus Callosum (Anterior to posterior)

- **R:** rostrum
- **G:** genu
- **B:** body (trunk)
- **S:** splenium

10. Hemorrhagic Metastasis

MR CT BB

- **M:** melanoma: metastatic melanoma to brain
- **R:** renal cell carcinoma
- **C:** choriocarcinoma
- **T:** thyroid carcinoma, teratoma
- **B:** bronchogenic carcinoma
- **B:** breast carcinoma

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