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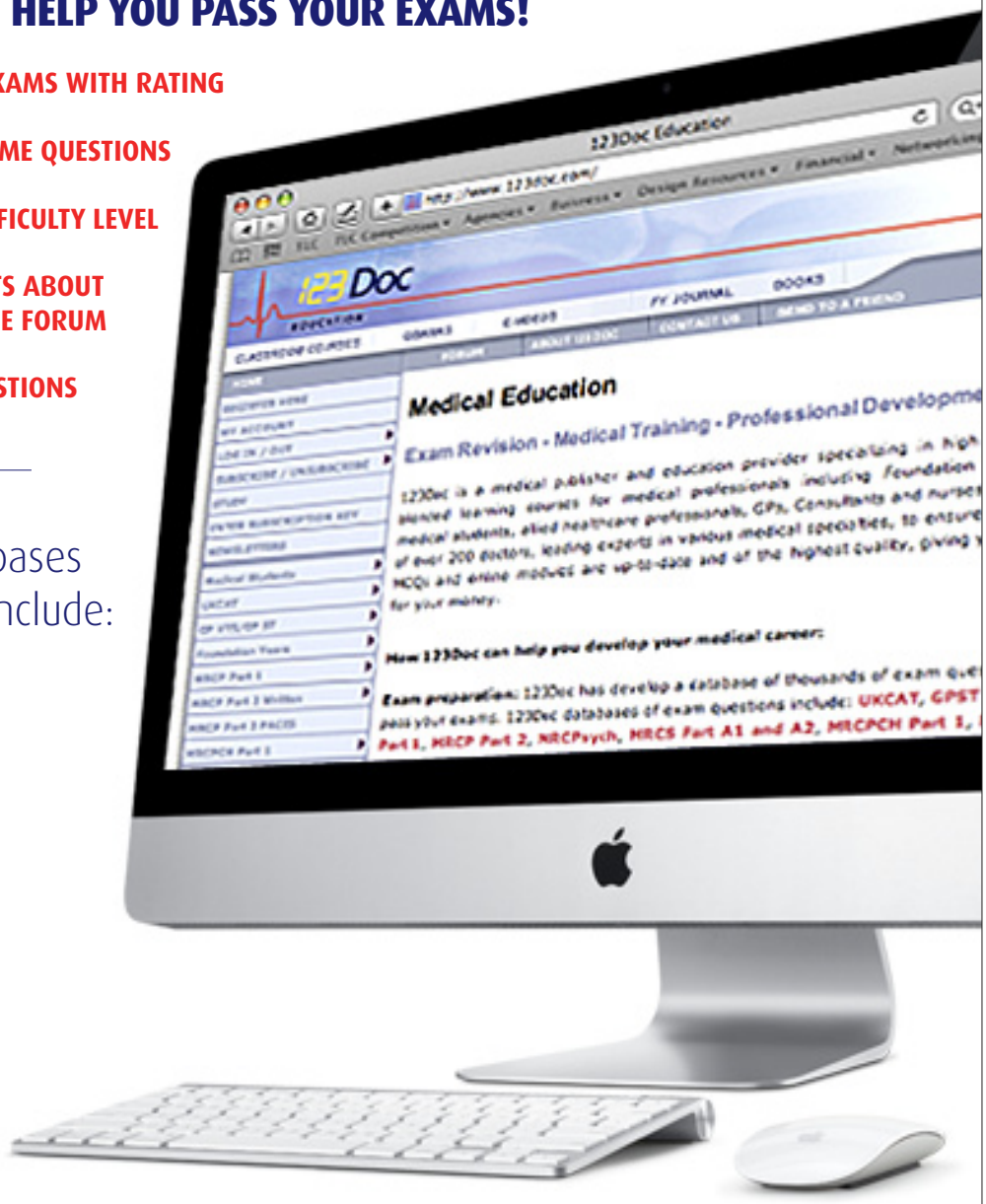

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Volume 10

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ASSESSMENT OF A PATIENT WITH PAPILLOEDEMA

S Blundell, G Giovannoni

Abstract

In the world of medicine, we have been able to look at the fundus since the creation of the first ophthalmoscope by Hermann von Helmholtz in 1851. In spite of all the medical advances in the past 165 years, we still rely on funduscopy as an essential clinical skill to identify abnormalities such as papilloedema. Although the word papilloedema is derived from the optic papilla, the optic nerve head, and oedema, the term papilloedema refers only to swelling of the optic nerve head/optic disc secondary to raised intracranial pressure (ICP).

It is therefore an important clinic finding to be able to identify in any clinical setting from A&E to GP, as it indicates a potentially serious underlying aetiology and requires urgent assessment. Additionally, if papilloedema is left untreated it can lead to progressive permanent visual loss. Papilloedema can be very quickly identified using an ophthalmoscope and although can be straightforward to the experienced examiner, early signs of papilloedema can be easily missed by the less familiar examiner. Here we discuss an approach full assessment of a patient found to have papilloedema, describe how to identify papilloedema and differentiate it from its differentials.

Case Presentation

A 35 year old females presents to the neurology clinic with a history of headaches of increasing frequency and severity. She reports daily headaches, worse in the morning and no longer responding to simple analgaesia. She denies any visual symptoms or any other focal neurological problems. She has no past medical history and takes only takes paracetamol 1g four times per day with occasional codeine. On examination of the upper limbs and lower limbs, there is no focal neurological abnormality. There are no cranial nerve deficits but funduscopy reveals bilateral papilloedema.

**This case is purely illustrative and is not based on a true patient.*

Assessment Of A Patient With Papilloedema
Patient Management

Clinical assessment of the patient

Often papilloedema will be found on examination in the context of a specific presentation, for example in the patient presenting with a headache, as in the case presentation above. As with every assessment of a patient, a thorough history and examination should be taken and these may elicit classical features of papilloedema in addition to pointing towards the underlying cause of the papilloedema.

Often the history taken will focus on the cause of papilloedema/raised ICP, as papilloedema will not be identified until the examination (for causes of papilloedema see table 1). However, if raised ICP is suspected from the history then further questions can be asked that may reveal symptoms due to papilloedema such as visual symptoms and the absence of pain. As papilloedema is painless, the presence of retro-orbital eye pain would suggest an alternative diagnosis, which is usually inflammatory such as example papillitis.

In terms of visual symptoms, papilloedema may not cause visual symptoms until the later stages. However, transient visual obscurations (TVOs) are considered classical of papilloedema and become more common as the papilloedema progresses. TVOs are sudden losses of vision lasting seconds to minutes and can be unilateral or bilateral. They are often positional or occur with increases of intrathoracic pressure, such as with coughing or the Valsalva manoeuver. Visual acuity and visual fields are often well preserved in the earlier stages of papilloedema and may not be noticed by the patient.

Causes of raised intracranial pressure and subsequent papilloedema
1) Space-occupying lesions of the CNS - tumours, intracranial haemorrhage
2) Reduced CSF drainage a. Reduced absorption – inflammation, infection (e.g. meningitis), impaired venous drainage (e.g. venous sinus thrombosis, mediastinal masses) b. Blocked drainage – obstructive hydrocephalus
3) Increased CSF production – choroid plexus tumours
4) Idiopathic intracranial hypertension (IIH, also known as pseudotumour cerebri)
5) Drugs – tetracyclines, corticosteroids, lithium, nalidixic acid, accutane

Table 1

ASSESSMENT OF A PATIENT WITH PAPILLOEDEMA

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Physical Examination

A thorough assessment of the anterior visual pathway is the essential part of assessment of a patient with papilloedema, which includes examination of the visual fields, visual acuity, colour vision, pupillary responses to light and funduscopy. This should be part of a full neurological examination.

Enlargement of the blind spot is an early diagnostic feature of papilloedema and may be the only defect elicited in the earlier stages of papilloedema(1). As papilloedema progresses there may be loss of the peripheral visual fields, as the persistent raised ICP causes permanent damage to the retinal neurons causing loss of the peripheral axons(2). Visual acuity may be affected if there is a severe visual field defect or if there are retinochoroidal folds over the macula.

Ophthalmoscopic findings

The features of papilloedema changes as it progresses through its stages. The Frisen classification divides papilloedema into stages 0-5 that focus on the changes seen at the margin of the disc, as these are the most consistent changes(3). The stages of papilloedema have been described by others and these look at additional features that are not consistently present but often aid identification of papilloedema. Here we will describe the features of papilloedema as the classification system described by Sanders (1997)(4).

Early papilloedema

In the early stage of papilloedema, changes are best seen at the margin of the optic disc(3). Blurring of the nasal portion of the optic disc is seen first with sparing of the temporal margin, resulting in a C-shaped halo (figure 1A). The use of a red-free light may help with identification of the blurred optic disc margins. There may be associated disc hyperaemia, due to capillary dilatation, and loss of the normal spontaneous venous pulsations (SVP). However, the loss of normal SVP may be transient and therefore may not be seen. There is also a minority of individuals who have no SVP with a normal intracranial pressure (5).

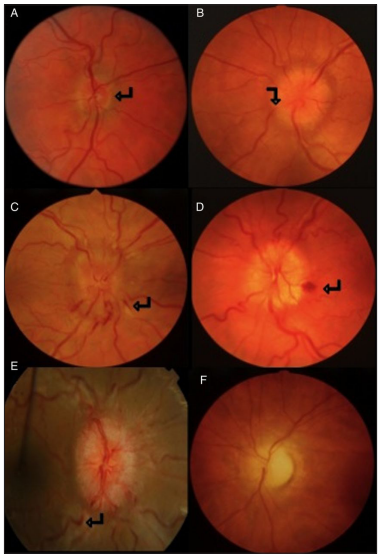

Figure 1

Fundus photographs showing stages of papilloedema. A – Early papilloedema with blurring of the nasal disc margin (arrow) and sparing of the temporal margin. B-E – Fully developed papilloedema with blurring of entire disc margin, obscuration of vessels as they leave the optic disc (B), haemorrhages (C, D, E), and tortuous veins (E, arrow). Optic disc atrophy (F). From Mollan et al (2014)(9) from Pract Neurol doi:10.1136/practneurol-2014-000821 under CC BY-NC 3.0 Creative Commons Attribution Non-Commer. http://creativecommons.org/licenses/by-nc/3.0/

Fully developed papilloedema

Blurring of the nasal optic disc margin is followed by elevation of the nasal margin, blurring of the temporal margin and finally elevation of the temporal margin. Elevation of the disc will result in obscuration of the major retinal vessels as they leave the disc (figure 1B). There may be associated flame haemorrhages and evidence of ischaemic events as cotton wool spots, which are both a consequence of acute compromise to the perfusion to the prelaminar tissues of the optic nerve head(4) (figure 1C and 1D). It may also be possible to see Paton's lines, which are retinochoroidal folds that develop as a results of the optic disc swelling. Importantly, there is preservation of the physiological cup.

Chronic/vintage papilloedema

Chronic papilloedema develops after several months of raised intracranial pressure. The cause of chronic papilloedema is most commonly due to CNS tumours and idiopathic intracranial hypertension where the increase in ICP may be a gradual process(4). Consequently, there are usually no haemorrhages or cotton wool spots that are seen, as retinal perfusion is maintained and any previous haemorrhages may have resolved(6).

The optic disc is pale with loss of the physiological cup and obscuration of the retinal vessels as they leave the optic disc. There may be small white dots on the optic disc that is due to axonal loss/degeneration that may be confused with superficial drusen. These white dots are due to aggregates in the axon and often take the form of a macular star.

Atrophic papilloedema

An atrophic optic disc is the consequence of long standing untreated papilloedema and subsequent axonal loss. The disc is pale and there is no swelling of the disc (figure 1F).

Differential Diagnosis

The features of papilloedema can easily be mistaken for pseudopapilloedema or local causes of optic disc oedema. These are important differentials that need to be excluded early during patient assessment. Pseudopapilloedema may be seen when there are normal physiological variants such as with hypermetropic, buried drusen (figure 2A), tilted discs or myelinated nerve fiber layer and will be bilateral. Local causes of optic disc oedema will usually be unilateral but may be bilateral in some cases. Similarly, a minority of patients with true papilloedema will present with unilateral papilloedema due to natural anatomical variations(7). The local causes can be divided into inflammation, infiltration, vascular causes or tumours of the nerve/sheath.

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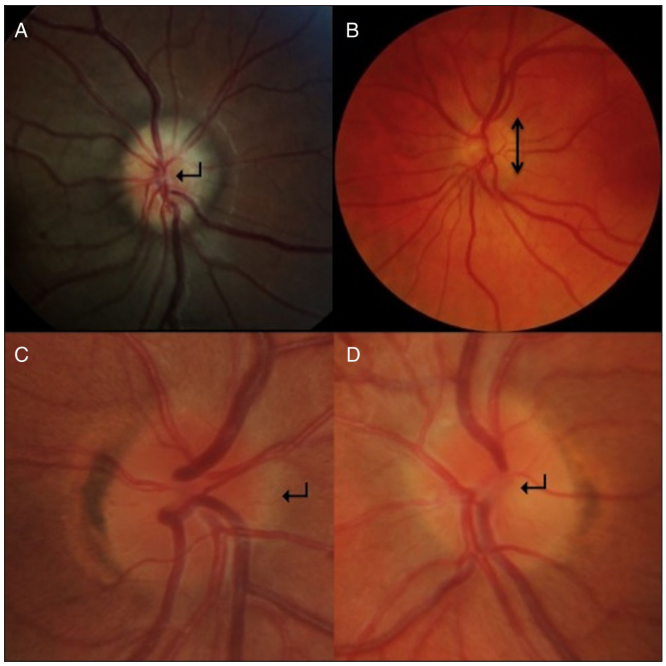

Figure 2

Fundus photographs showing examples of pseudopapilloedema. A – Optic nerve drusen. B, C, D – Normal anomalies with small optic disc (arrow in B) with loss of the physiological cup (arrow in D) and blurring of the nasal margin (arrow in C). From Mollan et al (2014)(9) from Pract Neurol doi:10.1136/practneurol-2014-000821 under CC BY-NC 3.0 Creative Commons Attribution Non-Commer: http://creativecommons.org/licenses/by-nc/3.0/

Key distinguishing features between papilloedema and pseudopapilloedema Pseudopapilloedema can be difficult to differentiate from papilloedema. The disc may be hyperaemic in true papilloedema with retention of the physiological cup until the later stages of papilloedema. Vascular congestion, loss of spontaneous venous pulsations and obscurations of the peripapillary blood vessels are features of true papilloedema.

There also may be associated haemorrhages and cotton wool spots seen in papilloedema that are not present in pseudopapilloedema. In contrast, there is loss of cupping (figure 2B-D) and the optic margin is often irregular in pseudopapilloedema, as seen with drusen.

Further assessment and investigations

Further assessment should include measuring the blood pressure to rule out severe hypertension. Basic blood tests should include a full blood count, urea and electrolytes, liver function tests and erythrocyte sediment rate. Imaging will depend upon where the patient is presenting. In acute assessment a CT head may be the first mode of imaging available.

Otherwise, an MRI would provide better imaging of the parenchyma, ventricles and dural venous sinuses. A magnetic resonance venography would fully assess for a venous sinus thrombosis. A lumbar puncture should be performed if all imaging is normal with the basic investigations including measuring the opening pressure, white cell count, red blood cell count, glucose, protein, Gram stain and culture. Imaging of the retina, such as with optical coherence topography can help to differentiate between causes of pseudopapilloedema such as buried drusen(8).

Conclusion

Identification of papilloedema is an essential clinical skill as it is an indicator of raised intracranial pressure. Assessment of papilloedema requires careful examination of the optic discs of both eyes with close attention to the optic disc characteristics, margins, vessels and any additional features such as haemorrhages and cotton wool spots.

This should be done in the context of a thorough history and full neurological examination. Once papilloedema is identified, further assessment should begin including measurement of the blood pressure, simple blood tests and imaging of the head.

Questions

Q1. Which of the following is consistently found in the earliest stages of papilloedema?

A. A pale optic disc

B. Flame haemorrhages and cotton wool spots

C. Blurring of the nasal margins of the optic disk with temporal sparing

D. Elevation of the nasal portion of the optic disc and blurring of all portions of the optic disc margins

E. Loss of spontaneous venous pulsations.

ASSESSMENT OF A PATIENT WITH PAPILLOEDEMA

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Q2. Which investigation is not routinely done to investigate the cause of a headache similar to that in the case above?

A. Blood tests including full blood count, urea and electrolytes, liver function tests and ESR

B. Optical coherence topography

C. MRI brain

D. Lumbar puncture

Answers

Q1 – C

Blurring of the nasal margins of the optic disk with temporal sparing is an early consistent sign of papilloedema. Elevation of the disc margin follows blurring of the disc margin and flame haemorrhages and cotton wool spots are also found later and are not consistently found.

Loss of spontaneous venous pulsations is not always seen, as it may be transient. There are also a proportion of individuals who have no spontaneous venous pulsations in the absence of papilloedema. A pale optic disc is a late sign.

Q2 – B

Basic blood tests are done as part of the investigation. The imaging modality of choice is an MRI brain. However, a CT head may be the done in the acute setting, as MRI may not be available. A lumbar puncture will be done if the imaging is normal. Optical coherence topography is not routinely part of the investigation of papilloedema.

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ASSESSMENT OF BLACKOUTS

EJ Pegg, T Majeed

Abstract

Patients with blackouts frequently present to the Medical Assessment Unit or Emergency Department. This article uses a case history to highlight the importance of taking a good history and provides a practical approach to the assessment of patients with blackouts including how to differentiate between seizures, syncopal episodes and non-epileptic attacks.

Case History

A 65 year old lady was an inpatient on a surgical ward following a hip replacement operation. During her admission she had a blackout associated with urinary incontinence and limb jerking. A seizure was suspected and she was referred to the on call neurology specialist trainee for review.

Further history was obtained. The patient felt well prior to the blackout with no preceding symptoms including light headedness, palpitations or nausea. She had been helped to a commode by a health care assistant who did not notice any pallor. Shortly after sitting on the commode, she blacked out for a few seconds and fell to the floor. The health care assistant reported that there were a few brief twitches of her body and she regained consciousness after 1-2 seconds. There was no tongue biting. She seemed a little disorientated initially but there was no confusion or altered behaviour.

There was no history of previous blackouts and her past medical history was significant only for osteoarthritis and hypertension. Medications included ramipril and co-codamol. There was no relevant family history. She was an ex-smoker and didn't drink alcohol. She was retired and did not have a driving licence.

Examination including full neurological and cardiovascular examination was unremarkable. There was no postural drop in blood pressure. Routine blood tests and 12 lead ECG were normal. A clinical diagnosis of micturition syncope was made and explained to the patient.

Assessment Of The Patient Presenting With A Blackout

Causes of blackouts

Before taking a history, it is worthwhile considering the common causes of blackouts:

- *Seizures*
- *Syncope- vasovagal, cardiac, orthostatic, carotid sinus hypersensitivity, situational (eg cough, micturition)*
- *Non epileptic attacks*

Seizures

A seizure can be defined as an episode of abnormal neuronal activity in the brain that causes an increase in motor function, convulsions, or a change in behaviour or sensory perception.

Seizures can be caused by a wide range of problems including central nervous system infection, cerebrovascular disease, subarachnoid haemorrhage, raised intracranial pressure, brain tumour, metabolic causes (e.g. hypoglycaemia), drug intoxication or withdrawal (including alcohol), idiopathic, pre-eclampsia, fever (febrile convulsions), neurodegenerative conditions, autoimmune conditions and genetic diseases.

Seizures can be classified by the location of onset within the brain (1). When the onset of abnormal neuronal activity is in both cerebral hemispheres simultaneously, a generalised seizure occurs and is usually (other than in myoclonic seizures) accompanied by loss of consciousness. A common example of a generalised seizure is a tonic-clonic seizure. Other generalised seizure types include absence, myoclonic, atonic and tonic.

When the onset of a seizure is in a focal area of the brain, a focal seizure occurs which may cause, for example, jerking of one limb or subjective sensory phenomena. Focal seizures can occur with or without impairment of consciousness or awareness.

ASSESSMENT OF BLACKOUTS

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Focal seizures can evolve to generalised seizures and produce a tonic-clonic seizure. In adults most generalised tonic clonic-seizures are initially focal onset, though this won't always be recognized or remembered.

Epilepsy may be diagnosed when there are at least 2 unprovoked seizures occurring more than 24 hours apart or following a single seizure where investigations such as MRI or EEG support an enduring predisposition to seizures (2). The diagnosis of both seizure and epilepsy should be made by a neurologist (3).

Syncope

Syncopal episodes can be caused by a variety of mechanisms. Cardiac syncope is caused by abnormalities which reduce cardiac output and consequently reduce cerebral perfusion such as arrhythmias or structural abnormalities.

Neurally mediated syncope is thought to be caused by reflex hypotension or bradycardia in response to a certain trigger. Examples include vasovagal syncope (which may be triggered by prolonged standing), cough syncope, swallow syncope and micturition syncope (as with the case above).

Syncope due to carotid sinus hypersensitivity results from a heightened response of the carotid baroceptors in response to stimulation of the overlying area in the neck e.g. shaving or a tight collar. Syncope can also be caused by postural hypotension.

If syncope is prolonged and reflex asystole occurs, reflex anoxic seizures can occur. In this situation, there will be a history of a syncopal episode followed by a generalised convulsion.

Non-Epileptic Attacks

Non Epileptic Attack Disorder (NEAD) causes episodes which may appear like generalised tonic-clonic seizures but without any abnormal cerebral neuronal activity. They can sometimes be difficult to differentiate from an epileptic seizure and require specialist assessment; a detailed description of the nature of the movements or even better a video of the attacks can be invaluable.

Blackout history

When taking a blackout history, it is useful to consider events before, during and after the blackout. Taking a collateral history from any witnesses is essential and can be done over the phone if the witness is no longer with the patient.

Before

- *What was the patient doing at the time? This is relevant particularly for syncope for example somebody with a vasovagal episode may be stood in a hot, crowded room.*

- *Were there any preceding symptoms? Palpitations, chest pain and light headedness points towards a cardiac syncope whereas somebody with a vasovagal episode may report nausea, feeling hot and visual disturbance or tinnitus. Patients with a generalised seizure usually have no warning symptoms.*

- *Was there any pallor noted by witnesses? If so this points towards a syncopal episode.*

- *Is there any history of headache or fever to suggest meningitis?*

During

This information would mainly come from a witness.

- *Was there loss of consciousness and for how long? A patient with a non-epileptic attack may be aware of people talking to them and that their limbs are shaking.*

- *Were the limbs jerking and for how long? What did it look like? A patient with a generalised tonic clonic seizure has symmetrical limb jerking but it is also possible to have a few brief limb jerks following a syncopal episode, including vasovagal syncope. The movements in non-epileptic attacks are often flailing with pelvic thrusting.*

- *Was there any tongue biting? Lateral tongue biting is typical of a seizure.*

- *Was there any incontinence? Bladder (and sometimes bowel) incontinence is common during a generalised seizure but can occur also with syncopal episodes. Incontinence is not typically a feature of non- epileptic attacks but can occur.*

- *Were the eyes open? In epileptic seizures, the eyes are usually open whereas in non-epileptic attacks they are often closed.*

- *Did the patient appear cyanosed? If the patient is in hospital, did their oxygen saturations fall? If so, this points towards an epileptic attack.*

After

- *Was there any confusion or tiredness and if so for how long did it last? Following a syncopal episode, recovery will be rapid. After a seizure, confusion and tiredness (post-ictal symptoms) last from several minutes to hours. Post-ictal symptoms should not occur following a non -epileptic attack.*

ASSESSMENT OF BLACKOUTS

EJ Pegg, T Majeed

Past medical History

It is important to check if there are risk factors for blackouts.

- *Seizures and epilepsy are more common if there is a history of perinatal birth injury, febrile convulsions, stroke, head injury or bacterial meningitis.*
- *Ischaemic heart disease and arrhythmias are risk factors for cardiac syncope.*
- *Non epileptic attack disorder can be associated with depression, self-harm and other functional disorders. It is important to bear in mind however that NEAD can occur in the absence of these diagnoses and that depression can occur in association with epilepsy.*

Drug History

It is important to take a full drug history and consider if any medications can cause:

- *Orthostatic syncope (e.g. antihypertensives).*
- *Prolonged QTc interval (many).*
- *A lowered seizure threshold eg tramadol, baclofe*n.

Family History

- *Epilepsy- some types of epilepsy have a genetic predisposition.*
- *Sudden death in young adults- this should alert you to the possibility of an underlying cardiac cause.*

Social History

It is important to ask about the following:

- *Drugs and alcohol - both (or withdrawal) can cause seizures.*
- *Driving - due to implications following a blackout (see below).*
- *Smoking- increased risk of malignancy (eg brain metastases).*
- *Hobbies and occupations – some are not advisable/ possible if there is a history of epilepsy.*
- *Contraception - this is relevant for women with epilepsy as antiepileptic medication may interact with hormonal contraceptives and may be teratogenic.*

Examination

General

This should include the patient's conscious level, any indicators of infection (including meningism) or any signs of alcohol abuse such as withdrawal symptoms.

Neurological

This should include a full neurological examination to search for localising signs and should always include looking at the fundi for papilloedema.

Cardiovascular

In particular listen for an ejection systolic murmur (aortic stenosis) and assess the heart rhythm. It is also important to check for postural hypotension by recording the patient's lying and standing blood pressure.

Investigations

- It is imperative to check blood glucose levels in anyone with a suspected seizure.
- Baseline bloods should be carried out to look for infection and electrolyte disturbance
- A 12 lead ECG must be done in any patient with a seizure or blackout to look for arrhythmias, conduction abnormalities and syndromes associated with sudden death eg Brugada Syndrome
- If a cardiac arrhythmia is suspected but the 12 lead ECG is normal, prolonged ECG monitoring should be carried out.
- An echocardiogram is required if a structural cardiac abnormality is suspected.
- MRI/CTB. In the acute stage following a first seizure, CT brain imaging is indicated to look for causes such as tumour, abscess or haemorrhage. MRI should subsequently be carried out to look in more detail for a structural cause for the seizure.
- An electroencephalogram (EEG) is used to help classify the type of epilepsy. It has a relatively low sensitivity and can be abnormal in patients without epilepsy therefore it cannot be used diagnostically.

Management

The management of a blackout depends on the suspected underlying cause. First seizures or suspected NEAD should be referred to neurologist. If there is cardiac syncope, a cardiology referral should be made.

All patients who have had a seizure or an unexplained or unprovoked blackout must be told that they cannot drive and that they must contact the DVLA.

ASSESSMENT OF BLACKOUTS

EJ Pegg, T Majeed

Test yourself

1. Which one of the following investigations is always indicated after a suspected first seizure presenting to the medical assessment unit?

a) 12 lead ECG

b) Echocardiogram

c) EEG

2. Which of the following aspects of social history is relevant following a blackout? Tick all that apply.

a) alcohol intake
b) occupation
c) driving

3. Which one of the following descriptions is in keeping with a blackout due to a generalised tonic-clonic seizure?

a) Shaving, felt light headed, fell to floor, loss of consciousness for 1 or 2 seconds, felt back to normal straight away.

b) Stood in a lecture hall, felt hot and nauseated, appeared pale, unconscious for a few seconds with 3 or 4 jerks of limbs, back to normal within few minutes.

c) No warning, unconscious for 1-2 minutes, symmetrical limb jerking, cyanosis, incontinence, lateral tongue biting, confused afterwards.

Answers

1. Answer = a.

A 12 lead ECG is vital in anybody presenting with a blackout. An echocardiogram is carried out if there is a suspected structural cardiac abnormality causing syncope. EEGs are used to classify the type of epilepsy, and will be requested by the neurologist if indicated.

2. Answer = a,b & c.

It is important to consider alcohol withdrawal (and intoxication) as a possible cause for a seizure. If a patient has had a blackout or seizure, it has implications on driving regulations and some occupations.

3. Answer = c.

A is consistent with carotid sinus hypersensitivity and b with a vasovagal episode.

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EPILEPSY ON THE ACUTE MEDICAL UNIT

P Nash, HR Cock

Abstract

Presentations to Emergency Departments and Acute Medical Units involving suspected seizures or epilepsy are common. Simple, practical advice on how to approach such patients can be difficult to find. This review will provide this advice in a step-by-step fashion for junior doctors who will typically have limited previous experience of epilepsy. Six commonly presenting scenarios are outlined, each with guidance on how to approach the problem followed by initial steps in investigation and management. These are derived from evidence based guidelines and personal experience of the senior author.

Introduction

Approximately 1% of the UK population are on treatment for a diagnosis of epilepsy, with and an estimated 20-30% more who may not yet be recognized/treated and around 0.5/100,000 new presentations every year (1). Incomplete seizure control may be associated with profound developmental, behavioral and psychological effects, which could lead to poor health outcomes (2).

Although 70% of people with epilepsy (PWE) could be seizure free with optimal care, epilepsy is still the largest single source of one day admissions amongst neurological conditions (3), so PWE, and those newly presenting with suspected seizures will be frequently encountered in any acute medical setting.

Whilst the diagnosis and management of epilepsy should be in specialist hands, ensuring all healthcare professionals in primary and secondary care have a framework to approach patients presenting acutely is in the interests of all, most importantly the patient.

Epilepsy On The Acute Medical Unit
Good Clinical Care

This review will in turn outline an approach to individuals presenting with a transient loss of consciousness (TLoC), new onset suspected seizures and acute symptomatic seizures, seizures in patients with known epilepsy, functional non-epileptic attacks and status epilepticus.

1. Transient loss of consciousness (TLoC)

In an individual presenting with a self-resolving blackout (TLoC), with or without reported motor/convulsive features, the main differential lies between syncope, a seizure or dissociative non-epileptic attacks. As well as being a common clinical dilemma, this is also a scenario easily assessed with simulated patients in medical school and post-graduate exams, so worth being familiar with. As is so commonly the case in neurology, the key step in diagnosis is history, both from the individual and wherever possible supported by an eye-witness account.

Documenting this in detail at the time of initial presentation can be invaluable to the specialist later assessing the patient, as the witness may no longer be available, or memories influenced by the passage of time. In addition to obtaining an open narrative account, specific hypothesis testing questions will usually be needed covering all stages of the event (Table 1). Asking a witness to mime the attack, or offering specific alternative descriptors (e.g. floppy trembling, rigid jerking, flailing, thrashing) is often more informative than relying on spontaneous accounts or lay-interpretations e.g. "like a seizure".

History feature	Examples
Risk factors	Known brain disorder/learning disability; previous meningitis, encephalitis, traumatic brain injury febrile convulsions; cardiac disease; medication and recreational drugs; family history of neurological, cardiovascular disease or sudden unexplained death
Circumstances	Posture/activity prior to and at onset; recent fluid/food/alcohol; temperature, intercurrent illness
Prodrome (+witness)	Lightheaded, sweaty, tinnitus, fading hearing/vision (suggesting syncope); chest pain, palpitations, shortness of breath; focal neurological symptoms (suggesting seizure) – motor, sensory, experiential such as déjà vu/jamais vu, gastric rising;
Event (+witness)	Duration, colour, motor features, incontinence, tongue biting, vocalization/verbalization, injuries sustained; any previous minor/incomplete events (pre-syncopal, unrecognized focal seizures)
Post-event (+witness)	Time to responsiveness, orientation and full recovery; presence or absence of headache, myalgia, confusion, focal neurological symptoms

Table 1: Key historical features in the initial assessment of TLoC / suspected seizures.

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There is no single feature which is absolutely diagnostic: up to 80% of individuals with syncope may have convulsive features, typically a few myoclonic jerks or brief (seconds) posturing; similarly minor focal seizures, absences and myoclonus are overlooked prior to the first presentation with a generalized tonic clonic seizure; any cause of TLoC might have associated incontinence, tongue biting or injury depending on the circumstances at the time.

Even where a witness account is not available, there may be sufficient circumstantial evidence to strongly point towards the likely cause and direct next steps. A useful screening tool based on key features in the history is a questionnaire devised and validated by Sheldon and colleagues (4) (Table 2), which will correctly distinguish between syncope and seizures with a 94% sensitivity and specificity. Amnesia of more than 5 minutes of itself is considered a seizure marker, together with other features scored positively in Table 2.

Feature	Points
Wake with a cut tongue?	2
Déjà vu or jamais vu before?	1
Emotional stress precipitates your spell(s)?	1
Head turning during a spell?	1
Unresponsive, unusual posture, limb movement, or amnesia of spells? (any one of these)	1
Confusion after a spell	1
Lightheaded spells	-2
Sweating before spell	-2
Spell associated with prolonged sitting or standing	-2
If point score is ≥1 the likelihood is seizure; if <1 the likelihood is syncope.	TOTAL

Table 2: Sheldon's (4) questionnaire for patients with TLoC.

If the likely diagnosis isn't already clear, investigations are unlikely to help but everyone should have an ECG to ensure dangerous conduction defects aren't missed (5), and in the acute setting baseline bloods (full blood count, urea & electrolytes, glucose) to exclude major metabolic disturbances or anaemia as triggers are also justified.

For uncomplicated vasovagal syncope (remember the 3Ps: postural, provoked, typical prodromal symptoms), advice and explanation is all that is needed. Anyone with suspected pathological syncope (Box 1) should have an urgent (<24hours) cardiological assessment (5). For those in whom a seizure is suspected, next steps are outlined below.

- **Any ECG abnormality**
- **Heart failure, cardiac murmur or new unexplained shortness of breath**
- **TLoC during exertion, or unprovoked in sitting or lying**
- **Family History of sudden unexplained/cardiac death <40years or hereditable cardiac disease**
- **Age over 65years without prodrome**

Box 1: Features suggestive of pathological syncope.

2. First presentation of seizures.

Anyone with a suspected first seizure should be referred for specialist assessment (2) and ideally seen within 2 weeks. Those with a single self-terminating seizure, with no residual neurological symptoms or signs and normal screening bloods and ECG can usually be discharged with an outpatient referral into the local first-seizure pathway, who will then arrange additional investigations (typically MRI and EEG) as required. In all cases, even if the diagnosis is uncertain, as a minimum individuals must be given basic safety and driving advice (Box 2) pending further assessment.

- **Driving: by law must refrain from driving until specialist assessment. If a seizure is confirmed, the DVLA must be informed and a group 1 license will usually be revoked for a minimum of 6 to 12 months (depending on recurrence, and on the presence/absence of abnormalities on MRI/EEG)*.**
- **Safety & Lifestyle: shower rather than bath; avoid heights/ dangerous equipment; occupational/parenting guidance.**
- **First aid in the event of recurrent events, and who to contact**

***full details at https://www.gov.uk/guidance/current-medical-guidelines-dvla-guidance-for-professionals .**

Box 2: Minimum information to be provided to individuals with suspected seizures, pending specialist assessment.

Specialist assessment will often confirm, but might lift/shorten the initial driving guidance depending on the final conclusions and it is worth emphasising this to patients to encourage attendance. Anti-epileptic medication is not indicated for a single seizure in the absence of factors indicating a high risk of recurrence. Other than in status epilepticus (covered later), commencement should ideally always be a specialist decision.

Around 50% of first seizures in adults will be an isolated/non-recurring event, but some of course will be a first presentation of epilepsy. Suggesting that friends/relatives video any recurrent events will also endear you to local neurology services, and can be invaluable for diagnosis in some cases. Urgent CT brain imaging may be justifiable in those with clear focal features, prolonged or recurrent events to rule out an acute intracranial event as the cause.

Any with abnormal imaging or incomplete recovery, and/or those with suspected acute symptomatic seizures as detailed in the next section may justify a brief admission and more urgent (in-patient) medical and neurological assessment.

3. Acute symptomatic seizures.

An acute symptomatic seizure is defined as a clinical seizure occurring at the time of a systemic insult or in close temporal association with a documented central nervous system or systemic insult (6). These must not be missed, as management primarily depends on identifying and addressing the underlying cause, and the risk of later recurrent seizures is typically much lower than following unprovoked seizures, influencing driving and safety advice (7).

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There are three contexts in which acute symptomatic seizures commonly present:

- *Firstly in patients with comorbidities such as diabetes, heart failure, cerebrovascular disease and cancers, where severe metabolic disturbances such as hypoglycaemia and hyponatraemia are not uncommon, particularly where there is associated polypharmacy.*

- *Secondly in patients presenting with acute primary cerebral pathology such as stroke, subarachnoid haemorrhage, traumatic brain injury, meningitis or encephalitis.*

- *Finally seizures may be the presenting feature of drug or alcohol toxicity or withdrawal.*

The standard definition is the onset of seizures within seven days of the precipitating event, though some flexibility is needed depending on the nature of the insult as detailed in Table 3. With respect to recreational drugs, different categories of risk have also been defined, with for example cocaine, amphetamines and other stimulants, including some of the new "legal highs" being high risk, and heroin and cannabis relatively low risk though this is more controversial (8).

Provoker	Parameter
Cerebrovascular disease/Hypoxia	<1 week
TBI w/o subdural hematoma	<1 week
TBI w subdural hematoma	>1 week depending on entity
Intracranial surgery	<1 week
AVM (only during acute haemorrhage)	<1 week
CNS infections	Till clinical and laboratory findings have normalised
Multiple sclerosis	<1 week (of relapse)
Alcohol withdrawal*	7-48 h from the last drink
Serum glucose (documented within 24 h)	<36 mg/dL (2.0 mM) or > 450 mg/dL (25mM) associated with ketoacidosis
Serum sodium (documented within 24 h)	<115 mg/dL (<5mM)
Serum calcium (documented within 24 h)	<5.0 mg/dL (<1.2mM)
Serum magnesium (documented within 24 h)	<0.8 mg/dL (<0.3 mM)
Urea nitrogen (documented within 24 h)	>100 mg/dL (>35.7 mM)
Creatinine (documented within 24 h)	>10.0 mg/dL (>884 mcg)

Table 3: Proposed parameters for acute symptomatic seizures (8)

*TBI = traumatic brain injury; AVM = arteriovenous malformation; CNS = central nervous system; h = hours; * individual differences in susceptibility/drinking patterns play a substantial role (9).*

There will be clues in the history, or on examination in most instances. A minimum blood screen in addition to those for any TLoC would include liver function, C-reactive protein, calcium, magnesium and phosphate, with a low threshold for considering a toxicology screen.

Almost all such patients will require brain imaging, and in those where no cause has been identified on initial assessment and there is no contraindication, have a low threshold for lumbar puncture (protein, glucose, cell count, microbiology), remembering to measure pressure and enough fluid for additional tests if needed.

Appropriate specialist advice (e.g. endocrinology, neurosurgical, neurological, infectious diseases) should be sought as indicated. If a cause is found, it's easy for the seizure to then be overlooked, so don't forget these patients still benefit from specialist epilepsy advice to ensure full discussion about future recurrence risk, safety and driving is also completed.

For patients with multiple seizures/clusters, especially if the provoker can't be immediately addressed, AEDs will sometimes be prescribed but this should only be short term, typically one to two weeks, with a defined discontinuation date and plan. There is no evidence of long term benefit in acute symptomatic seizures, and definite risk of harm from side effects, particularly in patients who may anyway be unwell/neurologically impaired from their primary insult.

4. Acutely presenting seizure(s) in patient with known epilepsy

This is an extremely common scenario, with multiple medical and/or non-medical triggers e.g. a passer-by, or new carer calling an ambulance, unaware that this is "normal" for that individual. As always, a detailed history is key.

As well as getting a presenting history, asking about the worst, best and usual seizure frequency in that individual will help you decide whether the acute event is out of the ordinary or not for that patient, and thus inform investigation and management. Patients can present with a deterioration in their usual frequency for a range of reasons including:

- *AED related: e.g. poor adherence; changes in formulation/brand; during planned withdrawal/changes; drug interactions*
- *Alcohol and recreational drug use*
- *Intercurrent infections/systemic illnesses*
- *New acute symptomatic seizures, as covered in the previous section*
- *As part of the natural variability which is inherent in some epilepsies.*

Including a serum sample for AED levels in the initially screening bloods can be invaluable, even if at that point you are uncertain what medication the patient is on (you can always phone details through later). If the individual recovers to baseline, a clear cause can be identified and easily rectified, admission can be avoided with a plan for out-patient epilepsy follow up.

Try to resist the temptation for brain imaging unless there is a clear clinical indication, and EEG is rarely useful in this situation. If poor adherence is identified, try to establish why in order to address the root cause (e.g. side effects needing a change in drug/dose; memory problems – would a blister pack help?) Where senior advice or additional corroborative information is needed, contacting the team who know the patient (and their previous epilepsy/treatment history) may be more useful than the local on-call neurology services.

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In addition to addressing the cause, management may involve simple reassurance, dose adjustments to existing medication, or planned changes which can usually be continued as an outpatient. Clobazam (10mg once or twice a day) can be a useful adjunct as "extra protection" for a few days during intercurrent illnesses or patients undergoing rapid AED switches, under specialist guidance.

5. Functional non-epileptic attacks (FNEA)

This is a not uncommon problem which is often challenging to identify and manage for all concerned. Individuals with FNEA may have been erroneously diagnosed, or continue to report a diagnosis of epilepsy and take AEDs, and the undoubted occurrence of both epilepsy and FNEA in some further confounds the situation.

There is no single clinical feature which reliably distinguishes epilepsy from FNEA, though several which are more typical of FNEA, so this should serve as "red flags" to trigger a diagnostic review, shown in Table 4. Contrary to some teaching, whist stereotyped attacks are considered a hallmark of epilepsy, the same is true for most patients with FNEA, though huge variability between attacks should also be considered a red flag. Similarly, FNEA commonly results in physical injury, sometimes serious (10).

Long duration of individual attacks is also suggestive of NEAD. If suspected, urgent specialist review is recommended, often necessitating a brief admission depending on attack frequency and out-patient availability. Video of the attacks, with patient consent, can be diagnostic on specialist review even in the absence of concurrent EEG, and confirming the diagnosis essential to inform management.

Feature (% of patients experiencing)	NEAD %	Epilepsy %
Pelvic thrusting	**7-44**	0-8.3*
Thrashing/flailing movements	18-31	17
Post-ictal symptoms (confusion, obstructed breathing)	13-16	**61-100**
Waxing/waning, pauses	**69**	0-3.7
Asynchronous Limb movements	**46-96**	5-7.4*
Side-Side head/body movements	**36-63**	0-20*
Eye closure**	**34-90**	0-26*
Recall Items during event	**54-88**	-
Ictal crying (tears)	**5-43%**	0

Table 4: Utility of clinical features in distinguishing epilepsy from FNEA. Data from a systematic review of published literature (11) and international expert consensus (12). * can be seen in Frontal Lobe seizures; **A subsequent study reports ictal eye closure has a 51% sensitivity and 96% specificity for FNEA (13). Items bolded are useful discriminators, though all need to be considered in the broader clinical context. –: no figure provided, as this varies depending on the type of epileptic seizure, though is incompatible with e.g. generalized tonic clonic seizures.

In confirmed FNEA, or cases in whom this is strongly suspected, assuming it is considered safe to do so (i.e. the patient doesn't have significant cardiorespiratory or metabolic compromise), avoiding pharmacological intervention is preferable pending specialist advice, together with calm reassurance for the patient and friends/family.

Managing this situation well is as much a challenge of communication skills and managing the expectations of patients and relatives as it is of clinical judgement. There has been considerable progress in understanding and approach to managing patients with functional disorders in recent years, with some excellent internet resources for those interested (www.neurosymptoms.org, www.codestrial.org, www.nonepilepticattacks.info).

6. Status epilepticus.

Status epilepticus (SE) is defined as continuous seizure activity which has failed to self-terminate leading to a risk of neurological damage. There are many different subtypes, broadly categorized into convulsive and non-convulsive (including absence, focal/partial status) types (14).

Convulsive SE (CSE), defined as a generalized tonic clonic seizure lasting more than 5 minutes, or which recurs without recovery following a previous event represents a medical emergency, with significant morbidity and mortality beyond 30 minutes. Most hospitals will have local protocols you should ensure you are familiar with, based on published guidelines (2, 15) see also http://pathways.nice.org.uk/pathways/epilepsy/treating-prolonged-or-repeated-seizures-and-convulsive-status-epilepticus) so will not be detailed here.

All involve initial treatment with benzodiazepines (in adults typically up to 2 doses of 4mg of lorazepam or 10mg midazolam, which may include pre-hospital treatment by carers or paramedics), followed by an AED for on-going seizures. Phenytoin (and it's prodrug fosphenytoin) and phenobarbitone are the only AEDs currently licensed in this situation, and on current evidence speed and adequateness of dosing (20mg/kg for phenytoin, 10-15mg/kg for phenobarbitone) are probably more important than choice of agent. There is increasing interest in newer alternatives such as Valproate, Levetiracetam or Lacosamide but none are licensed for SE, and there is insufficient evidence to recommend any of these over existing treatments, pending the outcome of a large international trial which has just commenced (16). Those with on-going seizures despite this, termed refractory CSE, will need intubation, sedation and intensive care management with guidance from the local neurology team on further management.

In parallel with measures to control the seizures, investigation as to the cause must be instigated. In adults 50% of cases will present de novo (without a prior epilepsy history), so need approaching as acute symptomatic/new onset seizures, and those with known epilepsy approached as in section 4.

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Once seizures have been controlled, and indeed for any inpatient with epilepsy our personal recommendation is that "as required" benzodiazepines should never be prescribed other than as a single stat dose, with clear instructions as to when it should be used (e.g. convulsion lasting >5 minutes) and triggering a medical review. This minimizes the risk of patients being overdosed and definitive treatment/investigation being delayed.

Conclusions

Suspected seizures and epilepsy related issues are common acute presentations, ranging from the very straightforward to extremely complex and challenging. Rates of misdiagnosis remain high, so specialist input is always recommended but can be greatly facilitated by clear structured approach from those on the "front line".

Whilst acute neurology services are undergoing rapid change, and more likely to be directly accessible to acutely presenting patients than in the past, improving skills and knowledge in this area for all healthcare professionals is key to optimizing clinical effectiveness and patient experience.

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GUILLAIN BARRE SYNDROME & RELATED DISORDERS

EJ Pegg, T Majeed

Guillain Barre Syndrome & Related Disorders
Patient Management

Abstract

Guillain Barre Syndrome and related disorders are important differential diagnoses to consider in a patient presenting with acute limb weakness. A clinical case is used to illustrate the point that there can be overlap between the disorders. The discussion focuses on the main signs and symptoms of these conditions and provides an overview of patient management.

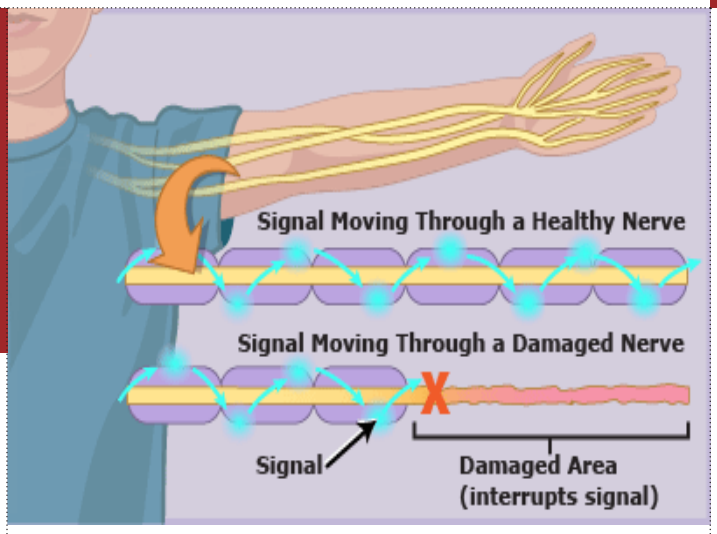
Case history

A 76 year old lady was admitted to the medical assessment unit via the Emergency Department with a 3 day history of progressive unsteadiness, generalised limb weakness diplopia and dysphagia. Swallowing was assessed to be unsafe so she was nil by mouth. On the day of admission she became increasingly short of breath and was found to have severe type 2 respiratory failure necessitating intubation and ventilation. There was no history of preceding diarrhoeal or respiratory illness and there was no relevant past medical history.

On examination she had bilateral ptosis with complete external ophthalmoplegia (i.e. she could not move her eyes in any direction). There was proximal and distal weakness in her upper limbs and proximal lower limb weakness. She was areflexic throughout and plantars were downgoing. Coordination and sensation were normal.

CSF examination revealed a very slightly elevated protein at 0.52 g/L , with a normal cell count, paired glucose and opening pressure. Nerve conduction studies were consistent with the Acute Motor Axonal Neuropathy (AMAN) variant of Guillain Barre Syndrome (GBS). A positive anti GQ 1b antibody was found, which is associated with the Miller Fisher (MFS) variant of GBS.

This patient therefore had an overlap syndrome of the AMAN variant of GBS and MFS. She was treated with intravenous immunoglobulin and slowly began to show signs of recovery. Her recovery has been complicated by the development of pneumonia and atrial fibrillation requiring direct current cardioversion. She is currently being weaned off ventilation and is likely to require a prolonged stay in neuro-rehabilitation.



Discussion

Guillain Barre Syndrome is an acute inflammatory polyradiculopathy which has a reported incidence in a number of countries of around 1/100,000. There is an increase in incidence with age (1).

The classic form of GBS is a demyelinating neuropathy (Acute Inflammatory Demyelinating Polyneuropathy) which causes an ascending, predominantly proximal, and symmetrical weakness with or without cranial nerve involvement. The weakness tends to evolve over days to a week or two, but this is variable. Respiratory muscle involvement occurs in around 25%. Because GBS affects the peripheral nervous system, typical signs include weakness, absent reflexes and hypotonia.

Facial nerve involvement is common and can be bilateral, which can mean that it is overlooked on examination. Tingling parasthesias in the extremities are frequent and are often the first feature. Sensory signs can occur. There may be autonomic involvement causing arrhythmias, fluctuating blood pressure and urinary retention. Many patients report low back pain which may radiate to the buttocks. This is likely to be due to nerve root inflammation.

In up to 90% of patients a preceding history of diarrhoeal illness or respiratory tract infection is found (2). Campylobacter is the most frequent identifiable infection, occurring in up to 30% of patients (3). Other associations include CMV, HIV, EBV and other bacterial infections such as mycoplasma.

Our patient had an axonal form of GBS which is known as AMAN (Acute Motor Axonal Neuropathy). A variant affecting the sensory nerves also exists- AMSAN (Acute Motor and Sensory Axonal Neuropathy). The distinction between demyelinating forms and axonal forms of neuropathies is confirmed with nerve conduction studies.

GUILLAIN BARRE SYNDROME & RELATED DISORDERS

EJ Pegg, T Majeed

Miller Fisher Syndrome is a triad of ophthalmoplegia, ataxia and areflexia. In the isolated form there is no limb weakness however overlap with Guillain Barre Syndrome can occur, as was the case with our patient.

Other forms of GBS also exist (see table 1) and frequently overlap therefore they are thought to be part of the same spectrum of illness and probably share a common autoimmune mechanism.

Bickerstaff's brainstem encephalitis shares the same features as Miller Fisher Syndrome but in addition there is impairment of consciousness.

Pharyngeal-cranial-brachial variant GBS presents with bulbar, cervical and upper limb weakness.

Syndrome	Main Clinical Features
Guillain Barre Syndrome (AIDP/AMAN/ AMSAN)	Ascending proximal limb weakness +/- cranial nerve palsies, autonomic features, respiratory muscle weakness
Phayngeal-cranial-brachial variant GBS	Bulbar, cervical and upper limb weakness
Miller Fisher Syndrome	Ophthalmoplegia, areflexia, ataxia
Bickerstaff's brainstem encephalitis	Ophthalmoplegia, areflexia, ataxia, impaired consciousness
Paraparetic GBS	Predominant paraparesis
Bilateral facial weakness with distal parasthesias	Bilateral facial weakness, distal parasthesias

Table 1: GBS and related syndromes.

Differential Diagnosis

GBS is the most common cause of acute flaccid paralysis in the UK and the clinical diagnosis is usually straightforward but it is important to consider differential diagnoses, guided by the history and clinical findings. (See Table 2)

Differential	Clues to differentiating from GBS
Acute spinal cord injury eg trauma, disc, thrombosis	History of trauma, rapid/sudden onset, prominent sphincter disturbance, sensory level.
Early transverse myelitis	Sensory level, prominent sphincter disturbance,
Viruses against anterior horn cells eg polio, West Nile Virus	Travel history, systemic symptoms, raised CSF white cells
Neuromuscular junction disorders eg myasthenia, botulism	Fatigueability and normal reflexes, (myasthenia gravis), dilated + poorly reactive pupils (Botulism)
Other acute peripheral neuropathies eg Tick paralysis, Lyme disease, toxins/poisons	Travel history, history of bites or toxin consumption
Critical illness neuropathy or myopathy	Recent history of critical illness eg ITU admission
Low serum potassium	Reflexes present, Low K+ (eg due to GI/ urinary loss or rarely familial hypopkalemic periodic paralysis)

Table 2: The differential diagnosis of acute flaccid paralysis.

Investigation of suspected GBS and related syndromes

- **Blood tests: FBC, U+Es, LFTs, inflammatory markers, consider campylobacter serology**

Routine tests should be sent to look for evidence of infection and to ensure serum potassium is normal. (Low K+ is a cause of acute neuromuscular weakness).

- **CSF**

CSF examination is important to exclude other conditions and to support the diagnosis of GBS. The key finding is a normal white cell count with a raised protein (known as cytoalbuminaemic dissociation). It may take a few days for the protein to rise so a lumbar puncture may need to be repeated if the diagnosis is unclear. Glucose and opening pressure should be normal.

- **NCS/EMG**

Electrophysiology confirms the diagnosis and distinguishes between axonal (sensory/motor or both) and demyelinating forms of GBS. This can help guide prognosis.

- **Antiglycolipid antibodies (also known as antiganglioside antibodies)**

This test is sometimes sent by neurologists. These are a group of antibodies which are associated with some of the GBS related syndromes eg the anti GQ1b antibody in Miller Fisher Syndrome. Currently there is no known antibody associated with the acute inflammatory demyelinating polyneuropathy form of GBS (the most common type of GBS).

Management

· Forced Vital Capacity (FVC) should be measured at least 4 hourly to detect respiratory muscle involvement. This is one of the most important aspects of the management of patients with GBS. NB- this is NOT the same as a peak flow. If the FVC is less than 1.5 L or it is falling, an urgent ITU opinion should be sought for consideration of mechanical ventilation and a blood gas should be performed. (Note- Dyspnoea, falling oxygen saturations and rising carbon dioxide levels are a late sign therefore you should not be reassured if normal).

· Thromboprophylaxis. Patients with GBS are at risk of venous thromboembolism.

· Monitor for autonomic instability. Autonomic involvement can cause arrhythmias (as with our patient) therefore it is vital to perform a baseline ECG and if any cardiac symptoms arise. Hypo and hypertension may need to be managed with intravenous fluids and short acting antihypertensive agents respectively.

· Intravenous immunoglobulin therapy or plasma exchange may be advised by a neurologist if there is significant limb weakness or respiratory involvement.

Prognosis

The mortality rate of GBS is up to 5%. Causes of death include cardiac arrest, autonomic instability, respiratory failure, sepsis and pulmonary embolism. Around 10% are left with significant disability and 5-10% develop relapses or a chronic form (Chronic Inflammatory Demyelinating Polyneuropathy). In those who make a recovery, this usually occurs over weeks to months but can be up to 2 years.

GUILLAIN BARRE SYNDROME & RELATED DISORDERS

EJ Pegg, T Majeed

Test Yourself

1. Which of the following is the most frequently identified antecedent infection?

a) Mycoplasma pneumoniae
b) Campylobacter jejuni
c) Cytomegalovirus

2. What are the features of isolated Miller Fisher Syndrome:

a) Areflexia, ataxia, opthalmoplegia, limb weakness
b) Areflexia, ataxia, ophthalmoplegia, reduced consciousness
c) Areflexia, ataxia, ophthalmoplegia

3. Which of the following CSF results is consistent with GBS and related syndromes?

a) Opening pressure 12cm H20, protein 0.3g/L, white cell count 2, red cell count 5, glucose 3.3 mmol/L (venous glucose 4.5)
b) Opening pressure 28cmH20, protein 1.3g/L, white cell count 120, red cell count 2, glucose 2.0 mmol/L, (venous glucose 6.5)
c) Opening pressure 15cm H20, protein 1.1g/L, white cell count 3, red cell count 3, glucose 4.0mmol/L (venous glucose 5.0)

4) How frequently should the FVC be monitored in GBS and related conditions?

a) Every 4 hours
b) Once daily
c) There is no need to do this if the oxygen saturations are normal and the patient is not short of breath

5) Which of the following statements is correct regarding prognosis?

a) 1% of people are left with a significant disability
b) Recovery can take up to 6 months
c) Mortality is up to 5%

Answers

1. Answer = b

Campylobacter is the most frequently identified antecedent infection but other bacteria and viruses are also associated with GBS including mycoplasma and CMV. In some people there is no history of preceding infective symptoms.

2. Answer = c

Miller Fisher Syndrome is a triad of areflexia, ataxia and ophthalmoplegia. Where there is also a reduced level of consciousness, the diagnosis is Bickerstaff's encephalitis. A co-existent limb weakness would be consistent with an overlap of GBS with Miller Fisher Syndrome.

3. Answer = c

In GBS there is a raised CSF protein but the white cell count and other parameters should be normal. Answer a is a normal CSF picture which may found in early GBS within the first few days. Answer b is consistent with bacterial meningitis.

4. Answer = a

This is extremely important. Around 25% of patients develop respiratory muscle weakness and monitoring the FVC is crucial in order to pick this up early.

5. Answer = c

Mortality of GBS is up to 5%. Recovery is usually over months but can take up to 2 years. 5-10% of people are left with a significant disability.

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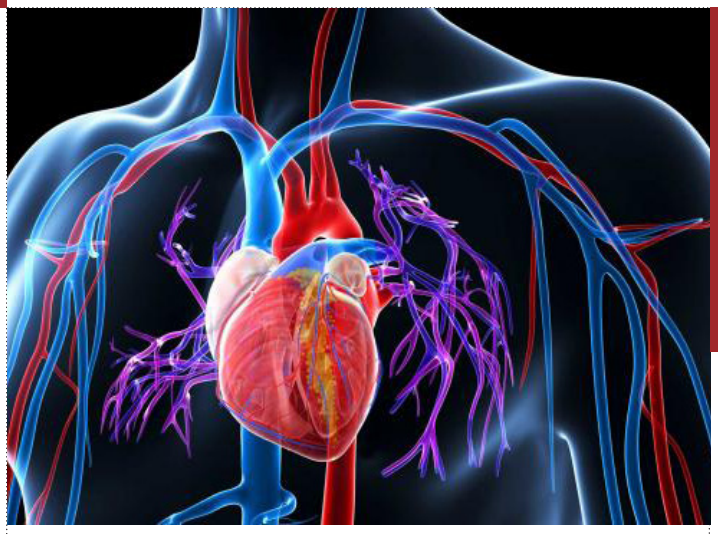
REBOUND INTRACRANIAL HYPERTENSION AFTER SPONTANEOUS CSF LEAK SECONDARY TO TRANSDURAL THORACIC OSTEOPHYTE

M Hussain, M Masood Hussain, R Nimeri, F Ahmed



Rebound Intracranial Hypertension After Spontaneous CSF Leak Secondary To Transdural Thoracic Osteophyte
Patient Management

Abstract

Spontaneous spinal CSF leak presenting with syndrome of low pressure is often difficult to diagnose. It has varied pathological etiology and different options reported to be effective for management. Thoracic osteophyte as a cause is reported rarely in literature. We are reporting a case of spontaneous CSF leak secondary to thoracic osteophyte. Contrary to other similar case reports, it was managed conservatively with resolution of spinal collection and subsequently developed rebound intracranial hypertension which required surgical intervention.

Keywords: *Intracranial Hypotension, Intracranial Hypertension, Thoracic osteophytes, spontaneous CSF Leak, Headaches.*

Introduction

Spontaneous spinal CSF leak causing intracranial hypotension and syndrome of low-pressure headache is a well-known entity frequently under diagnosed. Moreover - causation and localization of leak often remains obscure even with surgical exploration. There is lack of uniformly agreed guideline for diagnosis and treatment. Different etiologies and treatment options have been reported in literature.

We report a rare case of CSF leak from spontaneous perforation of the thoracic dura by a sharp osteophyte in a young patient. Literature review found 2 similar case reports for similar pathology at thoracic spine level. None of the similar cases sequentially found to develop intracranial hypertension and overall only one small case series found reporting phenomenon of rebound hypertension after treatment of spinal intracranial hypotension.

Case report

A 22 year old man was referred to neurosurgical service with a three day history of severe headaches associated with nausea and confusion. The patient reported several similar episodes of headaches but with reduced intensity and duration that started about five-year ago. These were accompanied by intermittent back pain, sometimes nausea and progressive worsening. The symptoms were initially related to physical activity (playing rugby, skateboarding or having sex) and the duration varied between a few hours to a few days.

The brain CT scan on admission did not show any obvious cause of the headache but the lumbar puncture was positive for xanthochromia. With a provisional diagnosis of subarachnoid haemorrhage, he had an angiogram of the cerebral vessels but this did not show any vascular abnormality. The patient had an MRI scan of the cervical and thoracic spine to rule out any vascular abnormality at these levels. This showed an abnormal fluid collection at the mid thoracic level which was extra-dural and antero-lateral on the right side.

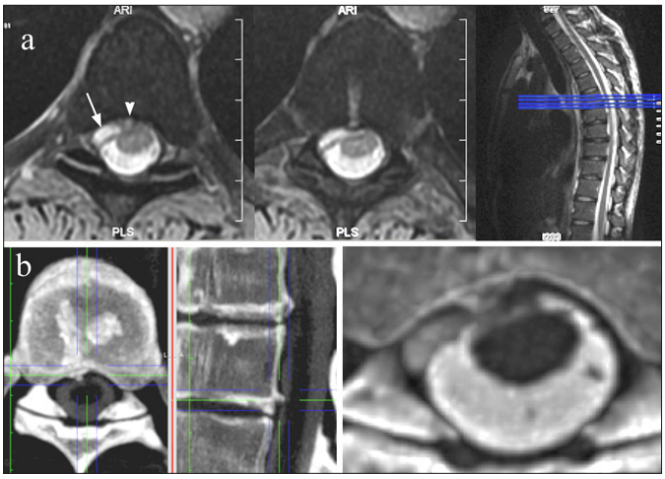


Figure 1

REBOUND INTRACRANIAL HYPERTENSION AFTER SPONTANEOUS CSF LEAK SECONDARY TO TRANSDURAL THORACIC OSTEOPHYTE

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It was consistent with CSF. The CT myelogram of the thoracic spine and further 3-D reconstruction of the spine showed sharp osteophytes coming from the body of the thoracic vertebrae and impinging on the dura. This was closely related to the extra dural CSF accumulation around this area both on MRI and CT Myelogram.

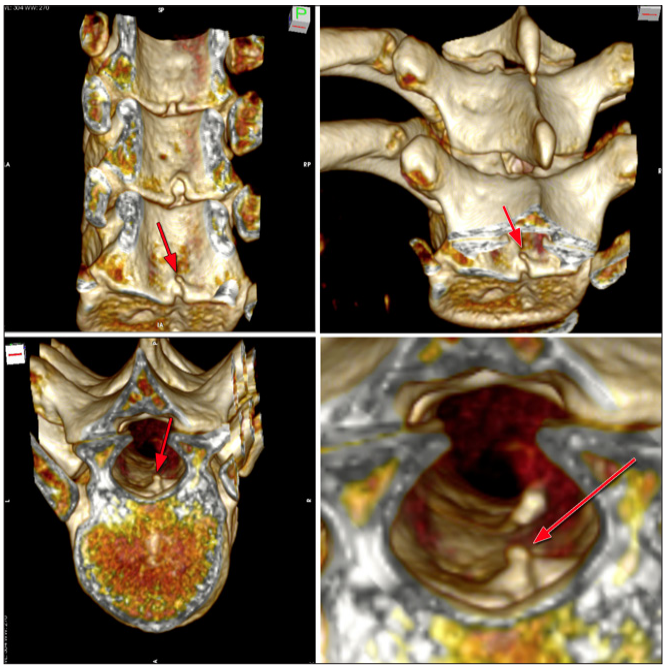

Figure 2

Different management options were considered for the treatment. The most logical approach was the removal of the causative element (osteophyte) and re-suturing of the fistula, however this was technically challenging and was considered to be associated with high surgical risks because of particular location in front of the thoracic spinal cord.

Looking at the complexity of the case, potential surgical risks and the paucity of treatment options in literature, the case was discussed at the multidisciplinary meeting followed by discussion with patient and his family and was decided to treat conservatively. It was advised to avoid activity that may increase intracranial pressure and precipitate CSF leak.

A review after 10 months of initial diagnosis, he complained of worsening headache. His symptomatology remained unchanged for that period until he started to develop continuous headache suggestive of raised intracranial pressure with double vision and found to have florid papilloedema. He underwent repeat imaging of the whole neural axis. MRI of spine showed dramatic decrease in spinal collection.

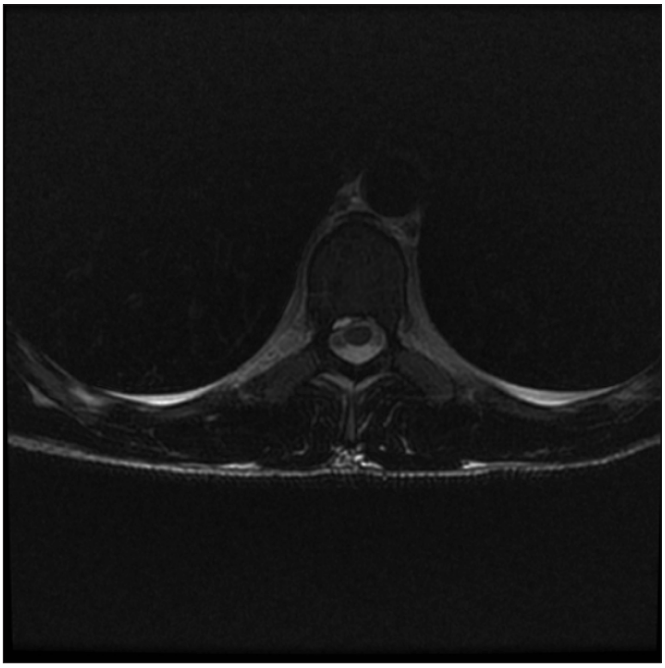

Figure 3

MRI of brain didn't show anything significant apart from hemosiderin deposit in mid cerebellar vermis. He underwent 2 attempts of lumbar drainage (with CSF opening pressure of 40 and 34cm H_2O) with transient relief and finally cured by LP shunt. There was no recurrence of headaches on follow up after 3 months.

Discussion

Clinical features

Spontaneous spinal cerebrospinal fluid leak causing intracranial hypotension (SIH) presenting with headache and a variety of clinical symptoms has emerged as a well-known entity. Other symptoms include neck stiffness, nausea, vomiting, vertigo, tinnitus, deafness, cognitive abnormalities and radicular arm pain.

The clinical picture can sometimes mimic frontotemporal dementia, and the behavior of some patients can sometimes be described as hypoactive-hypoalert, with somnolence, impaired attention, and stereotyped motor activity. Sagging of the brain, caused by leakage of the CSF, can cause lesions in the brainstem with stupor, gaze palsies, and cranial nerve palsies (5,16). It is estimated to have an incidence of 5/100,000 per year, with a peak around 40 years. Women are affected more commonly than men (2,15).

REBOUND INTRACRANIAL HYPERTENSION AFTER SPONTANEOUS CSF LEAK SECONDARY TO TRANSDURAL THORACIC OSTEOPHYTE

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Causes

Causes of intracranial hypotension can be classified as 1) spontaneous (primary), and 2) secondary. Most commonly, a small tear or defect in the spinal dural sac is the underlying lesion that results in a CSF leakage and intracranial hypotension. Mechanical stress may have a role in the pathogenesis of SIH, and meningeal diverticula and connective tissue disorders may be important risk factors. Secondary causes of intracranial hypotension include spinal trauma, iatrogenic causes and occasionally degenerative spine disorders also implicated at instances (8).

The location of the spinal CSF leak remains undetectable in approximately 50% of cases reported. Most of the CSF leakage sites were detected at the cervical or thoracic level of the spine (19). So far only 2 cases of thoracic osteophyte as a cause of CSF leak are reported in the literature (1,23). Other similar situation were also reported occasionally where mechanical lesions like thoracic disc was the cause in 2 cases and calcified meningioma was the cause in one case (13,20,22). Similarly few cases of spontaneous CFS leak with cervical level spur at C4/5 and C5/6 level and another single case of lumbar disc also found (3,9,19).

Diagnosis

Imaging modalities include MRI with myelography, 3-D CT myelography and radionuclide cisternography (7,15). Typical imaging findings visible on MRI head include diffuse pachymeningeal enhancement, descent of the cerebellar tonsil, brain stem sagging, enlargement of the pituitary gland and subdural fluid collection. Spinal MR imaging is helpful in showing characteristic features of SIH like distention of the epidural veins, epidural fluid collection on fat-saturated T2-weighted images and abnormal visualization of the nerve root sleeve (21). CT myelography has the superiority in highlighting bony lesions (3).

Opening pressure is often low, and examination of CSF may reveal pleocytosis, an elevated protein, and xanthochromia probably due to increased permeability of dilated meningeal blood vessels and decreased CSF flow in the lumbar subarachnoid space (15). Diagnostic criterion proposed by Schievink et al includes criterion A, the demonstration of extrathecal CSF on spinal imaging.

If criterion A is not met, criterion B, which is cranial MR imaging findings of SIH followed with at least one of the following:

1) low opening pressure

2) spinal meningeal diverticulum

3) improvement of symptoms after epidural blood patch

If criteria A and B are not met, there is criterion C, the presence of all of the following or at least 2 of the following if typical orthostatic headaches are present:

1) low opening pressure

2) spinal meningeal diverticulum

3) improvement of symptoms after epidural blood patch (16)

The symptoms described by the patient in this case report were suggestive of low ICP (incapacitating headaches, nausea, confusion) caused by CSF leak. Review of the images clearly suggested CSF leak at the thoracic level in the vicinity of a sharp osteophyte coming from the vertebral body into spinal canal. It was believed that this perforated the dura and caused the CSF fistula. This was precipitated by the sporting activities and the physical impact which this involved (rugby). The headaches were intermittent probably because the fistula was occluded by the osteophyte/spur which was working as one way valve.

Management

Most patients respond well to conservative measure and minor invasive procedures (8). The initial treatment of choice is a lumbar epidural blood patch, regardless of the location of the CSF leak. If the epidural blood patch fails the procedure can be repeated at the lumbar level, or can be directed at the exact site of the leak. Franzini et al proposed treating SIH with blood patch procedure at lumbar level regardless of the site of leak on the basis that dural leak is not the primary reason for SIH.

According to them the dural leak is effect of the epidural hypotension maintained by the inferior vena cava outflow to the heart. The goal of their blood patch procedure is not to seal CSF leak, but instead to help in reversing the CSF-blood gradient within the epidural space along the entire cord (4). Percutaneous placement of fibrin glue under CT guidance is proposed as an alternative in patients who failed conservative treatment and blood patch (5,18).

In cases where symptoms are severe and refractory to less invasive measures, surgical intervention is indicated. Tears in dura or leaking diverticulum that are identified as the source of CSF leak often can be ligated or repaired. When a source of CSF egress is not found intraoperatively, packing the epidural space with blood soaked gelfoam or muscle at the appropriate site is found to bring relief of symptoms (10).

Reported cases of thoracic osteophyte as a cause of CSF leak were treated diversely. One of them was treated with surgical intervention involving sternotomy and thoracotomy for thoracic level spur (1) while other one was treated with epidural blood patch, without targeting the offending osteophyte and achieving a good outcome (23).

REBOUND INTRACRANIAL HYPERTENSION AFTER SPONTANEOUS CSF LEAK SECONDARY TO TRANSDURAL THORACIC OSTEOPHYTE

M Hussain, M Masood Hussain, R Nimeri, F Ahmed

Likewise cases where thoracic disc (2 cases) was the reason for SIH were treated successfully with epidural blood patch (13,22) while case of calcified meningioma with neurological deficit was treated surgically (14). Cases of spontaneous CFS leak with a cervical spur and with lumbar disc were expected to be less challenging surgically, but outcome of cervical cases in 1 small series were complicated and results were not encouraging (3,9,20).

Learning from the case

It is not unknown to have intracranial hypertension after the treatment of spontaneous CSF leaks and intracranial hypotension. Mokri B reported 4 of his cases developing intracranial hypertension and all were treated conservatively. In contrast the case reported here required CSF diversion procedure (11).

PG Kranz has reported case series of 9 patients who developed rebound intracranial hypertension after treatment with blood patch. All of them were treated conservatively (12). This case is also unique where after suffering from long standing intra cranial hypotension, patient had complete reversal of pathology, so much so that patient required intervention. Insertion of LP shunt resolved all symptoms including papilledema & at one year follow up patient remained completely well.

Conclusion

The case report emphasizes the importance of assessing every case based on symptomatology. Intracranial hypertension following intracranial hypotension is extremely rare and may be missed if the symptomatology and clinical examination is not careful. Both conditions are potentially treatable and conservative approach must be undertaken before resorting to surgical treatment.

Test yourself

1. Most common symptom of intra cranial hypotension?

a) New headache that occurs shortly after assuming upright posture and relieves by lying down.
b) Sudden onset worst ever headache.
c) Headache waking up in the middle of night.

2. The best investigation for accurately defining the site and extent of leak is:

a) Magnetic resonant imaging
b) Computed tomography
c) Myelography

3. Typically CSF opening pressure should be less then following to diagnose intracranial hypotension:

a) 10 cm H_2O
b) 6 cm H_2O
c) 20 cm $H2_2O$

4. Mainstay of treatment of intracranial hypotension is:

a) Put patient in trendelenburg position
b) Autologus blood patch
c) Acetazolamide

5. Typically MRI finding if intracranial hypotension are except:

a) Enhancement of pachymeninges
b) Engorgement of venous structures
c) Lobar bleed

Answers

1. An orthostatic headache is the prototypical manifestation but other headache patterns occur as well, and associated symptoms are common.

Diagnostic Criteria for Headache Due to Spontaneous Spinal CSF Leak and Intracranial Hypotension According to the International Classification of Headache Disorders, 2nd Ed.

• Diffuse and/or dull headache that worsens within 15 minutes after sitting or standing, fulfilling criterion D and with ≥1 of the following:

Neck stiffness
Tinnitus
Hypacusia
Photophobia
Nausea

• At least 1 of the following:

Evidence of low CSF pressure on MRI (eg, pachymeningeal enhancement). Evidence of CSF leakage on conventional myelography, CT myelography, or cisternography. CSF opening pressure <60 mm H_2O in sitting position.

• No history of dural puncture or other cause of CSF fistula.

• Headache resolves within 72 hours after epidural blood patching.

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; MRI, magnetic resonance imaging.

REBOUND INTRACRANIAL HYPERTENSION AFTER SPONTANEOUS CSF LEAK SECONDARY TO TRANSDURAL THORACIC OSTEOPHYTE

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2. Myelography with iodinated contrast followed by thin-cut computed tomography of the entire spine (or with gadolinium followed by MRI) has been shown to be the study of choice to accurately define the location and extent of leak.

3. CSF opening pressure is typically less than 6cm H_2O and can be unmeasureable and negative. Some patient even can have normal pressure.

4. Trendelenburg position is for hypotensive shock and acetazolamide treatment of intracranial hypertension. Autologus blood patch is still main stay of treatment.

Typical magnetic resonance imaging findings include subdural fluid collections, enhancement of the pachymeninges, engorgement of venous structures, pituitary hyperemia, and sagging of the brain (mnemonic: SEEPS).

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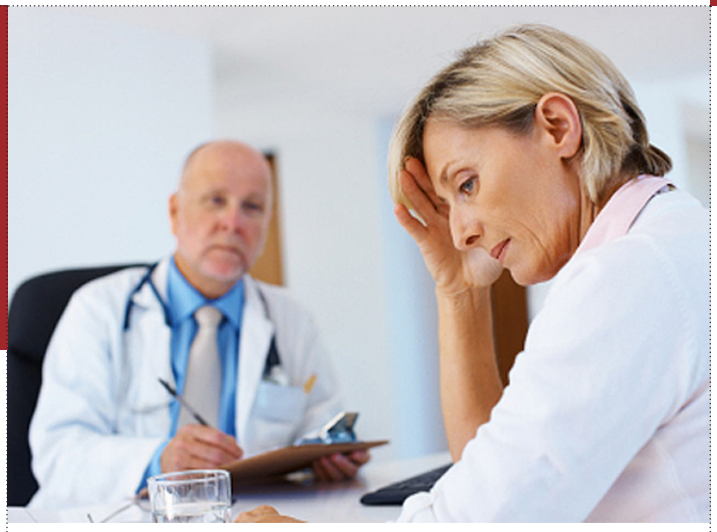
CASE-BASED DISCUSSION: THE CONFUSED PATIENT

SM Bell, DJ Blackburn, R Ghosh, K Harkness

Case-Based Discussion:
The Confused Patient
Patient Management



Abstract

Confusion is a common cause for acute hospital admissions and a systematic approach to assessment is essential. Having an ability to differentiate between delirium or dementia is needed so appropriate treatment can be initiated and safe care planning put in place. Assessment of a patient's capacity often forms part of a confusion assessment. Here we detail how to use the mental capacity act, how to perform a Deprivation of Liberty (DOLS) order and how to use antipsychotics in this situation.

Case History

You review an 84 year old gentleman admitted because his family are concerned about his behaviour. For the last few days he has been very confused. His past history includes hypertension, a small stroke, and benign prostatic hypertrophy. He currently takes aspirin, bendroflumethiazide, amlodipine, Adcal, and finasteride.

When you arrive the patient is trying to leave. The nursing staff and his family have tried to reason with him, but he has hit them with his stick. He is demanding to be let home.

You assess him and feel his capacity to make a decision about leaving the hospital is compromised. You try and reason with him but his aggression continues. You are concerned that this recent decline may be due to a drug side effect or underlying infection.

You feel a Deprivation of Liberty (DOLS) (1) assessment should be performed which you discuss with your consultant who agrees.

The patient will not allow you to treat them. You feel they are at risk of harming staff and themselves. All non-pharmacological methods to calm the patient have failed. You decide to give them an intra-muscular injection of haloperidol to try and calm them down. After administering the injection you examine him. He has mild weakness in his right hand from his previous stroke. His early warning score is 0, urine dipstick is negative for nitrites, and his Abbreviated Mental Test (AMT) (2) score is 6/10.

His delirium screen shows a normal renal function, full blood count, haematinics and thyroid function. Sodium is 124, CRP is <0.3, and calcium is 2.75. Mid-stream urine culture is negative. CT head scan shows an old small left basal ganglia lacunar infarct and mild small vessel disease. You stop his Adcal and bendroflumethiazide, as you think these are causing his biochemical abnormalities.

Over the next few days his confusion improves. A MOCA test (Montreal Cognitive Assessment) (3) reveals a score of 22 (Normal cut off >26/30). Mood screening with the PHQ-9 does not show any significant depression. He is assessed by the occupational and physiotherapists and deemed safe to live at home with short term additional daily carer visits. He regains capacity and consents to home support. He is discharged from hospital with a NOMAD and with follow-up in both memory and general medical clinics.

Discussion

What causes confusion?

Acute confusion is a very common reason for admission to hospital and has many causes including dementia, delirium and dysphasia. Often these conditions may co-exist and be overlapping. At any time an estimated 20-30% of acute hospital beds are occupied with patients with dementia and 15% of admissions are affected with a delirium (5). Patients with delirium have higher rates of morbidity and mortality. One third of patients will make a rapid recovery but a fifth will not return to their pre morbid state.

How do we identify confusion?

Tools used to identify delirium include the Cognitive assessment method (CAM) (6) and The Single Question in Delirium (SQID) (7). The SQID has been shown to be more sensitive than CAM in diagnosing delirium and is a simple tool to use (see figure 1).

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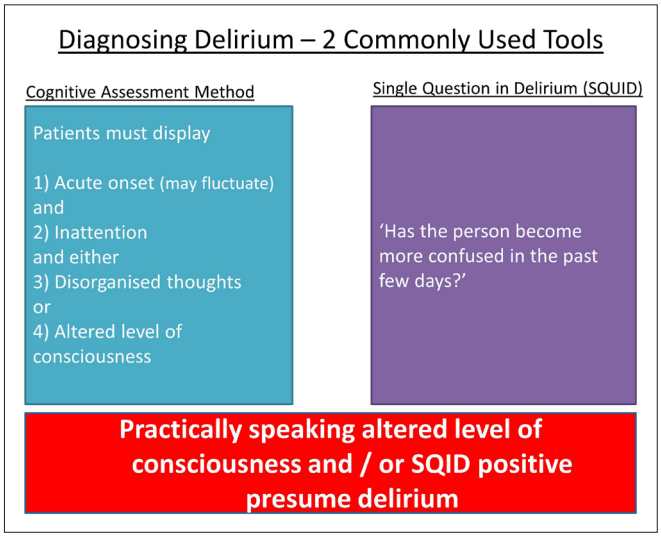

Figure 1: Delirium diagnostic tools: This figure highlights the questions used to diagnose delirium. Both methods have been validated, but the SQID is thought to diagnose delirium better in ward patients. The CAM was developed for use in Intensive Care.

A systematic approach to assessment includes a detailed collateral history of duration of symptoms and examination to look for focal neurological signs. Evidence of infection or hypoxia and relevant investigation to correctly diagnose and treat the patient's confusional state should be sought (see figure 2).

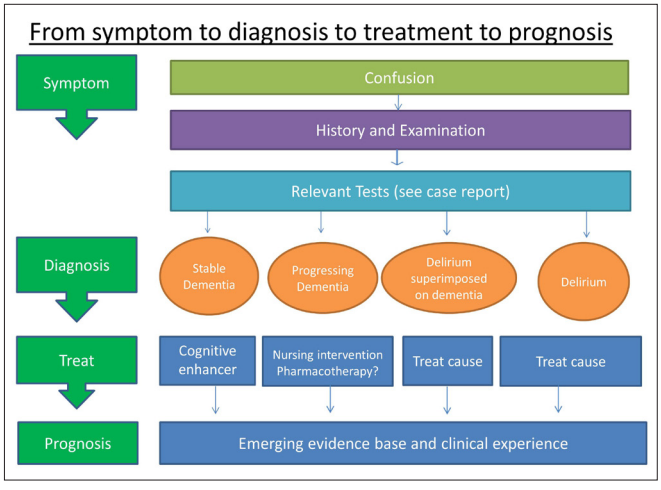

Figure 2: Cognitive testing proforma: This diagram shows the steps to go through when trying to diagnose a person's cause for confusion.

Managing Confusion

The behavioural component to managing confusion is often complicated and while we wait for the resolution of the cause of confusion, other management plans may be needed. Always, when treating confusion, we should try to use non-pharmacological methods first (8). Table 1 highlights these methods.

Non-Pharmacological Interventions for Confusion
High quality ward and nursing environment
Ensuring patient is not in pain
Nursing performed by same nurses where possible
Use of low stimulus environments, such as side rooms
Appropriate light levels in the persons room

Table 1: Non Pharmacological Interventions for Confusion. These methods should be considered before pharmalogical intervention is used.

It may be that a person's confusion is so bad that we are concerned it has affected their ability to make appropriate decisions. As a result of this we have to assess the patient's capacity.

Capacity in a patient is assessed in accordance with the guidance given in the mental capacity act 2005 (9). This is a 2 part process which first requires the doctor to decide if the patient has a condition, such as delirium or dementia might affect their capacity. Once this has been established, it then must be assessed if patient can understand the points in table 2 with regards to a decision they need to make;

If the patient cannot do the above then they are deemed to lack capacity and can be treated under "best interests". As part of the capacity assessment a Deprivation of Liberty Safeguards (DOLS) assessment needs to be performed to ensure a patient's liberty has not been inappropriately restricted. The senior nurse on the ward will initiate the application for DOLS as this is a legal requirement.

Assessing Capacity
Understand the relevant information given to them
Retain the information long enough to make the decision
Weigh up the information
Communicate their decision to you

Table 2: Assessing Capacity: These are the elements required in a capacity assessment.

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Use of Antipsychotics

If a patient's behaviour is potentially placing them or others at risk NICE guidance advocates short term (one week or less) use of antipsychotics (10). Olanzapine and haloperidol are the drugs of choice, and should be started at the lowest clinically appropriate dose with cautious titration. Neither medication has UK marketing authorisation. For this reason therefore clear documentation of use and consultation with family members needs to be documented. Benzodiazepines may be considered if antipsychotics are ontraindicated, such as in Lewy Body dementia, but are not recommended in NICE guidance.

Summary

Confusion is a very common reason for admission to medicine, especially in the elderly. Dementia and delirium are important causes of confusion. Using a systematic approach to assessment of the confused patient will help with identifying the cause of their confusion. Patients with confusion may need to be treated against their will. This should only be a step in the management of delirium if all other avenues for treatment have been explored.

MCQ's

1. How many people in the UK have dementia?

A) 100,000
B) 700,000
C) 850,000
D) 1,000,000
E) 1,200,000

2. Which of the following assessment tools is most appropriate when assessing cognition in vascular dementia?

A) Mini-mental State Examination (MMSE)
B) Addenbrooke's Cognitive Examination (ACE-R)
C) Abbreviated Mental Test (AMT)
D) Montreal Cognitive Assessment (MOCA)
E) The Brief Memory and Executive Test (B-MET)

3. Which of the following points in regard to the mental capacity act is true?

A) The act that we currently refer to was created in 2000
B) Patients should be encouraged to participate in the decision about their capacity
C) It does not apply to people with underlying mental health problems
D) Once an assessment of capacity has been made on a particular issue further review is not needed
E) A person is assumed to have capacity unless it is established that they lack capacity

4. Which of the following points are true with regard to a DoLS assessment?

A) Confirming a patient's age is part of the assessment
B) It is used as a way of confirming that a person does not have an inappropriate restriction in their freedom
C) The DOLS 'best interests' assessment must be completed by a doctor
D) The DOLS assessment is regularly reviewed and can last for 12 months once authorised
E) The assessment is used when a patient is detained under the mental capacity act 1983

5. Which of the following points about antipsychotic use in dementia are true?

A) 20-30% of people in nursing homes with dementia are on an antipsychotic
B) 1% of people over 65 are prescribed an antipsychotic
C) There is an increased risk of stroke and cardiovascular disease with use
D) They have been shown to have excellent long term effects in managing psychosis in dementia
E) They are associated with a higher incidence of death

Answers

1. C is correct.

Roughly 850,000 people have AD in the UK; this number is set to double over the next 20 years.

2. E

Is the only test that has been devised to be used in Vascular Cognitive impairment. A, B and D will all have some use in monitoring the progress of vascular dementia, but they mainly assess cortical dementias whereas the B-MET assesses subcortical dementias such as Vascular Dementia.

3. Both E and C are the appropriate answers in this case.

We currently use the mental capacity act that was revised in 2005. The act applies to all people whether they have mental health problems or not. Once a person has been deemed to not have capacity the decision does need to be reviewed at a regular basis.

If a delirium is causing a patients lack of capacity then this may resolve and the patient may be able to make decisions about the own care again. Patients should always be involved when possible in the assessment of their capacity and we assume all people have capacity until we can prove otherwise.

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4. A,B and D are the correct answers.

A Deprivation of Liberty Safeguards (DoLS) assessment is performed when a person is deemed not to have capacity under the mental capacity act. It is a method for preventing the inappropriate deprivation of a person's liberty. People have to be at least 18 years of age. The assessment should be performed within 21 days once an application has been received by an assessment authority, and is not to be used when a patients is detained under the mental health act.

The assessment is performed by 2 people. Many types of health care professionals can perform a DOLS assessment as the 'best interests' assessor' as long as they have the appropriate training and experience. The second assessor the 'Mental health assessor' must be a doctor who is able to assess if a person is suffering from a mental health disorder. The assessment should be reviewed regularly, but can be used for up to 12 months.

5. A,C and E are correct.

20-30% of nursing home residents are on at least one antipsychotic. 5.3% of the over 65 population that use the NHS have an antipsychotic drug prescribed. There is an increased risk of both cardiovascular and stroke risk with antipsychotics, and there is no good evidence that they help in the long term management of confusion in patients with dementia.

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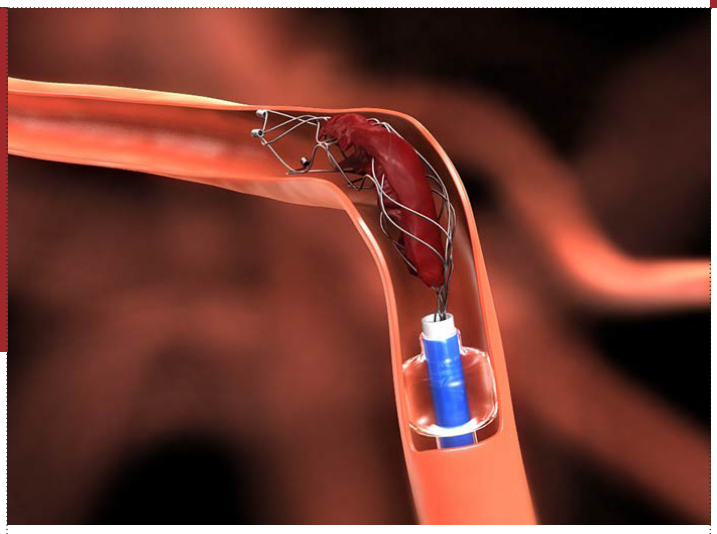
UPDATE ON ACUTE STROKE TREATMENT: THROMBECTOMY

CA Maduakor, B Patel, AC Pereira



Update on Acute Stroke Treatment: Thrombectomy
Patient Management

Abstract

Acute ischaemic stroke is a devastating condition and a medical emergency. A blood clot blocks an artery perfusing a region of the brain leading to immediate loss of function such as paralysis down the opposite side of the body. Untreated, patients may suffer severe permanent disability and even death. Thrombolysis with intravenous recombinant tissue plasminogen activator (rt-PA), which acts to dissolve the clot, has been the mainstay of hyperacute stroke treatment. Given within 4.5 hours of symptoms starting, 1 in 8 patients will be less disabled.

However, patients with very large clots do not benefit from thrombolysis alone as the clot is too big for safe doses of rt-PA to dissolve. 2015 has seen an explosion of evidence which shows intra-arterial clot retrieval by an interventional neuroradiologist benefits 1 in 4 patients with a large clot. Here we explain the rationale behind treatment and the evidence supporting both thrombolysis and thrombectomy.

We explain the process of how to take a patient through a standard hyperacute pathway for stroke care. By the end of this article, the Foundation Year doctor should be able to diagnose major stroke confidently, know how to prepare patients for thrombolysis and assess the need for thrombectomy. Clearly, this type of patient will need a multi-disciplinary approach but a Foundation Year doctor could easily be the first person who sees this type of patient and therefore, could be pivotal in the initial management and bringing the multi-disciplinary team together.

Introduction

Stroke is a very important, acute condition for Foundation Years doctors to understand. According to the Stroke Association's published statistics, there are about 152,000 strokes a year in the UK; that is one every 3 minutes 27 seconds. 12% are fatal within the first 30 days and 25% within a year. There are about 1.2 million stroke survivors in the UK of which over a third are dependent on others and of those, 1 in 5 are cared for by family and/or friends. *(https://www.stroke.org.uk/sites/default/files/stroke_statistics_2015.pdf).*

Stroke can be split into two major subtypes. Approximately 80% is ischaemic (where an intracerebral artery is occluded, usually by thrombosis or embolus). The other 20% is attributable to intracerebral haemorrhage. Hypertension is the main risk factor for all types of stroke. In this article, we will mainly deal with the biggest type of Acute Ischaemic Stroke (AIS) caused by occlusion of the largest arteries in the brain, which carry the worst prognosis (1). A comprehensive guide to stroke is available in a number of textbooks (2).

In AIS, patients' symptoms appear when the brain is starved of blood following occlusion of the feeding artery. Symptoms are attributable to the part of the brain affected and as brain cells die rapidly after the blood supply has been cut off, the occluded artery must be cleared as soon as possible to allow blood to reperfuse the brain and rescue ischaemic neurones. In this article, we discuss the newest and most exciting advance in this field: mechanical thrombectomy.

Here, an interventional neuroradiologist threads a catheter through the patient's arterial tree until the occluding thrombus is reached. They then extract it, "thrombectomy". This restores blood flow to the brain.

Five clinical trials have been published in 2015 showing this procedure dramatically improves outcome after stroke. A wholesale change in clinical practice is getting underway to reorganise services and stroke pathways so as many patients as possible may benefit from this new treatment.

Background Knowledge

During AIS, the actual moment when a patient notices their symptoms probably occurs within seconds of an intracerebral artery being occluded. The brain is exquisitely sensitive to lack of blood and when deprived of nutrients and oxygen will simply stop working. Eventually the ischaemic brain will die, a process known as infarction.

UPDATE ON ACUTE STROKE TREATMENT: THROMBECTOMY

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This is irreversible. Often, when we talk about stroke, people think about the areas of infarction visible on a brain scan. However, the actual disease is the clot in the artery at the start of this whole process. Somehow that clot has to be broken down or removed before the development of irreversible brain infarction.

Simply put, there are two ways in which the clot can be removed. The clot could be dissolved by drugs, a process called thrombolysis. The drug commonly used is called recombinant tissue plasminogen activator (rt-PA). It is administered intravenously and must be given within 4.5 hours of the onset of stroke. Small clots can be dispersed using this drug but large clots may not respond even when the maximum safe dose of the drug is given. The second method is to physically extract these clots. An interventional neuroradiologist threads a catheter up to the clot, deploys a stent (a wire lattice tube) that enmeshes the clot and then gently tries to pull the stent and clot out together thereby restoring perfusion to the brain. This is mechanical thrombectomy.

Clots that cause acute ischaemic stroke may either be formed at the site of occlusion (thrombosis) or embolise from the heart (atrial fibrillation or post myocardial infarction) or atherosclerotic plaques in the arterial tree from the ascending aorta through internal carotid artery to the intracerebral vessels (3). Once an intracerebral artery has been occluded, perfusion in the focal region of brain supplied by that vessel decreases. A collateral circulation could maintain function for a while. However, most arteries are end arteries so no collaterals are available to mitigate ischaemia. Symptoms occur straight away and decreased perfusion results in cell death.

In the anterior circulation, the internal carotid artery splits into the anterior cerebral and middle cerebral arteries. The middle cerebral artery is the larger and more direct route for blood flow so clots tend to go preferentially up this vessel. The carotid circulation supplies the anterior two thirds of the brain and strokes that occur in this distribution are described as anterior circulation infarcts. In the posterior circulation, the two vertebral arteries join to form the basilar artery and then terminate as posterior cerebral arteries. Posterior circulation infarcts are identified in this vertebro-basilar territory.

Occlusive thrombus may block any calibre of vessel and cause a stroke syndrome. Blockage of the larger vessels: the Carotid T (the area where the terminal internal carotid artery splits into the anterior and middle cerebral artery), the middle cerebral or basilar artery cause large regions of brain to be affected and very severe stroke syndromes. However, these large vessels are accessible for mechanical thrombectomy.

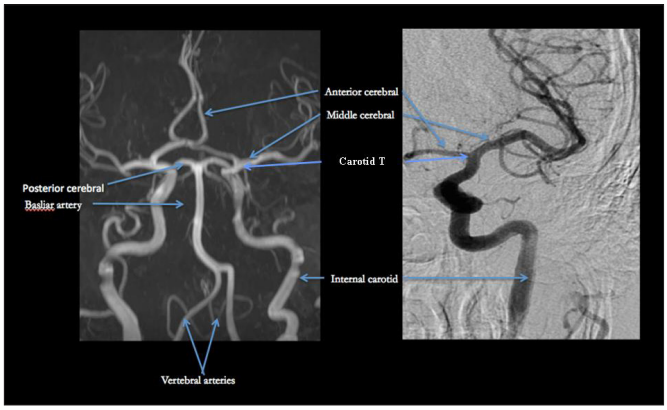

Figure 1: The Cerebral Arterial Tree.

Legend

This figure shows the large arteries which make up the Circle of Willis. The first image is an MR angiogram showing the vessels in a coronal view. The second image is a formal cerebral angiogram, showing the left internal carotid artery and its bifurcation into the middle and anterior cerebral arteries.

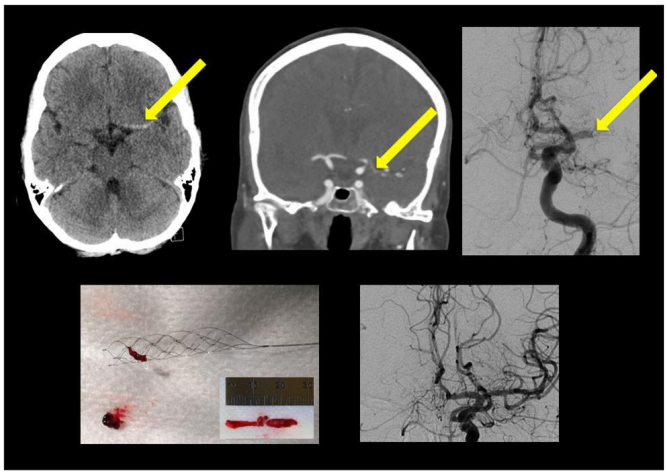

Figure 2

Legend

Across from the top left, the picture shows a slice through the brain on plain CT. You can see a clot in the left middle cerebral artery. In the top middle panel, you can see that the left middle cerebral artery on the CT angiogram terminates abruptly. This is more clearly demonstrated in the intra-arterial angiogram shown on the top right. These features are highlighted by the yellow arrows.

Across from the bottom left, you can see a stent which has been used to pull out a clot. A little bit of clot is still adherent to the stent. The inset shows a larger clot that was extracted.

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In the bottom left panel, you can see that the clot extraction has allowed perfusion in the left middle cerebral artery to return to normal. All acute stroke patients who may be suitable for thrombolysis or thrombectomy must undergo urgent CT brain imaging immediately. The plain CT brain often appears normal. This is a good thing as it means extensive, permanent damage to the brain has not occurred yet giving hope for recovery if the patient is treated rapidly.

It is useful to use a quantitative CT score to measure how much of the brain has been affected by early ischaemic change such as the Alberta Stroke Program Early CT Score (ASPECTS). This is a predictor of the response to thrombolysis. The smaller the abnormal area on CT (and higher ASPECT score), the better the outcome. Training for this is available online. A CT angiogram (CTA), which shows the vessels supplying the brain, is added to the plain CT to identify a large vessel occlusion which may be amenable to thrombectomy.

While CT is the imaging modality most widely available, it is often easier to see an infarct using MR and diffusion-weighted imaging (DWI) is the sequence of choice. It can identify an infarct within a few minutes of onset. Magnetic resonance angiography (MRA) can show the vessels. Sometimes, more advanced imaging routines are employed to look at the perfusion deficit or to compare the hypoperfused area of brain to that which is already infarcted. This gives a rudimentary measure of the volume of ischaemic brain that is endangered but not yet dead.

Evidence Base

Intravenous recombinant tissue plasminogen activator (rt-PA) activates tissue plasminogen found on endothelial cells, which then causes clot breakdown. It has been the mainstay of hyperacute stroke management since the National Institute for Neurological Disorders and Stroke (NINDS) trial showed a good outcome (38% versus 21% receiving placebo) in patients treated within 3 hours of stroke onset in 1995(4).

In 2008, European Cooperative Acute Stroke Study III (ECASS III) showed extending the thrombolysis window to 4.5 hours was beneficial to a proportion of patients (52% versus 45% receiving placebo)(5). Meta-analyses have not supported treatment beyond 4.5 hours (overall 33% have a good outcome compared to 31% receiving placebo)(6).

However, while clearly an effective treatment for many, a substantial number of patients do not respond to thrombolysis, possibly because the occluding clot was not dissolvable by rt-PA. Higher doses risked causing haemorrhage. The next major logical step was to investigate whether physical intervention, mechanical thrombectomy, could improve outcome here.

The Mechanical Embolus Removal in Cerebral Ischemia (MERCI) device was amongst the first thrombectomy devices to be investigated, after animal studies in 1996 showed good outcomes. Once designed for human use, case series suggested good patient outcomes with MERCI. This device would corkscrew through the clot and then be pulled out. It featured in the 2013 trials which were disappointingly neutral: Mechanical Retrieval and Recanalization of Stroke Clots Using Embolectomy (MR RESCUE)(7), SYNTHESIS(8) and Interventional Management of Stroke III (IMS3)(9).

There were several reasons for this, including study design. Many SYNTHESIS patients did not receive rt-PA prior to thrombectomy greatly delaying reperfusion. In MR RESCUE, the average time for any treatment was 330 minutes which we now know is too long. IMS3 had the fastest times but half the patients had large infarcts and poor prognosis from the outset.

Newer devices called stent retrievers were pioneered and two pivotal studies (10,11) showed they gave better recanalization rates and better long-term patient outcomes. This paved the way for further clinical trials focusing on improving the timing of treatment compared to the 2013 trials, changing the selection criteria (smaller core infarction, larger salvageable tissue) and using newer devices. 2015 became the year of thrombectomy trials with 5 positive trials, Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands (MRCLEAN)(12). Extending the Time for Thrombolysis in Emergency Neurological Deficits - Intra-Arterial (EXTENDED-IA)(13), Endovascular Treatment for Small Core and Proximal Occlusion Ischemic Stroke (ESCAPE)(14), Solitaire With the Intention For Thrombectomy as Primary Endovascular Treatment (SWIFT PRIME)(15), and Randomized Trial of Revascularization with Solitaire FR Device versus Best Medical Therapy in the Treatment of Acute Stroke Due to Anterior Circulation Large Vessel Occlusion Presenting within Eight Hours of Symptom Onset (REVASCAT)(16), published at the time of writing.

Trial	MR CLEAN		EXTEND IA		ESCAPE		SWIFT PRIME		REVASCAT	
N	500		70		315		196		206	
Age	Any		Any		Any		<80		<85	
DATA	treatment	control	treatment	control	treatment	control	treatment	control	treatment	contr
N	233	267	35	35	165	150	98	98	103	103
Core Infarct Size (most used ASPECTS)	small	small	small	small	small	small	small	small	N/A	N/A
NIHSS	17	18	17	13	16	17	17	17	17	17
mRS 0-2	32.6%	19.1%	71%	40%	53%	29.3%	60.1%	44.10%	45%	29%
Mortality	18.90%	18.40%	3%	7%	10.40%	19%	9.20%	12.40%	18.40%	15.50%
Time to tPA(minutes)	85	87	127	145	110	125	110.5	117	117.5	105
Time to Angio (minutes)	260	N/A	210	N/A	185	N/A	224	N/A	269	N/A
Recanalisation	75.40%	32.90%	94%	43%	72.40%	31.20%	82.80%	40.40%	N/R	N/R

Table 1: Thrombectomy trials from 2015

Treatment arm=IV TPA plus thrombectomy;
Control= IV TPA alone; mRS=modified Rankin Score

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Each of these trials used intravenous rt-PA before thrombectomy. Mr CLEAN, ESCAPE and REVASCAT used CTA to identify large vessel occlusion. SWIFT-PRIME and EXTEND-IA used an imaging technique which outlined the ischaemic and infarcted tissue. This allowed selection of patients with small infarct core but larger penumbra. Most of the patients in the trials had MCA or carotid T occlusions. (In practice, thrombectomy is offered to basilar strokes too.)

Aspects Score Was Used In All

Table shows data from the 2015 intra-arterial trials. It shows that with smaller infarct cores (and higher ASPECT scores) and more rapid treatment, thrombectomy after rt-PA was superior to intravenous rt-PA alone in selected patients. mRS is the most used disability score in all stroke trials and is a consistent outcome marker (0-2 is no or minimal disability). In all the trials, more patients were less disabled 90 days after the stroke if they had been treated by thrombectomy.

These trials have clearly shown that thrombectomy after thrombolysis in patients with an occluded middle cerebral artery improved the outcome in 1 in 4 patients. It must be remembered intravenous thrombolysis is still the mainstay of treatment for patients who do not have evidence of a large vessel occlusion on the CT angiogram.

Assessment Of The Stroke Patient

A clear, focused history is essential in the initial evaluation of the acute stroke patient. Concentrate on the time and mode of onset of symptoms (sudden or gradual). Simply put, stroke is always sudden onset rather than building up over hours. As soon as the blood supply is cut off, the patient experiences dramatic symptoms. As soon as you think this could be a stroke, you must determine the time of onset or the time when the patient was last seen well. The clock is ticking!

You must also find out the circumstances of the stroke. Was there chest pain preceding it (myocardial infarction)? Do they have palpitations (Atrial fibrillation, AF)? Ask about associated vascular risk factors (AF, hypertension, diabetes, ischaemic heart disease, smoking, peripheral vascular disease and migraine) and social history. Ask about drugs: are they on warfarin or a novel oral anticoagulant? Anticoagulants are contraindications to intravenous thrombolysis but not mechanical thrombectomy. Although time is of the essence, do not be slapdash. Learn to take a fast, focussed and comprehensive history.

While assessing the patient, think about the arterial territory affected. Regarding selection for thrombectomy, with a right middle cerebral artery syndrome, the patient will have symptoms and signs attributable to ischaemia of the cortex (the thinking brain) as well as the sub-cortical structures (the white matter tracts and deeper nuclei, e.g. basal ganglia). Typically, a patient with a right middle cerebral syndrome will be drowsy with left-sided paralysis, loss of sensation, neglect, left homonymous hemianopia but remarkably they may still be able to speak.

Occlusion of the left middle cerebral artery also produces a mixed syndrome with cortical and subcortical features. These patients will be drowsy, paralysed on the right with loss of sensation, neglect and a right homonymous hemianopia. If they are left hemisphere language dominant, they will also lose the ability to communicate. They may not be able to understand what is said or formulate words (aphasia). Listen very carefully to distinguish garbled (dysphasia) from slurred speech (dysarthria).

Occlusion of basilar artery (supplying the brain stem and the occipital lobes) is absolutely devastating. Patients will probably be unconscious with small pupils and eyes pointing in different directions (skew deviation). They will probably be quadriplegic or quadriparetic and they may have crossed signs (i.e. they may have a lower motor neuron facial weakness on one side and upper motor neuron paralysis of the limbs on the other).

It is very important to examine a patient fully but quickly and have a system for doing it. Examine the patient's conscious level using the Glasgow Coma Score, higher mental function, visual fields, speech, swallowing and neglect. Examine the motor and sensory systems carefully. Then do the reflexes (which may be reduced after stroke) and plantars (usually up-going). Co-ordination may be difficult if weakness is present.

Once a diagnosis of stroke has been made, you should determine the severity of the patient's impairments using the National Institute of Health Stroke Scale (NIHSS) (free online training available at www.stroke.org). This is a standardised method of evaluating the severity of the condition. It is reproducible and can be used to measure patient deterioration or improvement. It is essential for clinical trials and audit.

Do not forget to look at the rest of the person. Record the vital signs (especially blood pressure) and examine the cardiac, respiratory and abdominal systems. Always look for scars and feel for a pacemaker. Feel the peripheral pulses especially the femorals; this is where access for thrombectomy will be needed.

These are the blood tests most helpful for acute stroke when considering thrombectomy: full blood count including platelets, serum electrolytes, urea and creatinine, cardiac enzymes and troponin (evidence of IHD), INR and APTT, glucose. An ECG is essential (MI or AF) and a chest X-ray can be helpful.

See Box 1 for a scheme describing the steps essential for looking after a thrombectomy patient. Once the patient is stable from the initial acute management, they should be transferred to a unit where close physiological monitoring can be performed: a hyper-acute stroke unit, high dependency unit or even ITU if necessary. CT scanning should performed 24 hours post thrombolysis to identify the size of the infarct and to ensure there was no haemorrhagic transformation. The NIHSS should also be repeated at 24 hours.

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Immediate management:	Resuscitate as necessary	Use ABCD approach
Diagnosis:	History and examination	Time of onset or last seen well Obtain initial NIHSS score
	Questions to ask	Is patient on anticoagulation? Last time patient ate or drank
	Identify and initiate treatment for following	High blood pressure (BP) Hypo or hyperglycaemia Myocardial infarction Atrial fibrillation (if unstable) Infection
Investigations	Arrange urgent CT brain and CTA Close physiological monitoring Blood tests Estimate weight of patient	Review with senior clinician Pulse, ECG, BP, oxygen saturation FBC, U&E, glucose, clotting
Management	Discuss with Stroke consultant If for rt-PA within 4.5 hours of onset If also for thrombectomy within 6 hours of onset	 0.9mg/kg to be given (10% dose bolus then rest as an infusion) Alert interventional team and consent patient or speak to next of kin
Post treatment management	Admit to High Dependency Unit e.g. Stroke Unit	Monitor BP and pulse, GCS, blood glucose, temperature Swallow assessment or intravenous fluid therapy VTE assessment Repeat imaging 24 hours later or if GCS drops by 2 points Obtain 24 hour NIHSS score

Box 1: Steps to manage an acute stroke suitable for thrombolysis and thrombectomy.

This shows a systematic approach to manage an acute stroke patient who should be considered for thrombolysis and thrombectomy. Once the patient has had their CT scan, a senior stroke member should be involved in the decision-making. However, it is important to understand the whole pathway to enable a junior member of the team to work efficiently and potentially allow forward planning.

Final Thoughts

Whilst thrombectomy is very new and exciting and will revolutionise stroke care worldwide, it is very important to remember the timeless practice of medicine. Stroke is not all about state of the art treatment like thrombectomy. Many stroke patients are elderly and have other comorbidities and these must also be addressed.

Be alert for complications after stroke and treat them aggressively and early to avoid patients suffering further morbidity and possible disability. Crucial issues include feeding and proper hydration. Therefore, ensure that patient has a swallowing assessment. Identify and treat infections aggressively and early (especially urinary and chest infections).

Identify and manage the risk of venous thromboembolism. Consider patients for rehabilitation and adjustment to life after stroke. It is very important that patients should be managed through a co-ordinated Stroke Unit as these have been shown to be the single most effective means of reducing mortality and morbidity in stroke. Lastly, remember to think about what could have caused the stroke and institute secondary prevention to treat the risk factors.

In conclusion, the purpose of this article is to highlight the new treatment that is available for stroke, mechanical thrombectomy. This is used in patients who have suffered large stroke syndromes due to an occlusion of the carotid T or middle cerebral arteries (or basilar artery). Patients with these syndromes are often very sick. They must be identified very rapidly, have urgent brain imaging and be referred without delay to the specialist stroke team for thrombolysis and mechanical thrombectomy.

Questions

1) A 68-year old lady was brought in with sudden onset right sided weakness of the face, arm and leg. Her husband noticed her speech to be slurred and called the ambulance. On arrival of the paramedics she was mute and found to have right facial weakness and right hemiparesis. She had no significant past medical history and no evidence of head injury.

On clinical examination, she was in sinus rhythm with a rate of 90 beats per minute, blood pressure of 140/87 and capillary glucose of 5.4mmol/L. There were no bruits and murmurs on auscultation, and the rest of her respiratory and abdominal examination remained unremarkable. Which of the following steps is most crucial for stroke management?

a) Arrange for urgent CT

b) Ascertain more collateral history especially the time of onset?

c) Assess using the ABCDE Approach

d) Obtain NIH stroke score

e) Inform the research nurse

2) A 64 year old male developed a sudden onset right face, arm and leg weakness while walking home from work. He had a past medical history of hypertension on Lisinopril and family history of hypercholesterolaemia. On arrival in the Emergency Department, he had a profound expressive dysphasia with right facial droop, weak right arm and leg.

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On clinical examination, he was in atrial fibrillation with a rate of 100 beats per minute, blood pressure of 160/99 and capillary glucose of 4.0 mmol/L. There were no bruits or murmurs on auscultation and the rest of his respiratory and abdominal examination were unremarkable. CT head and CT angiogram performed within 2 hours of the onset of symptoms were unremarkable. The plan is to initiate IV rt-PA. Which if the following is most important?

a) Get an urgent collateral history for other risk factors for stroke (e.g. smoking)

b) Perform a VTE assessment

c) Send an INR

d) Arrange an urgent DWI sequence MR brain scan

e) Arrange urgent carotid Dopplers

3) A 58-year old lady attended the Emergency Department (ED) following a sudden onset episode of right sided weakness in the face, arm and leg with a clear onset time. In the ED, she was mute and found to have right facial asymmetry and right hemiparesis. She had no significant past medical history and no evidence of head injury.

On clinical examination, she was in sinus rhythm with a rate of 90 beats per minute, blood pressure of 140/87 and capillary glucose of 5.4 mmol/L. There were no bruits and murmurs on auscultation, and the rest of her respiratory and abdominal examination remained unremarkable. She went on to have a CT head scan and CT-angiogram which showed a thrombus in the left middle cerebral artery proximally. Which of the following statements is correct?

a) Commence IV rt-PA only

b) Commence IV rt-PA and reassess the patient using NHISS

c) Refer immediately for mechanical thrombectomy

d) Commence IV rt-PA and involve neuroradiology for potential mechanical thrombectomy

e) Commence oral Clopidogrel and Aspirin

4) A 88 year old male was brought in by ambulance after being found unresponsive on the floor by his son. The ambulance team found him to have a patent airway, breathing was 19 breaths per minute bilateral air entry, pulse of 110bpm and regular, GCS E2 V2 M5 and slurred speech.

His past medical history includes IHD and ex- smoker. He was assessed in the ED and in addition to his unchanged vital signs, he was noted to have a GCS of E3, V2, M5 (10/15). A&E say that he has a left sided Bell's palsy and a dense right sided hemiplegia. His brain CT and CT angiogram have been done. Where will you look for the thrombus?

a) Right Carotid T

b) Left ACA

c) Left Carotid T

d) Left MCA

e) Basilar Artery

5) A 78-year old female was brought in by ambulance after she woke up unable to talk. On arrival to the emergency room, she was conscious, mute, mild right upper limb drift noted with normal power in the legs. She had no visual and sensory inattention, no limb ataxia, normal eye movements. She had no past medical history of note. What is her NIHSS score?

a) 30

b) 15

c) 10

d) 19

e) 20

Answers

1. B is the answer.

You do not know the time of onset. This could well be a stroke syndrome and the primary urgent specific treatment is thrombolysis. However, you have to know the time of onset and this crucial piece of information is missing from the synopsis.

2. C is correct.

He is in atrial fibrillation and also has hypertension. It is very possible that he is on warfarin. This would be a contraindication for IV rt-PA. There is no clot on the imaging so thrombectomy is not feasible.

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3. The correct answer is D.

There is a large artery cerebral occlusion. While IV rt-PA is essential, it is unlikely to improve the patient's outcome dramatically so mechanical thrombectomy should be considered. However, intravenous thrombolysis should be started a soon as possible.

4. The answer is E.

Crossed signs are classical from the brainstem.

5. B is the correct answer.

1a level of consciousness (0), Ib LOC questions (ask month; age) (2), Ic LOC commands (2), Best gaze (0), Visual (0), facial palsy (0), Motor (Arm) (1), Motor (Leg) (0), Limb ataxia (0), Sensory (0), Best Language (3), Dysarthria (2), Extinction and inattention (0).

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